THE DISEASES OF

THE NERVOUS SYSTEM

*A TEXT-BOOK
FOR PHYSICIANS AND STUDENTS*

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WITH 178 ILLUSTRATIONS

NEW YORK
D. APPLETON AND COMPANY
1893

PREFACE.

To add yet another to the vast number of text-books on nervous diseases which already exist, and most of which are admirably written, is a somewhat serious undertaking, and the question whether the want of such a book is really felt can be answered in the affirmative only with some diffidence. If, despite this, the author has ventured to enter the field with writers like Erb, Eulenburg, Strümpell, Seeligmüller, Möbius, and others, some responsibility must rest upon the shoulders of his publisher, who has more than once assured him that there is still room for another text-book in addition to those already in our libraries. He was influenced, too, by the hope that here and there on certain points the treatment of the subject, by varying from that ordinarily adopted, might while rendering it more easily comprehensible to the beginner make the book none the less reliable as an adviser to those of wider experience. With this end in view, we have considered the manner of dividing the subject matter to be of no small importance, and have therefore chosen to discuss the diseases of the brain, those of the spinal cord, and those of the general nervous system separately, while diseases of the peripheral nerves, cranial as well as spinal, have been fully treated of by themselves. We shall not, as others have done before in treating of the cranial nerves, discuss one here, one there, and the peripheral nerves of the spinal cord partly in one place, partly in another, according as we are dealing with their motor or their sensory disturbances, but shall speak of the former and of the latter in the order that their anatomical position would indicate. In this way we shall be enabled to obtain a comprehensive view of them, which is undoubtedly highly desirable. To please all is impossible. Of course, one reader would wish that one point had been treated in this particular way and in that par-

ticular place, while another would have fancied something to-
tally different. About this we cherish no fond illusions. A
systematic discussion of the sympathetic system, as well as of
affections due to specific micro-organisms—e. g., tetanus, lyssa
—we have not attempted, partly for other reasons, and partly
because the space allotted to us would not allow of it. Further
deviations from the usual course—e. g., the attempt to divide
the functional neuroses into two classes, and to put tabes not
among spinal diseases, but among those affecting the general
nervous system—are sure to meet with sharp criticism, and
we do not expect that the position we have assumed toward
hypnotism and the treatment by suggestion will for the present
meet with general acceptation. Every new and strange idea
necessarily requires time to win its way and gain for itself a
large circle of adherents. We hope it will be our pleasant task
to record in a new edition (if such by good chance be called
for) the assent of more of our professional brethren to this
view. Finally, the list of references which we have inserted
at the end of each chapter, thinking it would be of conven-
ience to such of our readers as wish to study fully any particu-
lar point, this list, we confess, will not bear too severe a criti-
cism; naturally, we could not mention every work dealing
with the subject; for such a task neither the knowledge of the
author nor the capacity of the book could suffice. Only the
most important, and of these only the more recent, works could
be quoted.

Only too well aware of his shortcomings, but convinced
that he has set down concisely what he has learned from oth-
ers and observed himself in an extensive hospital as well as
private practice—convinced also that the illustrations, for which
he has to thank the generosity of the publisher, will do much
to enhance the interest of the text—the author bows willingly
to the dictum of the old proverb,

<div align="center">" Habent sua fata libelli."</div>

<div align="right">DR. LUDWIG HIRT.</div>

BRESLAU, *September, 1889.*

INTRODUCTORY NOTE.

THE pleasure of introducing to the profession of this country a translation of a standard work is enhanced by the opportunity it affords of acknowledging how great is our debt to those —translators and publishers—who have made current in English the works of Trousseau, Niemeyer, Virchow, Cohnheim, and others. All recognize the necessity of teachers knowing the classical works in all languages, but of equal importance is it that the practitioners in all countries should have easy access by means of translations to the thoughts and experience, the ways and methods, of the masters of our art the world over. No better corrective exists to the vice of Philistinism—that narrow conceit of the special prominence of medicine in any one country—than a wide diffusion in all of the best works of each.

Early in 1890 my attention was called by Dr. Weir Mitchell to the first part of Prof. Hirt's Handbuch der Nervenkrankheiten, which he characterized as an exceptionally well arranged and thorough work on diseases of the nervous system. The completed work seemed in many respects so admirable a text-book that I wrote to Prof. Hirt and asked his permission to have it translated.

The arrangement of the subjects to which the author refers in the preface, though somewhat novel, is justifiable and entirely satisfactory ; and it is a distinct advance in classification to place tabes dorsalis and dementia paralytica among the diseases of the general nervous system, instead of in the sections on diseases of the cord and diseases of the brain respectively.

The fact which makes the work of value to the teacher, the student, and the practitioner is the graphic description of the anatomy and symptomatology of the different diseases. Where all is so good it is invidious to select, but the chapter on tabes

is an illustration of our author's lucid and, at the same time, thorough treatment of his subject. The various affections are treated of also from an advanced modern standpoint; conflicting theories and passing observations are submitted to a wise criticism through which the author's own large and varied experience is very apparent.

An attractive aspect of the work is the excellent character of the illustrations, which, as they are in great part original, will be a pleasing relief to the hackneyed cuts which have for so long passed from book to book in English works.

Pursuing the *via media* in the important question of treatment, neither displaying the pessimism which too many maladies of the nervous system would seem to justify, nor an optimism so flagrant as to savor of quackery, Prof. Hirt is a safe guide in the highways and byways of neurotherapeutics.

And, lastly, I think the author has been fairly handled by his translators, who, bearing in mind the admonition of Dryden, "not to lackey by the side of his author, but to mount up behind him," have given a clear and interesting rendering of the original.

WILLIAM OSLER.

BALTIMORE, *January, 1893.*

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LIST OF ILLUSTRATIONS.

DISEASES OF THE BRAIN AND ITS MENINGES, INCLUDING THE CRANIAL NERVES.

THE study of brain diseases, we must confess, has not made the strides that might have been expected after the numerous and varied researches that the last decades have seen. For this our present very imperfect knowledge of the anatomy, and still more our doubts as to the physiological functions of the different parts of the brain must be held largely responsible. The structure as well as the physiological functions of the human brain are, up to the present time, so little understood that we are far from having any sure basis upon which to lay the foundations of a cerebral pathology. No small progress has been made from an anatomical standpoint through Stilling's method of serial sections, a method which Meynert, Henle, Wernicke, and others have not been slow to use, in their admirable researches, to which important additions have been made by the embryological studies of Flechsig, and by the method of "arrested development" used by Gudden and his pupils (atrophy method; *Degenerationsmethode*, Schwalbe); but with all this we have only here and there single stones which we have not as yet been able to combine for the construction of a harmonious whole. Brilliant from a physiological standpoint as was the discovery of Fritsch and Hitzig (1870) of the electrical irritability of the cortex, and of the existence of motor regions therein, unexpected as were the results which the experimental method of Munk brought to light, extraordinary and interesting as are the conclusions based upon the clinical and post-mortem observations of Charcot and his school— all these, wide-reaching and admirable as they were, are far

from having given us a full understanding of the functions of the different parts of the brain, and an explanation of the disturbances to which they are subject. Constant and untiring work is still needed, and the best results are promised from the intelligent combination of clinical observation with pathological research. The pathology of the brain can not be better advanced than by the patient clinical observation of cases during life and a careful autopsy after death. In institutions where not only the fullest opportunities are afforded for clinical observation and for the systematic conduct of post-mortem examinations of the brain, but where also the best men are found to supervise the work, in these will cerebral pathology make the greatest strides.

We shall divide our description of cerebral diseases into three parts. In the first we shall take up the diseases of the meninges, in the second those of the cranial nerves, while the third will embrace the diseases of the brain in the stricter sense, i. e., those of the white and gray matter of the hemispheres and of the central ganglia.

PART I.

DISEASES OF THE MENINGES OF THE BRAIN.

THE meninges are relatively more frequently affected by disease than the brain substance itself, and quite a considerable number of the cases which we commonly call diseases of the brain are really to be classed as affections of the meninges. Since these diseases can develop under the most varied conditions, and can be primary as well as secondary, they are of great practical importance, and we must try to distinguish most carefully between the different forms which they assume.

A clear understanding of the pathological processes in these diseases will be facilitated by some remarks upon the anatomy of the meninges.

The outermost, tough, fibro-tendinous membrane, called the dura mater, forms at the same time the inner periosteum of the cranial bones. It has an outer, rough, and an inner, smooth surface. For the nerves as they emerge from the skull this membrane supplies sheath-like coverings, among which that of the optic (vagina optici) is the most conspicuous. The dura consists of two layers, which at certain places separate, forming spaces (sinus duræ matris). The cerebral layer sends two leaf-like processes into the cavity of the skull—one vertical, constituting the so-called falx cerebri (processus falciformis major), which extends from the crista galli to the internal occipital protuberance, and the so-called falx cerebelli, which penetrates between the two hemispheres of the cerebellum (processus falciformis minor); another horizontal, the so-called tentorium cerebelli, which is pushed in between the lower surface of the occipital lobes of the cerebrum and the upper surface of the cerebellum. Both together are called the processus cruciatus duræ matris. The blood-supply of the dura is derived from branches of the meningeal arteries. That it possesses its own nerves is doubted by some (among them Luschka), confirmed by others (Ruedinger, Alexander). It is most probable, however, that it is the trigeminus which chiefly provides for the innervation of the dura.

The second membrane, the arachnoid, is delicate and contains no vessels. Its outer surface is smooth and looks toward the subdural space, while the inner is rough and turned toward the pia mater. The so-called subarachnoid space, which is situated between the arachnoid and pia, contains between the meshes of the subarachnoid tissue the serous cerebro-spinal fluid. At the convexity of the brain the two membranes lie in close proximity, while at the base the arachnoid is in places raised from the pia, thus helping to form large spaces, the so-called cisternæ subarachnoidales (Fig. 1). As to the significance of the arachnoid villi (Pacchionian bodies) different observers are at variance. These consist of smaller or larger bulbous excrescences of the arachnoid, are of a whitish color, and grow into the tissue of the dura, usually in the region of the sinuses. Their function is probably to facilitate the flow of the serous fluid from the subarachnoid space into the sinuses of the dura as soon as the pressure in the latter becomes lower (Key and Retzius).
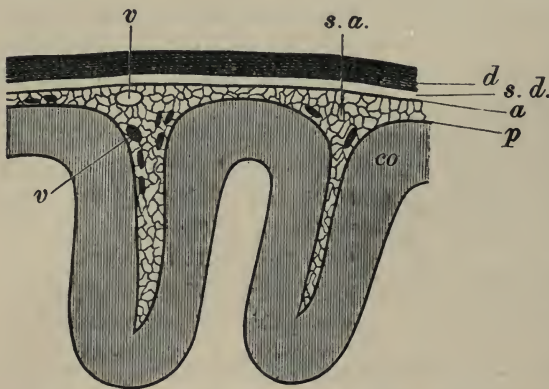
Fig. 1.—Cross Section through the Cerebral Cortex and its Membranes.

co, Cortex; *p.* pia mater; *s. a*, subarachnoid space; *s. d*, subdural space; *d*, dura mater; *v. v*, blood vessels.

The third membrane, the innermost, the one which lies directly on the surface of the brain, is called the pia mater. It dips down into the depths of the sulci and forms a continuous lining of those parts of the brain-stem which are covered by the cerebrum and cerebellum, and apparently also sends processes into the interior of the brain through the so-called transverse cerebral fissure. These processes, which are called telæ choroideæ, present peculiar villous formations, very rich in capillary vessels, and therefore of a deep-red color (plexus choroidei). There is a tela superior with the lateral choroid plexus and a tela inferior (sive cerebelli), which lies between the ventral surface of the cerebellum and the dorsal surface of the medulla oblongata. The covering or ependyma of the ventricles is not a part of the pia mater, but is simply a layer of epithelial cells. The nerves of the pia mater belong to the sympathetic.

The diseases of the meninges of the brain consist mainly of

inflammatory processes affecting either the pia or the dura mater. We shall study the diseases of the two membranes separately.

CHAPTER I.

INFLAMMATION OF THE INNER SURFACE OF THE DURA MATER, PACHYMENINGITIS INTERNA HÆMORRHAGICA, HÆMATOMA DURÆ MATRIS.

The origin of the extravasations of blood which at the autopsy are often found on the inner surface of the dura, and which can be easily scraped off with the knife, is not altogether understood. Some (Virchow, 1856) hold that the primary affection is an inflammation, and the hæmorrhage takes place secondarily into the newly formed, highly vascular connective tissue, while others look upon the hæmorrhage as primary; and, indeed, recent observations (Sperling) seem to be very much in favor of this latter view. If extensive hæmorrhages occur, after spreading over more or less of the inner surface of the dura they become encapsulated, and are then called *hæmatomata duræ matris.* Such a hæmatoma may contain from three hundred to four hundred grammes of extravasated blood, may attain the size of a man's fist, and so exert a deleterious pressure upon the brain. The walls are sometimes smooth, sometimes rough; the contents are not always sanguineous, but may be serous or purulent. They are most commonly situated at the vertex near the falx cerebri, sometimes also in the frontal region, very rarely at the base. The arrangement of the hæmatoma in layers, which is seen on section, proves that the whole process consists of extravasations which have occurred at different times. In the least-marked cases only a delicate reddish membrane is found, presenting reddish or brownish specks, and is easily stripped off from the dura. Only gradually the different layers are developed, the one nearest to the brain, of course, being always the most recent, the one lying on the dura the oldest. Between the layers are the hæmorrhages. If it happens that the most recent layer is perforated by the hæmorrhage there occurs free extravasation of blood between the dura and the arachnoid— that is, an intermeningeal hæmorrhage.

Ætiology.—In the ætiology of the affection, diseases of the heart and kidneys, but especially chronic diseases of the brain, play by far the most important part. It is seen in almost all affections which lead to an atrophy of the brain; further, it may be met with in infectious diseases—for instance, in typhoid fever, scarlet fever, acute rheumatism; also in conditions of what we may call blood-dissolution, as in the general hæmorrhagic diathesis. Among the exciting causes are traumatism of the cranial bones and inflammation in the neighboring parts —for instance, in the petrous portion of the temporal bone. Of predominating importance, as an ætiological factor, is the abuse of alcohol. Almost in all autopsies on old drunkards we find a more or less well developed pachymeningitis interna, which has recently also been experimentally produced in dogs by continued doses of alcohol (Leyden). The fact that statistics have established that men, and more especially old men, are by preference affected by this disease also seems to point to alcohol as the principal cause.

Symptoms may be entirely absent. This is the case if the hæmorrhage, or the newly formed membranes were not of sufficient extent; but if symptoms are present, then among the most important we find headache, which may persist for years, but which of course in itself, even if we have a history pointing to this disease, as, for instance, the abuse of alcohol, is never sufficient to justify the diagnosis. With a sudden rise of intracranial pressure we always have apoplectiform attacks, in which consciousness is lost for a variable time, and in which the patient may die without regaining consciousness. Vomiting, slow pulse, and a very conspicuous narrowing of the pupil are not wont to be absent. Repeatedly peculiar dreamy conditions have been observed after such a coma, during which the patients seem completely dazed and the urine and fæces are passed involuntarily. If the hæmatoma lies over the motor area, epileptiform convulsions and hemiplegia may result, serious motor disturbances, limited to one side, which may entirely disappear in a short while, or may last for months. Unilateral nystagmus and papillitis have been reported by some (Fuerstner). The further course depends upon the absorption of the clot or the occurrence of a further hæmorrhage, as the case may be. The repeated development of severe cerebral symptoms, after striking and rapid improvement, speaks under certain circumstances for the existence of hæmatoma of the

dura, because it is just this frequent change in the condition of the patient which is characteristic of the course of the disease. Months and even years may thus pass without a fatal result, and much more rarely than one would be led to expect is it possible to make a positive diagnosis during life, because all the symptoms which we have just mentioned can be found just as well in other cerebral affections, in hæmorrhage, embolism, new growths, etc., and the only thing we have to fall back upon is the history, if this be one of alcoholic excesses. The paroxysmal appearance of new symptoms is not to be overlooked, inasmuch as it confirms to some extent the diagnosis of pachymeningitis. However, under all circumstances the task is a difficult one.

Prognosis.—The prognosis for recovery of course is absolutely bad if thickening has reached any degree worth mentioning, and when we have to deal with a large hæmatoma which encroaches considerably upon the intracranial space the prospect for life is, to say the least, not hopeful. On various anatomical grounds death can occur suddenly and unexpectedly.

Treatment can only be of any value in the earlier stages, but unfortunately then the disease is usually not recognized. Interdiction, or at least restriction, of the use of alcohol, if this plays a part, energetic antiphlogistic treatment in the form of local bloodletting, the ice-cap to the head, counter-irritation by inunctions of mercurial ointment, and active purgation (calomel) would surely give us good results; but, as we have said, these means are, as a rule, used too late, and as a matter of fact the progress of the disease is usually not altered by any therapeutic measures.

The new growths of the dura mater, as far as they at all manifest themselves by symptoms, will be described under the head of brain tumors.

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CHAPTER II.

A. Pathological Anatomy. Ætiology.

INFLAMMATIONS of the soft cerebral meninges occur either at the base or at the convexity of the brain, according as they are primary or secondary—*i. e.*, associated with other diseases —and one can, indeed, with a few exceptions and bearing in mind the transition forms, put it down as a rule that secondary, metastatic meningitis affects the convexity, while a primary meningitis is usually found at the base.

In contradistinction to what takes place in the dura, where the only purulent inflammations that we find are such as have extended by contiguity from neighboring parts, here we have to deal with purulent inflammations alone. This purulent inflammation of the soft membranes of the brain, the leptomeningitis cerebralis, is an infectious disease, and occurs in epidemics as epidemic cerebro-spinal meningitis, or more rarely sporadically, the two forms, however, being ætiologically identical. Besides these, we find developing in the course of tuberculosis, sometimes very early, sometimes late, a specific form of meningitis, the tubercular meningitis.

Pathological Anatomy.—The pathological processes can be traced in the pia as well as in the substance of the brain. In the meshes of the former we find a purulent exudate, which is in rare cases limited to one hemisphere; if it is copious, the pia can easily be stripped from the brain; if it is scanty, this can not be done without loss of substance. The brain substance is œdematous and fills up the skull more than usually, so that the convolutions appear flattened. The ventricles are filled with an unusual amount of fluid (hydrocephalus internus). The hæmorrhages which are recognizable in the brain substance do not exceed in size that of a pin's-head, and are either isolated

or are seen especially near the ventricular walls in greater numbers, the so-called capillary apoplexies. Besides these there are other small punctiform hæmorrhages, or rather spots of red softening, and minute hæmorrhages closely grouped together. All these focal changes are to be looked upon as due to the influence of the specific virus. If the process has become a chronic one, then the characteristic features are œdema of the pia, wasting of the brain substance, hydrocephalus internus, and thickening of the ventricular ependyma, which gives to the surface a velvety appearance and changes the shape of the ventricles in a characteristic manner, the normally sharp edges becoming rounded off (chronic meningitis).

In tubercular meningitis we find not only signs of an inflammatory process, but also the formation of tubercles; both, however, do not progress *pari passu.* There may be a very extensive eruption of tubercles and a relatively slight inflammation, or *vice versa*, but always, especially in children, the greater part of the jelly-like exudate is situated at the base (basilar meningitis), between the pons and the anterior perforated space, and imbedded in it are the grayish-white tubercles which are seen as nodules, sometimes as large as millet-seeds, and are found in the greatest numbers among the larger vessels of the fissure of Sylvius, on the chiasma, pons, etc. The vessels are fuller than usual, and small hæmorrhages can occasionally be seen in the pia. The substance of the brain is affected in the manner above mentioned—hydrocephalic effusions into the ventricles are rarely absent, and there is a decided fullness of the choroid plexuses. Foci of softening are noted chiefly about the basal ganglia; they are produced sometimes by the occlusion of an artery, sometimes by the pressure which the exudate exerts on the vessel, or, again, by an arteritis obliterans. Regeneration has been known to occur even in tuberculous meningitis. Dilatation of the ventricles and other signs of an increased intracranial pressure may continue, and collections of fluid in the pia and in the ventricles may still be present, but the fluid may again become clear, the pia moist and non-adherent to the cortex, and the tubercles present no inflammation around them (Wernicke).

A chronic form of basal meningitis, in which the pia is in places either thickened and indurated, or where we have a formation of brittle crusts, can be of a gummatous nature (Wernicke).

Ætiology.—As has been stated, cerebro-spinal meningitis has to be looked upon as an infectious, sometimes epidemic, disease, the parasitic nature of which was demonstrated by Leyden in 1883. He found in the tissues of the pia and in the turbid cerebro-spinal fluid diplococci, which A. Fraenkel recognized as identical with the pneumococcus (Deutsche medicinische Wochenschrift, 1886, 13). Whether or not these cocci gain access to the meninges through the nasal cavity and the foramina of the ethmoidal plate, we are unable to say. Children and young people are more easily affected by the disease—which is, by the way, not contagious—than adults, and the infection can be carried by them from place to place. In inclosed and crowded localities, e. g., in prisons and barracks, the disease may become endemic, a thing which Hermann (cf. lit.) has also observed in private houses.

But even when there is no epidemic, the disease may appear sporadically anywhere, and then also must be regarded as being just as much of a parasitic nature.

A tangible cause for meningitis we find in traumatism of the cranial bones, causing injury of the soft parts, so that the pathogenic organisms can penetrate through the open wounds. The (septic?) *Streptococcus pyogenes* (Eberth), which is less delicate and more resistant than the above-mentioned coccus, has been demonstrated in such cases. If, however, in traumatism, the air remains excluded, as happens in fractures at the base, then the presence of a purulent meningitis is difficult to explain.

The diseases of the bones of the skull, more especially those of the petrous portion of the temporal bone and of the auditory apparatus, play an important part in the ætiology of meningitis. From an otitis media may be developed a caries of the petrous portion of the temporal bone which may perforate the thin roof of the tympanic cavity. Another extension of the inflammation may come from the mastoid cells if an embolus passing from the veins of the bone lodges in one of the venous sinuses, which then becomes the seat of a purulent thrombo-phlebitis.

That the tuberculous meningitis has its origin in tuberculous processes in other organs is clear, and the ætiology is therefore identical with that of tuberculosis in general—i. e., there is invariably an invasion of the tubercle bacillus. It is an interesting fact, however, that though the primary disease in other organs need not necessarily have produced any or at

least no marked symptoms, we can still have secondary disease of the pia with the symptoms peculiar to it, which we shall describe. Children especially are not rarely attacked by meningitis, the tuberculous nature of which is only recognized at the autopsy, and we may not have the faintest suspicion of the existence of a previous tuberculous infection. In other cases, however, the meningitis only appears after the pulmonary tuberculosis has made great progress. Caseous bronchial and mesenteric glands, as well as solitary tubercles in the brain, may be the starting point of the meningeal affection, while it less commonly follows tuberculosis of the joints, bones, tuberculous affections of the intestines and genito-urinary apparatus.

The relation of meningitis to other diseases—i. e., its simultaneous appearance with pneumonia, scarlet fever, and typhoid fever, ulcerative endocarditis, etc.—has to be subjected to further study, and especially has the question to be inquired into whether in those cases we have to deal with a double infection or whether we have a single noxious agent which produces both the meningitis and the affection which accompanies it (Fel. Wolff, cf. lit.).

It is well established that children and young people are more frequently and more severely attacked by meningitis than older persons, and it seems as if the disease is never found in old age. Early childhood, the period between two and three years of age, furnishes relatively the greatest number of victims and gives the most unfavorable outlook.

B. Symptoms, Diagnosis, and Treatment.

Symptoms.—The idiopathic, purulent meningitis of the adult usually begins after insignificant prodromal symptoms, such as digestive disturbances, hebetude, etc., with headache, which soon attracts by its severity and its duration the attention of the physician. Exceptionally the patient has hours of comparative ease; usually the headache is so intense that he becomes almost frantic. He tosses about in bed with sighs and groans, and, even when the mind has become dulled, involuntarily again and again puts his hand to his head. Sometimes delirium develops early, to cease again and sooner or later give way to a dull and somnolent condition, which in its turn passes into a deep coma, the immediate forerunner of death.

In some cases the diagnosis is facilitated by characteristic

symptoms, such as rigidity of the neck and remarkable hyper-æsthesia of the skin and muscles. The former is especially well recognizable when the patient is asked to sit up in bed, which he can not do without intense pain; the latter is often detected in the examination of the patellar reflexes, which themselves present no particular abnormalities. If we then find besides these symptoms in the beginning of the disease occasional (cerebral) vomiting, a strikingly slow pulse, which is in remarkable contrast with the elevation of temperature (102° and more), and, what is not uncommon, marked contraction of the pupils, we can not easily make a mistake in the diagnosis. Only exceptionally, however, do we meet with a combination so favorable for the task of the diagnostician. More frequently, as we shall explain at length, he has to encounter considerable difficulties. There is no doubt but that the vomiting is of cerebral origin; but where the center for this is to be sought, whether in the medulla oblongata or, as Hlasko claims (Dorpat, Inaugural Dissertation, 1887), in the corpora quadrigemina, still remains undecided, as also does the question whether or not we are dealing with a functional stimulation of this center. Choked disks and transient paralysis of the ocular muscles are occasionally observed. The former is not easily recognized when the patient quickly passes into sopor; the latter, however, is recognized without difficulty by the strabismus which it causes and the nystagmus-like movements of the eyeballs. Symptoms of irritation, partly referable to the cortex, in the form of general or unilateral convulsions, partly to individual cranial nerves (grinding of the teeth, trismus, facial spasm), have been repeatedly noted. They seem, however, not always to occur, and for diagnosis must be considered as of minor importance.

The course of the purulent meningitis in the adult is different in different cases. As a general rule, however, certain symptoms, especially headache and the rigidity of the neck, sometimes hyperæsthesia of large areas of the skin, persist from the onset and increase, while others, as, for instance, the vomiting and the cranial nerve symptoms, are only transient.

The duration of the disease can be two, three, four, to eight, more rarely ten to fourteen days, and the younger the patient the more dangerous is usually the disease. The patients die, as a rule, without regaining consciousness, but the coma may last for days.

The symptoms of the epidemic cerebro-spinal meningitis are on the whole, as far as the brain is concerned, quite similar to those of the idiopathic form. In both the headache is the predominating symptom, and the rigidity of the neck is rarely absent, but in the epidemic more frequently than in the idiopathic form the disease begins with a chill. The course of the fever presents nothing characteristic. It is sometimes of a remittent, sometimes of an intermittent type, the temperature sometimes reaching a height of 104° to 107° F. More or less severe disturbances of consciousness may occur even without a marked elevation of temperature. Among the cranial nerve symptoms, the disturbance in hearing caused by the auditory nerve taking part in the inflammatory process has to be mentioned. Visual disturbances are more uncommon, but optic neuritis has been repeatedly noted. If other cerebral symptoms—convulsions, hemiplegia, aphasia—occur, they have to be considered as complications due to an extension of the inflammation to certain parts of the brain substance.

The spinal symptoms, which are superadded, may consist of a distinct tenderness along the whole vertebral column, of a hyperæsthesia of the legs (which is of diagnostic importance), and of twitchings of the extremities. A peculiar but, as it appears, extremely uncommon symptom is the so-called flexor contracture of Kernig: the patient when in a sitting posture is unable to extend his knees, because a contracture in the flexors is developed, which disappears as soon as the thigh is no longer flexed at the hip-joint. Bull (cf. lit.) has made some communications on this point. The mechanism of micturition is only influenced when the patient becomes unconscious; then the urine is passed involuntarily. Besides this there are no important bladder symptoms. The urine sometimes contains albumen or sugar, also some tube-casts. Sometimes the quantity voided is greatly increased, a polyuria, which we have to consider as a cerebral symptom.

Other organs rarely take part in the disease. The circulatory, respiratory, and digestive apparatus usually remain normal, and serious stomach affections, endocarditis, and pneumonia, of which we have already made mention above, are seen only rarely as complications. Moderate splenic enlargement often occurs. Among the skin eruptions which sometimes accompany cerebro-spinal meningitis, besides urticaria and (much more rarely) roseola, we have a herpes labialis,

which, without being of any prognostic value, possesses a certain diagnostic significance.

The course of epidemic meningitis is still more uncertain and variable than that of the idiopathic form. It can be rapid, and may end fatally within a day or a day and a half, in which case convulsions are followed by deep and persistent coma. It can, however, also be protracted and with remissions, during which the patient is in fairly good condition, may last for weeks. In the beginning of the epidemic usually grave cases are more common, while the longer it lasts the milder they become. It seems as if an attenuation in the virulence of the microörganism had taken place. There occur, besides, abortive cases, in which, while they undoubtedly must be classed with the epidemic disease, only quite a small, sometimes insignificant, part of the symptoms are developed. The period of incubation is from three to five days.

The symptoms of tuberculous meningitis differ somewhat in children and in adults.

(*a*) In children the disease runs either a very acute or a more chronic course. In the first case only a few days may elapse between the onset of the trouble and the fatal issue; in the latter, weeks and months may pass before amelioration and recovery, or in these cases also death takes place.

The acute form usually begins suddenly with epileptiform convulsions. Apparently healthy, robust children fall into convulsions and then complain of severe headache and nausea, which is often followed by vomiting; the pulse becomes irregular, and its variations in frequency are more striking than in any other disease. On examination, we find the temperature only moderately elevated, but the patient is very restless, throwing himself about in bed and complaining of pain in the abdomen, chest, etc. Strabismus, trismus, grinding of the teeth, are often noted, and on mechanical stimulation of the skin striking circumscribed red spots, *Trousseau's tâches cérébrales*, appear. The patients sigh deeply when examined, or give an unexpected loud, sharp cry, the *cri hydrocéphalique*, a very unfavorable symptom which is of far greater importance than the spots, from the appearance of which we are not justified in drawing either favorable or unfavorable conclusions. The approach of death is announced by an enormous increase in the frequency of the pulse, by renewed convulsions, and deep coma.

The chronic form begins insidiously and gradually, the first thing to attract our attention being the change in the disposition of the child. Previously gay, friendly, playful, and companionable, he becomes peevish, irritable, unmanageable, and willful. On the least provocation he begins to cry and to be naughty, so that the parents find it necessary to punish him. It is not until the sleep begins to be disturbed and the child tosses about all night and groans in its sleep, wakes up in the morning without having rested, and complains of headache that the parents become apprehensive, and the loss of appetite, the occasional vomiting, the obstinate constipation, and the pale, sickly appearance confirm the fear that a serious malady is on the point of showing itself. The symptoms may for weeks remain obscure; high temperature may alternate with low, a frequent with a slow pulse, without it being possible to say anything definite about the case. Only when one day an epileptiform attack occurs, the headache increases in intensity, the child becomes somnolent, cries out during sleep, shrinks on being touched (hyperæsthesia of the skin), only then is the condition clearer, and finally can not be mistaken when such a focal symptom as paralysis of the eye muscles appears. Even then remissions may occur, and decided, though in most cases only temporary, improvement is not excluded. The outlook is always doubtful, and can, even when the prospects appear most favorable, be very serious.

(*b*) In adults the difference between the chronic and acute form is less marked than in children. Patients who have by no means presented definite signs of tuberculosis begin to complain of vague headache, general prostration and *malaise;* their sleep becomes disturbed and restless; especially in the morning they feel tired and unstrung; they complain of loss of appetite, and may have occasional vomiting spells. In some cases the psychical symptoms are the most prominent, and it may happen that the disease begins with the symptoms of a delirium tremens, especially if the patient be a drinker. In all cases the sensorium becomes sooner or later dull; the patient appears dazed, gives confused answers, and conveys in general the impression of a man whose mind is affected. Not rarely delirium comes on; in it the excitement and exaltation are the most prominent features. But with all these symptoms the influence of a severe, agonizing headache still makes itself known, and even during unconsciousness the patients often raise the hand

toward the head, throw themselves about in bed restlessly with groans, and seem sensitive to the slightest touch or tap on the head. The participation of certain cranial nerves, especially the oculo-motor and the abducens, is evident from the transient ptosis, the inequality of the pupils, and the strabismus; the ophthalmoscopic examination not uncommonly reveals choked disks. If in looking for the latter we are able to find tubercles in the choroid, this is of course of the highest importance for the diagnosis. The facial nerve, which often becomes affected, may be the seat of spasm or of paresis. If we remember that the base of the brain is the chief seat of the inflammation we can easily understand why these cranial nerves should be implicated. If motor disturbances, consisting of general or unilateral convulsions, or of hemiplegia or paresis, as well as speech disturbances, make their appearance, we may assume that an eruption of tubercles has occurred in the brain cortex. The more pronounced these disturbances, which are to be regarded as focal symptoms, the more likely is it that circumscribed tuberculous softenings exist in the cortex. Sometimes also a peculiar tonic rigidity develops in all four extremities which seems to be of reflex origin. The reflexes, at first increased, but presenting nothing characteristic, usually lose in intensity as the disease goes on, and finally disappear altogether. With regard to the sensory changes, it should be remarked that hyperæsthesia of the skin is not as regular a symptom in this as in the first described form of meningitis. The temperature, as a rule, is somewhat above the normal, yet it varies, and occasional remissions may be followed by elevations, or it may remain constantly between 101° and 102° F., or thereabouts. Nothing certain, however, can be said about it. Strümpell reports a temperature of 88° during the agony. Equally variable is the pulse, which as a rule is slowed. We may count 40 to 50 beats a minute, while in a few hours it may rise to 100 or 120.

Other organs take but a small share in the disease, and even the lungs show signs only when simultaneously affected with miliary tuberculosis. If the respiration assumes a Cheyne-Stokes type (after a series of shallow respirations, which become deeper and deeper, a complete pause), this is usually a bad omen.

To say anything positive about the course of tuberculous meningitis is impossible. It is not constant, but sometimes

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acute, sometimes chronic, sometimes presenting long inter-
missions, and sometimes steadily progressive.   A subdivision
into different stages may look very well on paper, but to dem-
onstrate them at the bedside is only rarely possible.   A
period of cerebral irritation has been distinguished from one
of increased intracranial pressure, and this again from a period
of paralysis.   The first has been thought to be characterized
by headache, vomiting, and delirium ; the second by slowing
of the pulse and paralyses ; the third, finally, by increase in the
frequency of the pulse, elevation of temperature, and deep
coma.   But such a division entails no practical benefit, as the
so-called " stages " are often not distinguishable from each
other, but pass directly one into the other.   From the instruct-
ive treatise of Hirschberg (cf. lit.) we learn that even the man-
ner of onset may vary much, and that it may be difficult, even
in the stage of focal symptoms, to make a diagnosis.   If a con-
sumptive suddenly develops symptoms of motor or sensory
paralysis or irritation, this should always make us suspect the
existence of a tuberculous process in the brain.

Diagnosis.—None of these different forms of meningitis that
we have described is easy to diagnosticate, with the excep-
tion, perhaps, of the epidemic cerebro-spinal.   When several
cases have occurred in a community the recognition of new
ones presents no difficulty, especially if we keep in mind the
frequency with which herpes labialis is met with in the disease.

Of all diseases, perhaps typhoid fever is most likely to be
mistaken for meningitis.   There is no doubt, and it has been
shown by reliable observers (Curschmann), that there are cases
in which meningitic symptoms are very well marked, but in
which typhoid bacilli are found in the cord at the autopsy to
be the infectious agent.   We might be led to believe that at
least the characteristic temperature curve, the splenic enlarge-
ment, the condition of the stools, and the rose spots would be
sufficient to make a mistake impossible, but this is by no means
always the case; there are instances in which typhoid fever
can not with certainty be excluded, and then the differential
diagnosis is simply impossible.

If uræmia enters into the question of diagnosis, the exam-
ination of the urine (for tube-casts, etc.), suppression of the
urine, if it should be present, and the appearance of the con-
vulsions will facilitate the recognition of the true condition.

Whether we have to do with a case of croupous pneumonia

or with meningitis is, in the majority of cases, easy enough to decide. Both affections may, however, occur together, and then it is important to remember that marked hyperæsthesia of the skin, staggering gait, and rigidity of the neck all may be present with pneumonia alone. If this be complicated by œdema of the glottis, so that respiration is difficult, the patient will fix his head in order to bring into play the auxiliary muscles of respiration, and thus in the recumbent position too the rigidity of the neck is simulated (Wernicke). The existence of meningitis is only, then, to be assumed if pronounced basal symptoms are present, especially if paralysis of the eye muscles has existed for a certain period of time.

More frequently delirium tremens is associated with meningitis, and we are not always able to decide whether the delirium, the tremor, and the epileptiform convulsions are referable to the latter or to the former.

According to Reynaud (cf. lit.) there are cases in which, although the symptoms of tuberculous meningitis seem pronounced, in a few weeks the patient completely recovers, in which instances the assumption that there is a pseudo-meningitis of hysterical origin seems necessary. Of course, the previous history of the patient, the family history, etc., have to be taken into consideration before such a diagnosis, which we think is always very risky, can be even thought of.

The occurrence of meningitic symptoms as a consequence of worms, which Devaux (cf. lit.) has upheld, is certainly exceptional, and can hardly, for any length of time, give rise to an error in diagnosis.

With sufficient care we can easily avoid confounding meningitis with eclampsia infantum.

Prognosis.—The prognosis in every case of meningitis is very serious; we are never in a position to predict with any certainty the outcome, not even when everything seems to be going on very favorably, and grave symptoms have not declared themselves. These may suddenly develop in one night, and a patient whom we have left in fairly good condition in the evening may the following morning be hopelessly ill. On the other hand, we should not give up our patient too soon; the gravest symptoms may fade away, and improvement is still possible even where the case seems desperate. Undoubtedly, however, meningitis is one of the most serious diseases, and one in which recovery is rare, the epidemic cerebro-spinal

meningitis being the only form which runs occasionally a more favorable course.

Partial recoveries are much more frequently seen than absolute ones; if, for example, in the course of meningitis, a hæmorrhagic inflammation of the inner ear develops, this gives rise to permanent deafness, which in younger children, as a rule, leads to deaf-mutism; or if purulent inflammation of the eyeball, a panophthalmitis or a choroiditis coexist with the meningitis, this may entail a grave disturbance of sight, even phthisis bulbi, and complete amaurosis. In either of these cases the meningitis may get well, but in one deafness, in the other impairment or loss of sight, and in the most unfavorable cases both remain behind without the development of any mental defects. Blindness may also be a consequence of an optic neuritis, which does not get well, but causes shrinking of the optic nerve and atrophy of the disk. Cases of meningitis confined to the convexity sometimes recover, leaving a more or less marked feeble-mindedness.

Treatment.—The treatment is first to be directed against the inflammation, and later endeavors should be made to aid absorption of exudates if such be present. The former is to be combated by local bleeding and the application of cold, also by inunctions of mercurial ointment, four to eight grammes (ℨ j to ℨ ij) a day to the shaved head. In some cases we shall succeed with such measures in lessening the severity of the symptoms, but often little or nothing is achieved by them. Painting the shaved head with tincture of iodine is objectionable, owing to the disagreeable and painful tension which it produces, and which is but little alleviated by ice. That free purgation with large doses of calomel actually produces an antiphlogistic effect can not be proved, but there is no reason why it should not be tried, the drug being given until the characteristic stools appear. The absorption of exudates is attempted by large doses of potassium iodide, 4 to 6 grammes (ℨ j to ℨ jss) a day in hot milk, a medication which is especially indicated in the gummatous form of meningitis.

During coma the patient may be put into a tepid bath (90° to 93° F.) and cold water (66° to 60° F.) be poured over his head. These cold-water affusions may be continued for eight or ten minutes, with the result, frequently, of actually rousing the patient out of his unconsciousness, an improvement, however, which generally does not last very long. The

repetition of this procedure several times a day is therefore necessary, notwithstanding the considerable difficulties with which it is (at least in private practice) attended.

Symptomatically the agonizing headache and the jactitations may be met with morphine. The same drug is used against the obstinate vomiting, which is hard to treat, and indeed may resist all efforts. It may happen that all internal medicines, cracked ice, champagne, opium, aromatic tinctures, etc., as well as all applications of spiritus sinapis, etc., remain without effect; then we are forced to resort to morphine, the subcutaneous administration of which generally accomplishes more than all remedies previously used. The regulation of the bowels should of course never be overlooked.

We can only, then, reasonably hope for success from our therapeutic efforts if we pay careful attention to the nutrition of the patient. As soon as this is left out of sight the battle is practically lost in spite of all medicines and inunctions. More than in any other disease it is here the chief task of the physician to see that the strength of his patient is kept up, so that he be fit if necessary to stand an illness of weeks, and more than in any other disease is here the prolonged use of wine indicated, and is much more important than all drugs. Besides wine, a tablespoonful of beef-tea is to be given every hour. This is prepared by gradually heating lean beef, cut into small cubes, after the addition of a little salt, in a lightly closed glass bottle over the water-bath, and cooking it until the pieces are completely disintegrated. Two pounds of meat furnish about a cupful of beef-tea.

In very exceptional cases operative measures are indicated, namely, where we have sufficient reason to suspect the existence of an exudation in the ventricles, which would manifest itself by an aggravation of the symptoms of increased intracranial pressure. Trephining may then be resorted to if the circumstances are in other respects favorable.

The treatment of tuberculous meningitis in children has to be conducted according to the plans just laid down, with this difference—that the inunctions of the head with mercurial ointment are to be replaced by the administration of calomel, three to five centigrammes (grs. ss. to j) every two hours. Besides, the inunctions of the head with iodoform ointment, lately so warmly recommended, should be tried; but here, too, the preservation of the strength must be our chief aim. Milk,

with the addition of a little Hungarian wine or a few drops of cognac, should always be kept ready.

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PART II.

DISEASES OF THE CRANIAL NERVES.

IF we once have a clear idea that in the cranial nerves we have to distinguish the origin, which in all probability is found in the cortex and the nuclear region of the medulla oblongata, from the partly central (intracerebral), partly peripheral (extracerebral) course, it is self-evident that the diseases of the cranial nerves are divisible into those which affect the nerve at its origin, the center, and those which affect it in its course. As we shall come to deal in the next part of our book with the affections of the brain substance proper, it necessarily results that in the following chapters we must either touch upon things which properly belong to Part III, or that in the latter we shall not be able to avoid some repetition. Neither of these courses is without objections ; still, from a practical point of view, we have deemed it best to treat of the diseases of the cranial nerves here *in toto*.

The central lesions of the cranial nerves often form merely a part of a more general disease of the nervous system. Those of peripheral origin occur also independently—for instance, as the result of exposure to cold, traumatism, etc. In very many cases we are not able to determine definitely whether the disease has a central or a peripheral origin. For a clear understanding of the following chapters, a knowledge of the anatomy of the parts naturally can not be dispensed with. Some remarks bearing on this, which, of course, are not meant to take the place of a detailed study, have therefore been inserted at the head of each chapter to recall to the reader's mind in outline the necessary anatomical relations.

CHAPTER I.

DISEASES OF THE OLFACTORY NERVE.

THE olfactory nerve begins in a small pyramidal lobule, the tuber olfactorium (caruncula mamillaris), the base of which is situated in front of the anterior perforated space. At its beginning, the nerve
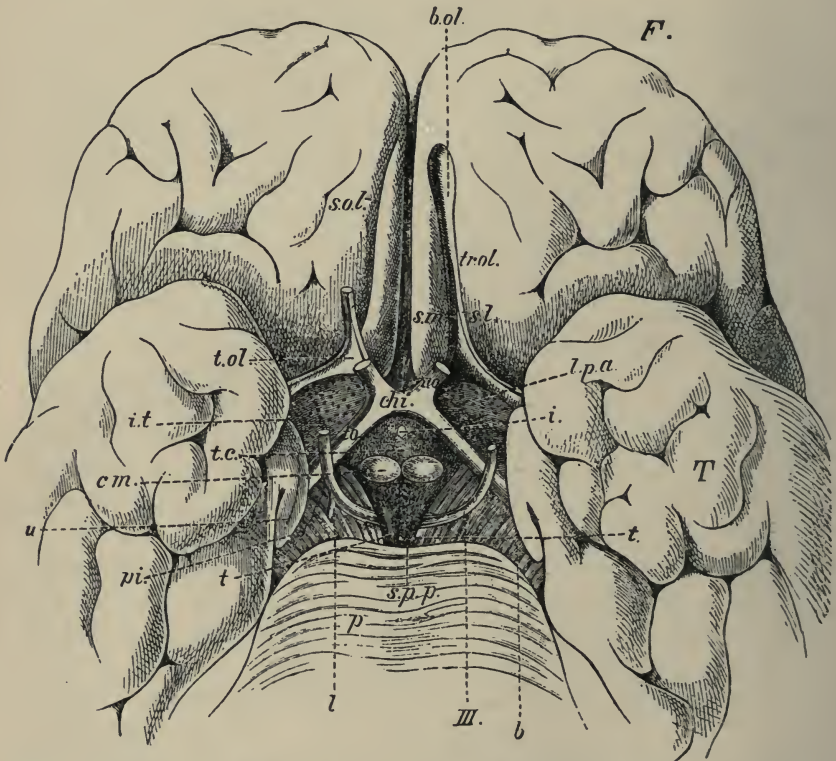
Fig. 2.—ANTERIOR AND MIDDLE PORTIONS OF THE BASE OF THE BRAIN. *F.*, frontal lobe. *T.*, temporal lobe. *b. ol.*, olfactory bulb. *tr. ol.*, olfactory tract. *t. ol.*, tuber (trigonum) olfactorium. *s. m.*, middle, *s. l.*, lateral root. *i.*, infundibulum (cut off). *c. m.*, corpora albicantia. *l. p. a.*, anterior perforated space. *s. p. p.*, posterior perforated space.

is broad, but narrows into a band somewhat prismoidal on section, which is called the olfactory tract, and which in its turn ends in an oval gray swelling, the olfactory bulb (*vide* Fig. 2). From the lower aspect of this bulb, which lies on the cribriform plate of the ethmoid bone, two groups of fibers pass through the little openings of the bone into the nasal cavity, and it is only the sum of these filaments

(the fila olfactoria) which can be looked upon as the nerve of smell in the strict sense of the term. The olfactory tract and bulb are parts of a cerebral lobe, the so-called olfactory lobe.

The origin and the course of the roots of the olfactory nerve (the striæ olfactorii, Schwalbe) are not known. It is, however, generally agreed that there are three roots. The outermost, the strongest, is said to be traceable into the island of Reil. Schwalbe supposed the existence of a lateral root (radix lateralis, seu longa, seu externa) originating in the hippocampal convolution, and of a median (radix medialis, seu interna, seu brevis), coming from the gyrus fornicatus. Others have looked upon the anterior commissure and the corpus striatum as the starting points of the olfactory nerve, but nothing positive is known. An olfactory center has been assumed in the gyrus hippocampi and in the gyrus uncinatus. Lately Zuckerkandl has claimed that the cornu Ammonis is a part of the olfactory center.

Notwithstanding the fact that the affections of this nerve are not of very great practical importance, they afford a great deal of interest, because they may under certain circumstances (i. e., if a careful clinical description is followed by an exact and accurate post-mortem account) give us some information about the anatomical and physiological questions concerning the course and origin of the nerve, and again because they may attain a considerable importance and value in the diagnosis of certain cerebral diseases.

The olfactory nerve may be diseased in its central or in its peripheral portion. In the former case it may be the olfactory center which is affected, or the conduction may be interfered with somewhere in the course of the intracerebral paths.

Since, as we have stated, the situation of the olfactory center is not definitely known, we can not be expected to know much about its diseases. It would appear, however, that it may be affected by destructive as well as by irritative lesions; the latter manifest themselves by hallucinations, the former by loss of smell (anosmia). Among the diseases in which hallucinations of smell occur are various psychoses, also migraine, tic douloureux, epilepsy, and tabes. Usually the smell which such patients describe is bad, disgusting—of fæces, sometimes of poisonous plants, putrid substances, etc. (kakosmia)—and it is rare for them to imagine that they smell pleasant substances. One of my cases, who, owing to an ocular paralysis, was treated with the galvanic current passed through his head from one

side to the other, declared that he smelled oil of lavender from the moment the current was closed until it was again broken. This seems to point to the possibility that by the galvanic current the olfactory center may be stimulated. Central anosmia is sometimes observed in cerebral lesions which cause hemiplegia and aphasia, the disturbance being confined to the nostril on the same side as the lesion. Anosmia is also known to occur in hysteria and in old age; in the latter case it is probably to be attributed to atrophy (senile anosmia). Cases have been repeatedly noted in which tumors of the anterior fossa of the skull, exostoses, meningitis at the base of the frontal lobe, have given rise to anosmia.

Interference of conduction in the olfactory nerve may be assumed in cases where there is a history of traumatism—a fall upon the head, more especially upon the occiput. According to Carbonieri, complete loss of smell suggests strongly disease of the olfactory tract or bulb.

The treatment in the central affections of the olfactory nerve must of course be directed against the underlying disease.

Of greater practical interest are the peripheral affections of the olfactory, which chiefly consist in a decrease of the power of smell. Leaving out of consideration the common cases in which an acute or chronic nasal catarrh causes partial or even temporarily complete anosmia, the sense of smell may be affected as the result of abnormal dryness of the nasal cavity (diminution in the secretion of tears in trigeminal anæsthesia, diminished flow of tears into the nasal cavities in facial paralysis). Not rarely certain occupations give rise to anosmia, which is sometimes associated with a tolerance of disagreeable odors which at first were highly obnoxious to the workers. Such anosmias are to be found in soap-boilers, catgut spinners, tanners, skinners, and butchers, whose sense of smell is often considerably dulled; again it may be due to disturbances in nutrition, to the action of caustic substances, or injury to the peripheral nerve endings—effects which are due to the chemical composition of the inhaled substances. Thus we have observed loss of the sense of smell in those working in chlorinated lime, while it was found to be diminished in working men occupied with the pulverization of chromeironstone. Stricker has also known it to occur in an entomologist in consequence of the protracted inhalation of ether.

The treatment consists in faradization (Beard and Rockwell)

and galvanization (Fieber) of the nasal cavity, or painting with a one-per-cent solution of strychnine (in olive oil). The use of irritative snuff powders has repeatedly been recommended for anosmia of peripheral origin, but has frequently been used without benefit. Spontaneous recovery is not rare. Finally, we may say a word or two about the method of testing the sense of smell. All those substances which irritate the trigeminus should be avoided, as, for instance, acetic acid, smelling salts, snuff, tobacco; the patient would feel what he can not smell, and we might be thus led astray in our conclusions. Cologne water, oil of rosemary, musk, camphor, anise, oil of turpentine, asafœtida, sulphureted hydrogen, are sufficient for most tests. That each nasal cavity must be tested separately goes without saying.

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CHAPTER II.

DISEASES OF THE OPTIC NERVE.

THE optic nerves derive their fibers from the occipital lobes, the optic thalami, the outer and inner geniculate bodies, the anterior corpora quadrigemina, and the cerebellum (through the superior peduncle of the cerebellum).

What are known as the optic tracts before the chiasm is reached, after this point become the optic nerves. These are round hard cords, about four millimetres in diameter, which, passing in a diverging direction through the optic foramina, enter the orbits and reach the eyeballs after their passage through the orbital fat. Here they pass the sclerotic and choroid and spread themselves over the fibrous layer of the retina. The outer covering of the nerve, which is a process of the dura mater, is called the dural sheath; the process of the pia, the inner or pial sheath. The two are separated by a space which belongs to the lymphatic system, the so-called intervaginal or subvaginal space. The arteria centralis retinæ, a branch of the internal carotid, enters the optic nerve about fifteen or twenty millimetres from the eyeball and runs together with the vein of the same name in the substance of the nerve to the retina.
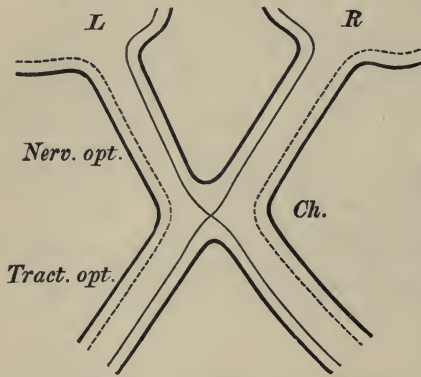
Fig. 3.—DIAGRAM SHOWING THE COURSE OF THE OPTIC FIBRES IN THE CHIASM.

The chiasm, which is formed by the union of the optic tracts, is a flattened four-sided body, in which the crossing of the optic fibers takes place. This crossing, as we now know with a fair amount of certainty, is, however, only partial, a semidecussation. The fibers from the outer half of the retina (represented by an interrupted line) pass to the center without decussating, while those of the inner half

cross over and pass to the centre of the opposite side (cf. Fig. 3). Each occipital lobe, therefore, receives fibres coming from the temporal as well as from the nasal half of the retina. Thus, for instance, the left receives fibres from the outer temporal half of the left and from the inner nasal half of the right retina. In diseases of this lobe, therefore, images falling upon the left half of the retina, or, in other words, those which lie in the right half of the field of vision, are no longer perceived—right-sided hemianopia.

The optic tract, the superficial fibres of which can be traced into the white covering of the pulvinar (the so-called stratum zonale thalami), originates by two roots—an outer, much stouter, the end ganglia of which are the anterior corpus quadrigeminum, the outer geniculate body, and the pulvinar, and by an inner root which can be easily followed to the inner geniculate body (Wernicke). These end ganglia of the optic tracts form at the same time the terminal points of certain fibres of the corona radiata, which run in a sagittal direction forward from the occipital lobe, and are connected with the pulvinar, the brachium anterius of the quadrigeminal body, and the outer geniculate body. This bundle of fibres is the sagittal medullary tract of the occipital lobe, or what is called the optic radiation, and is designated in the diagram by *s* (*vide* Fig. 4).

The exact localization of the cortical centre of vision has not as yet been established. According to Ferrier, it is in the angular gyrus; according to Munk, it is in the convex surface of the occipital lobe.

It would be beyond the scope of the present work to treat *in extenso* of those diseases of the optic nerve which belong strictly to the domain of ophthalmology; they can be considered here only as far as they are connected with the nervous system. To these belong, first of all, certain inflammatory conditions which act upon the intraocular end of the nerve, the papilla (disk), and give rise to what we therefore term papillitis (choked disk). The name optic neuritis, which is frequently used as a synonym for papillitis, is inexact, because it may imply an affection of the whole nerve trunk.

The papillitis, choked disk (*Stauungspapille*, as the Germans call it, after von Graefe, 1859), is frequently, although not always, met with in cases of intracranial tumors, and is (according to von Graefe) to be attributed to a high grade of venous engorgement, produced by an impediment to the reflux of the venous blood into the skull cavity. Later, when Schwalbe had discovered that there was a communication between the fluid

contents of the skull and the intervaginal space of the optic
nerve, it was shown that the subdural space was distended
with a serous inflammatory fluid, and that the optic nerve at its
passage through the lamina cribrosa of the sclerotic becomes
compressed (Schmidt-Rimpler). Finally, Deutschmann (cf. lit.)
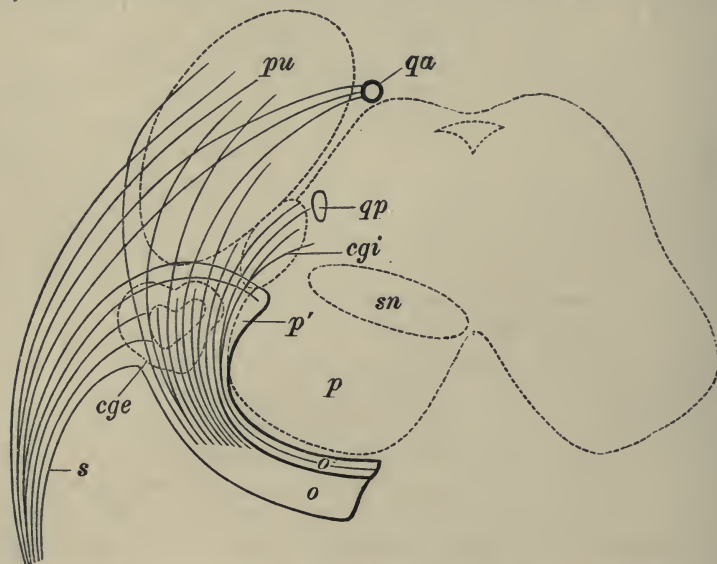
Fig. 4.—DIAGRAM SHOWING THE ORIGIN OF THE OPTIC NERVE. (After WERNICKE.)
p, crusta of the crus cerebri; *sn*, substantia nigra; *cgi*, inner, *cge*, outer geniculate
body; *qp*, brach. post. corp. quadr.; *qa*, brach. ant. corp. quad.; *pu*, pulvinar; *s*, optic
radiation.

has put forth the view that papillitis is not caused by mechan-
ical influences, but that it is due to the action of pathogenic
organisms which enter from outside. How far this view is
correct further experience will show. Besides the pure papil-
litis there is also found a papillo-retinitis, the ophthalmoscopic
picture of which differs from that of the former affection, and
which is to be referred to a meningitis, which has advanced
along the sheath of the optic nerve.

A pure papillitis, as we have said, is chiefly found in intra-
cranial tumors. Patients in whom a brain tumor is suspected
ought to be examined for choked disks even if they do not
complain of any subjective symptoms pointing to it, because
sight may, even if the disk is markedly swollen, remain normal
for a long time. Only when the nerve or the chiasm is strongly
compressed does amblyopia or amaurosis occur in the early
stages.

The seat of the tumor has nothing to do with the occurrence of papillitis. Basal neoplasms can, through direct pressure upon the optic nerve, cause a simple atrophy of the same. Nor does the nature of the tumor play any part here. Gummata, tubercles, entozoa (cysticerci, echinococci), carcinomata, gliomata—any one of these may produce a papillitis, which is usually bilateral (in ninety-three per cent of the cases, Annuske and Reich), although the processes need not necessarily be equally developed in both eyes.

Of practical importance are the sudden spells of blindness which occur sometimes in the course of a papillitis, termed by H. Jackson epileptiform amaurosis. They are probably due to a temporary swelling of certain tumors and the consequent compression of certain areas of the brain or the vessels (Leber) distributed to them. These attacks may last for hours or days, and either disappear completely or leave a permanent increase in the amblyopia. The ophthalmoscopic examination does not teach us anything about this periodical blindness.

A papillitis rarely ever gets well; in by far the greater number of cases a papillitic atrophy and total amaurosis takes place, first in one and then in the other eye. Cases in which one eye is seriously damaged while the other remains perfectly well are extremely rare. I have, however, had occasion to observe an instance of this with Magnus. More frequently both eyes become diseased, one soon after the other. Dropsy of the ventricles may give rise to a simultaneous amaurosis of both eyes.

Papillo-retinitis is not very rare in tubercular basilar meningitis; in epidemic cerebro-spinal meningitis it is exceptional. Chronic cerebral affections of children often lead to it, the amaurosis in these instances usually developing quickly, while the general symptoms become intensified.

Inflammations of the optic-nerve trunk occurring alone may be caused by cold, febrile diseases, disturbances in menstruation, and hereditary influences. On ophthalmoscopic examination either nothing remarkable or only a slight blurring of the disk is recognizable, because the inflammation affects more especially that part of the nerve which is behind the eyeball (retrobulbar neuritis of von Graefe). The disturbance of vision usually begins gradually, and is confined either to the periphery of the field of vision or it consists of a central amblyopia or a circumscribed central amaurosis. It does not terminate in com-

plete blindness; frequently only marked disturbance of color vision remains.

To the neurologist the cases of optic neuritis in patients with a neuropathic family history are of extreme interest. Such persons usually suffer even in early youth from migraine, nervous palpitation of the heart, vertigo, sometimes also from epileptiform attacks. Between the ages of twenty and thirty they begin to complain of trouble with their sight, either of subjective light or color sensations or else that objects appear to them enveloped in a dense mist; within from four to six weeks they may become completely blind, but their blindness as a rule does not persist, but gives place to a central ambly-opia with normal sight at the periphery of the field of vision. The prognosis differs markedly in different families. It is of interest to note that as a rule only the male members of the family are wont to be affected by the disease.

In the second place we will consider atrophy of the optic nerve. It consists in a wasting of the nerve elements, and may be either primary (genuine) or inflammatory, the consequence of a previous neuritis. It may also affect the trunk of the nerve as well as the intraocular end of it. If the nerve, besides the wasting of its pulp, also undergoes a diminution in its volume, so that it appears like a gelatinous grayish-yellow cord, the atrophy is known as gray degeneration.

Tumors and inflammatory exudates, as well as splinters of bone, may by pressure, by shutting off the blood supply (as, for instance, in embolism of the arteria centralis retinæ), and through interference with the nutrition lead to atrophy.

The progressive atrophy, or, as it is better termed, progress-ive gray degeneration, which may be of cerebral or spinal origin, is characterized clinically by a diminution in the acute-ness of the central vision, a contraction of the whole visual field, and disturbance of the color sense. In the ophthalmo-scopic examination the bluish-white discoloration of the disk and the atrophic excavation of the nerve (due to wasting of the substance of the disk) are very apparent. The acuteness of vision, grows gradually but progressively less, and months and years may pass before complete amaurosis is developed. On the other hand, the whole process may run its course in two or three weeks. The contraction of the field of vision is rarely concentric; usually the defects are in one direction only, and are often sectorial (Leber). Enormous contraction of both fields of

vision, with at the same time normal acuteness of sight in the center, which was eventually followed by blindness, has been observed by Schweigger. The disturbance in color vision is at first limited to the perception of green, which is confused with white or gray, the perception of blue and yellow being relatively longest retained. The atrophy develops bilaterally, although one eye alone may at first be affected, and the other eye remain intact for years.

Foci of softening in the brain, progressive paralysis of the insane, sometimes also epilepsy, are the cerebral diseases in which the affection is not rarely observed. It is besides also noted in multiple sclerosis, although in this disease it never leads to total amaurosis, a fact which Charcot is in the habit of emphasizing in his lectures.

More important is the fact that in locomotor ataxia optic atrophy is comparatively frequent. Wharton Jones (British Medical Journal, July 24, 1869) makes the sympathetic responsible for this, assuming that the paralysis of the vaso-motor nerves, producing first hyperæmia, leads finally to atrophy of the optic nerve. This explanation, however, is at once overthrown by the fact that in the optic atrophy of tabes there are at no time any traces of hyperæmia. We shall again have to refer to this question in our account of tabes, where we shall also consider the treatment of optic atrophy.

Congenital optic atrophy can sometimes be traced to hereditary influences, or to consanguinity of the parents; sometimes it is due to hydrocephalus. Injury to the skull in consequence of instrumental interference at birth very rarely has anything to do with it.

The diseases of the chiasm and optic tract may be considered together, since they possess one symptom in common which is of special interest to the neurologist—viz., hemianopia. This symptom is also found associated with diseases of the centre of the optic nerve in the occipital lobe. It is the only form of visual disturbance where one can with certainty diagnosticate a central affection of the optic nerve. It is likely to be of cortical origin if the hemianopia occurs suddenly as the only symptom, there being no change to be found on ophthalmoscopic examination; whereas if other symptoms accompany it—aphasia, hemiplegia, etc.—this idea of a cortical lesion must be given up. By hemianopia in general we mean a loss of one half (the right or the left) of the field of vision, so that patients

3

affected with right-sided hemianopia see only the objects which
are in the left half of their visual field, whereas those to the
right are not perceived. If the disturbance affects the halves
on the same side of both eyes—that is, the nasal on the one, the
temporal on the other—we call it a homonymous hemianopia.
If in both fields the temporal halves are lost, this constitutes
what is known as temporal hemianopia, which is of rarer occur-
rence; the absence of both nasal halves of the field of vision
does not seem to occur, and the superior and inferior hemian-
opia, where the line of division is not vertical but horizontal,
seems to be extremely rare.

The explanation of the hemianopia in lesions of the cortical
center for sight is quite evident if we accept, as is now gener-
ally done, the existence of the above-described semidecussa-
tion of the fibres in the chiasm. The path from the optic tract
to the cortex of the occipital lobe may be divided into the fol-
lowing segments (Wernicke): The first includes the optic radia-
tion in the occipital lobe, the lesions of which give rise to
homonymous hemianopia without any other focal symptoms;
the second will include the place where the fibres of the optic ra-
diation enter the internal capsule, and the ganglia of origin of the
optic tract, the pulvinar, and the outer geniculate body—hemi-
anopia and hemianæsthesia; the third will include the optic
tract in its course at the base of the brain—hemianopia with
hemiplegia. If in the region of the visual centre or the optic
radiation a bilateral focal lesion occurs, then we may have
complete blindness setting in with an apoplectiform attack.
This is in reality a bilateral hemianopia (Wernicke). Of diag-
nostic value in these cases is sometimes the so-called hemian-
opic pupillary reaction (Heddaeus, Wernicke), although it is
not absolutely reliable. With the mirror of the ophthalmo-
scope we reflect the light first upon the left, then upon the
right half of the retina, and observe the pupillary reaction.
If the reflex occurs normally, the optic tract must be intact,
and the disturbance must be due to a bilateral lesion of the
optic radiation in the occipital lobe, or in the cortical center.
If the reflex is not obtained we must assume a lesion of the
optic tract of the corresponding side. Light perception and
pupillary reflex go in this case hand in hand.

In diseases of the chiasm hemianopia has been repeatedly met
with, but in this case we have not a homonymous, but a bitem-
poral hemianopia, as in the case of Oppenheim, where gumma-

tous disease of the chiasm was responsible for the disturbance (cf. Virch. Arch., 1886, Bd. civ, 2, p. 306). Quite lately the same author has described an " oscillating " bitemporal hemianopia in diseases of the chiasm, which he considers as pathognomonic of basal cerebral syphilis (cf. lit.).

If the tissue injured by the lesion which has caused the hemianopia is capable of regeneration, as may be the case where we have a hæmorrhage or an inflammation, the defect will pass off completely, whereas if this is not the case the trouble remains stationary, without, however, any additional disturbance of sight. Such a condition, which often develops as the consequence of an apoplexy, may persist for years, but no second attack, by which the centres of the other tract also may be disturbed, is to be feared, as such a thing has never been observed.

The examination in a case of hemianopia may (roughly) be conducted in the following manner : The patient is to be placed at a distance of about two feet from the examiner, and, if the right eye is to be examined, asked to cover his left eye with his hand, while with the right eye he fixes the left of the examiner
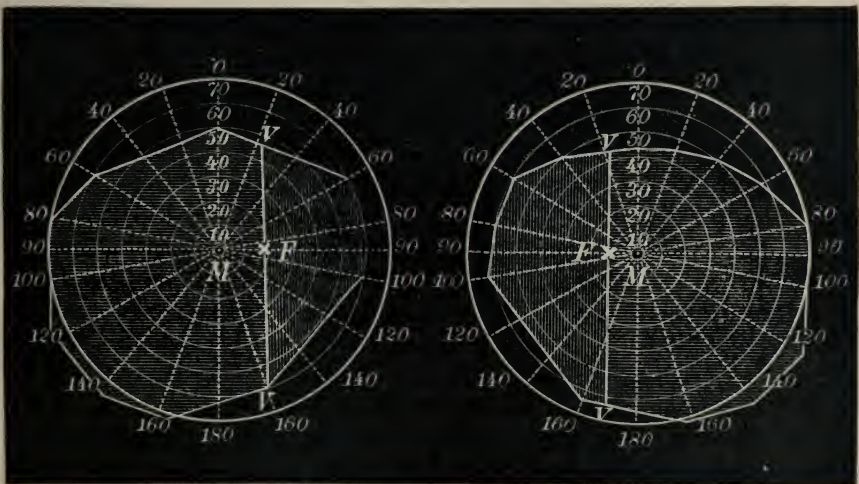
Fig. 5.—Field of Vision of the Left and Right Eye. (After Förster.)

who covers his right eye. The examiner then holds up his finger between the patient and himself, and moves it in different directions as far as the border of his own field of vision, the patient at the same time being asked how far out he is able to see

the finger. The examiner is thus enabled to notice every motion of the patient's eye toward the object, and, judging from his answers, can compare the patient's field of vision with his own. Instead of the finger a small piece of white paper fastened on
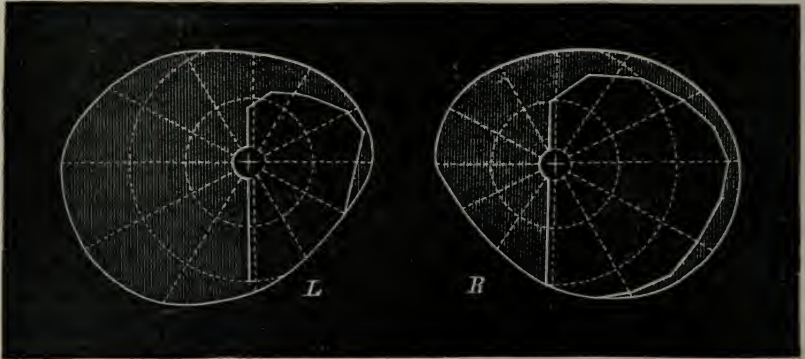
Fig. 6.—Field of Vision of the Left and Right Eye in Left-sided Hemianopia. (After Gowers.)

a dark penholder may be used in a similar way. These tests should be made in a good light (Donders, Gowers).

The more extensive defects can always be found out by this method; for slight ones a perimetric examination is indispensable. An accurate determination of the field of vision with the help of the perimeter can only be attained by practice. A description of the instrument and its use is here not necessary. Figs. 5 and 6 illustrate (1) the normal fields of the left and right side; (2) the fields in a case of left-sided hemianopia.

The so-called flitting scotoma (amaurosis partialis fugax, or temporary hemianopia) has in all probability also to be regarded as an affection of the centre for vision. The disturbance comes on in paroxysms. At first a dark spot appears in the field of both eyes, which increases in a crescentic or horseshoe form. It begins to scintillate and becomes bounded by a bright zigzag line of brilliant colors. If this has after fifteen or twenty minutes reached the border of the field of vision, it disappears from the centre toward the periphery and the field clears up again. Most probably in all cases the affection is bilateral. The attacks, which last from a half to three quarters of an hour, occur with variable frequency, sometimes only once during the whole life, and it is interesting to note that they are almost always associated with attacks of migraine. Of the causes

nothing is known, although the belief that hard mental work-
ers are especially prone to it is not without foundation ; but
there are numerous cases in which we are reduced to regard
sexual and alcoholic excesses, cold, etc., as ætiological factors.
As we are not acquainted with any remedies for the disease,
we have to be satisfied in prescribing tonics and strengthen-
ing diet, quinine, and, above all, mental as well as bodily rest.

The nature and the seat of those forms of amblyopia which
develop under the influence of certain toxic substances are still
obscure. Material changes in the retina are usually absent,
and only quite late may a slight or partial discoloration of the
papilla become visible.

To this class of substances belong more especially alcohol,
tobacco, and lead.

The alcoholic amblyopia is the most frequent form. In the
mildest cases it manifests itself as a simple central amblyopia
without distinct scotomata, without disturbances in color vision,
and without contraction of the visual field ; whereas in the most
serious forms, which may occur after excessive indulgence in
spirits, especially in persons of previously moderate habits,
there may be an acute, almost total blindness. After the recur-
rence of such attacks a more severe form of atrophic disease of
the optic nerve may develop, with which is associated discol-
oration of the whole disk. Central colored scotomata and sim-
ple scotomata, disturbances in color sense in the whole visual
field, are then not rare. The ophthalmoscopic examination
does not reveal anything very characteristic. Vision rarely
becomes less than $\frac{1}{10}$ to $\frac{1}{20}$, and complete recovery even in the
most marked cases is possible. The few examinations of the
optic nerve which have been made after death seem to indicate
that alcohol exerts a direct injurious action upon the nerve it-
self. The latter has several times been found in a state of fatty
degeneration with or without compound granular corpuscles
and thickening of the interstitial tissue which contains the ves-
sels (Erismann, Leber, cf. lit.). Since it has recently also been
shown that alcohol can act in a similar way upon the peripheral
nerves this pathological condition is more easily understood.

Similar in its development and in its course is the so-called
tobacco amblyopia, which, *cæteris paribus*, is, however, more
rarely met with than the alcoholic form, and is more benign,
inasmuch as it usually passes off after the cause is removed.
The diagnosis is, as a rule, easy enough, as other signs of

chronic nicotine poisoning (digestive disturbances, palpitation of the heart, insomnia) are rarely wanting. The disease seems only to occur among those who use tobacco in some form or other, in smokers or chewers, while the workers in tobacco, who are exposed to the inhalations of the tobacco dust and of a certain amount of nicotine, seem, so far as experience goes, not liable to the complaint.

The one form of amblyopia which has been more carefully studied than any other, but which nevertheless is not much better known or understood than the affections which we have just treated of, is lead amblyopia (amblyopia saturnina), in which the field of vision may remain normal or in which there may have developed central scotomata or contraction of the visual field. Pronounced neuritis, with decided swelling of the disk and with peripapillary hæmorrhages, has been observed, and the termination in complete amaurosis is not rare.

Under certain still unknown conditions a sudden bilateral blindness may develop without previous decrease of vision— amaurosis saturnina. It is commonly preceded by lead colic. The affection, which bears a certain resemblance to the amaurosis of uræmia, may sometimes improve with remarkable readiness after the removal of the injurious cause.

In a given case we should, for the sake of confirming our diagnosis, never fail to search for other cerebral symptoms common to chronic lead poisoning, such as epileptiform attacks, hemiplegia, speech disturbances, and so forth.

About the relative frequency of the disease no definite statement is possible, nor do we know which particular occupation in the lead industry is the most dangerous, or after how long an exposure eye trouble develops in lead workers. The *rôle* which the so-called individual predisposition plays in this connection seems as important as it is obscure.

In the treatment of the alcoholic amblyopia, local bleeding with Heurteloup's cups, active purgation, diaphoretics, and later strychnine injections are of service. In tobacco amblyopia the treatment is the same, but bleeding may be dispensed with. In the saturnine form purgatives are indicated, also opium and subcutaneous injections of morphine. In all cases, however, the prompt and permanent removal of the injurious agent is a *sine qua non;* where this can not be done the outlook for recovery is always very doubtful.

Besides the substances mentioned, quinine and mercury

may lead to disturbances of sight, which in their course resemble those just described.

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CHAPTER III.

DISEASES OF THE NERVES SUPPLYING THE OCULAR MUSCLES—I. E.,
THE THIRD (MOTOR OCULI), THE FOURTH (PATHETICUS), AND
THE SIXTH (ABDUCENS).

THE third nerve emerges from the brain at the inner margin of the crus close to the anterior border of the pons; it passes obliquely forward and outward, reaches the outer wall of the cavernous sinus, enters it, and then divides into two branches, which, passing through the sphenoidal fissure, enter the orbit. The upper division, which supplies the levator palpebræ superioris and the rectus superior, is the smaller of the two. Of the three branches of the lower division, the one supplying the inferior oblique is the longest; the two others, one of which goes to the inferior rectus, the other to the internal rectus, are shorter. The longest branch, that of the inferior oblique, gives off a short root to the ciliary ganglion, the filaments of which are distributed to the ciliary muscle (tensor choroideæ) and to the constrictor of the iris (sphincter pupillæ); consequently these intrinsic muscles of the eyes are also innervated by the third nerve, while the dilator pupillæ, on the other hand, is provided for by the sympathetic.

Fig. 7.—CROSS SECTION THROUGH THE REGION OF THE ANT. CORPORA QUADRIGEMINA.

qu.a. anterior corpora quadrigemina ; g.c. gray matter around the aqueduct of Sylvius ; aq, aqueduct of Sylvius; nIII nucleus of the third nerve ; hl. posterior longitudinal bundle ; r. k. red nucleus (tegmentum) ; sn, substantia nigra (locus niger) ; p, cerebral peduncle.

The nuclei of the third nerve, a column of multipolar ganglionic cells, lie above the posterior longitudinal bundle, between it and the aqueduct of Sylvius, and the root fibres coming from them divide into several fasciculi, pierce the posterior longitudinal bundle, the tegmentum, with the red nucleus and the substantia nigra, and emerge from the brain at the place shown above (cf. Fig. 7).

Experimental as well as clinical observations seem to indicate that in the collection of ganglionic cells of this nerve nucleus there exist three centres, the anterior of which is the centre for the ciliary muscle (accommodation); the next the centre for reflex stimulation of the iris by light; the third, by far the largest, the centre for the extrinsic ocular muscles (Gowers). Observers, however, by no means agree with regard to the number and position of the individual oculo-motor nuclei or centres. The view held by Gowers is diagrammatically illustrated in Fig. 8.

That there exists a cortical centre for the ocular muscles and the levator palpebrarum is beyond question; nothing certain is, however, known about its situation; most probably it lies in the upper or lower parietal lobe (cf. Exner, Untersuchungen über die Localisation der Functionen in der Grosshirnrinde des Menschen. Wien, Braumüller, 1881, p. 42).

Fig. 8.—Diagramatic Longitudinal Section through the Pons with the Nuclei of the Ocular nerves. (After Gowers.) *C. Q.* Corpora quadrigemina; *a a' b b'* and *c c'* represent the centres and the nerve-fibres; *a,* for accommodation, *b,* for the reflex activity of the iris, *c,* for the extrinsic ocular muscles; all three are contained in the oculomotorius. *IV,* pathetic. *VI,* abducens.

The fourth, the trochlear or pathetic nerve, is the smallest of the cranial nerves, but has the longest course within the skull cavity. It leaves the brain close behind the corpora quadrigemina at the upper surface of the valve of Vieussens; from here it takes a lateral and downward course, winds around the outer side of the crus cerebri, and reaches the base of the brain. Its course is now forward; piercing the dura mater behind the anterior clinoid process, it reaches a small channel of the cavernous sinus, and runs alongside of the third to the sphenoidal fissure, pierces its fibrous membrane, and finally enters the superior oblique muscle.

The nucleus of the fourth lies behind the collection of cells from which emanates the third nerve (Wernicke), to the ventral side of the aqueduct of Sylvius, on the posterior longitudinal bundle, in the gray matter around the aqueduct. From this nucleus the root

originates, which, passing to the mesial side of the descending root of the fifth (Fig. 9, Vd), extends as a round bundle (IV') to the posterior corpus quadrigeminum; in the substance of the valve of Vieussens it is crossed by the nerve of the opposite side, and emerges finally in the above-described manner on the side opposite to that in which its nucleus is situated.

The sixth nerve, the abducens, leaves the brain at the posterior margin of the pons, between it and the anterior pyramid. It
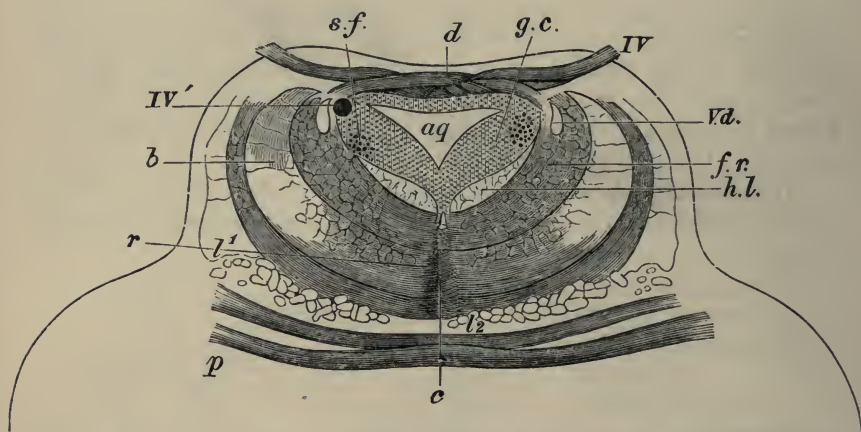
Fig. 9.—Cross-section through the Region of the Tegmentum. (After Schwalbe.) *d*, patheticus-crossing. *IV*, Patheticus at its exit. *IV'* cross-section of the patheticus in its course to the nucleus. *V.d.* descending root of the trigeminus (cross-section); *aq*, aqueduct, *g. c*, central gray substance around the aqueduct, *s.f*, substantia ferrugin. *b*, sup. peduncle of cerebellum crossing at *c*; *r*, raphe; *f.r.* formatio reticularis; *h.l.* posterior longitudinal bundle.

takes at once a forward course and passes into the cavernous sinus, piercing its posterior wall; it then runs, surrounded by the dural sheath, alongside of the internal carotid, and, emerging through the sphenoidal fissure, enters the external rectus, in the substance of which it breaks up into branches.

The nucleus of the abducens, which was at one time thought to be connected with the root of the facial nerve (hence the facial-abducens nucleus of Meynert and Stilling), lies in the floor of the fourth ventricle, from which it is separated by the ependyma. The abducens root, passing through the peduncular portion of the pons to the outer side of the pyramids into the tegmental region of the pons to the median side of the upper olive, enters finally this nucleus (cf. Fig. 10). The tegmentum behind the lemniscus is divided into three parts by the abducens (and facial) root, the inner two of which Meynert has called the motor region of the pons.

The affections of the nerves supplying the ocular muscles belong, strictly speaking, also to the domain of ophthalmology. Since, however, they are of such importance for the diagnosis and the prognosis in certain nervous diseases, it is necessary to devote a few pages at least to the description of their symptoms and the proper methods of examination.

The independent diseases of the muscles of the eyes may be of a paralytic or of an irritative (spastic) nature, the latter
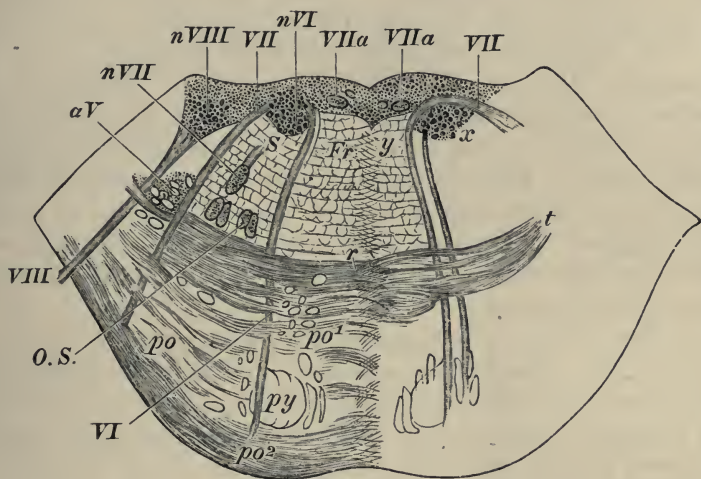
Fig. 10.—Cross-section through the Pons. (After Schwalbe.) *nVI*, abducens nucleus. *VI*, abducens. *O.S.*, upper olive. *aV*, ascending root of trigeminus. *nVII*, nucleus of facial. *nVIII*, auditory nucleus (so-called external nucleus). *VII*, emerging portion of facial root. *po*, transverse fibres of the pons which are divided into superficial *po¹* and deep *po²*. *py*, pyramidal tract.

class, however, being by far the less frequent of the two. Their seat may be central or peripheral, although we should state that an undoubted central affection of the abducens and of the patheticus has never been observed. Of oculo-motor paralysis, we are acquainted with a peripheral and a central form.

A peripheral affection may have its seat in the stem or in its branches; a central, in the nucleus or the (supposed) cortical center of the nerve. The former will be characterized by the absence of all cerebral symptoms, which, in the central form, are almost always present. It can be brought about by pathological changes in the orbit, in which case the eyeball not infrequently protrudes and becomes immobile. Further, it may develop as a rheumatic paralysis from exposure to cold

(*a frigore*), also in constitutional syphilis, diphtheria, and in other acute infectious diseases; exceptionally it is seen after traumatism. In one of my cases a man was kicked by a cow in his right eye; after the acute symptoms had passed off, a paralysis of the levator palpebræ superioris remained for months. Power of vision was not interfered with.

The central paralysis is met with in the course of meningitis, multiple sclerosis, progressive bulbar paralysis, and, above all, locomotor ataxia. It affects but rarely all the ocular muscles at the same time, but either the extrinsic or intrinsic alone. It has, moreover, been seen after poisoning with fish (Alexander, Bresl. ärztl. Zeitschr., 3, 1888). Although the diplopia of the tabetics is neither a constant nor a pathognomonic symptom of the disease, the occurrence of transient double vision in otherwise apparently healthy persons ought always to make us suspicious, and ought to induce us to subject the patient to a more careful examination. The nature as well as the anatomical seat of this oculo-motor paralysis occurring in tabes is entirely obscure.

Of the cortical oculo-motor paralysis we know little or nothing; the only well-established fact is that an isolated paralysis of the levator palpebræ superioris may be associated with cerebral affections—for instance, with a cerebral hæmorrhage and a consequent hemiplegia. The reason of this may possibly be that the levator is the only muscle supplied by ocular nerves which can be voluntarily moved on one side only (Möbius); were this only possible in conjunction with the other, the paralysis would not occur. This "blepharoptosis cerebralis" needs much further investigation.

In very exceptional instances in otherwise healthy individuals there occurs a paralysis of the eye muscles, which, defying all treatment, may persist for years without the appearance, however, of any other symptoms, whether cerebral or spinal. I have the notes of several cases of this nature; since, however, none of them came to autopsy, I shall abstain from any detailed report.

In studying the symptoms of the paralyses of the ocular muscles we shall first consider those of the oculo-motor paralysis, more especially of the complete form, in which all branches of this nerve are implicated.

The upper eyelid droops completely, and the eye can only be opened slightly by the aid of the frontalis; the movements

of the eyeball are also at fault; the eye, deviated outward as it is, can not be moved toward the nose; similarly any upward motion is impossible, as such depends upon the superior rectus and the inferior oblique. On the other hand, the outward movements are unhampered (rectus externus), while the downward motion is performed by the superior oblique, the pure action of which can here be well studied, the rectus inferior, which otherwise also assists in the downward motion of the bulb, being now inactive.

From the different directions of the axes of the two eyes there results a very apparent symptom—namely, strabismus—which may be convergent or divergent, according to the muscles affected. This strabismus, due to paralysis of the ocular muscles (paralytic), differs from that caused by spasm (spasmodic), inasmuch as (1) in the latter the deviation exists with all movements, while in the former only with those which call into action the paralyzed muscle; (2) in spasmodic strabismus the secondary deviation of the sound eye, of which we shall presently speak (cf. p. 47), does not occur.

The double vision, "diplopia," which is associated with strabismus, is especially marked at the beginning of the disturbance, before the patient has learned to suppress the "false image" seen with the affected eye, and only to pay attention to the "true image" seen with the healthy one. At first these double images cause him much annoyance until later on he learns to close the affected eye by contraction of the orbicularis, or to put the head into a position in which the affected muscle is not called into play. By these devices he not only avoids the unpleasantness of the double images, but also the consequences which the erroneous projection of the visual field entails, namely, a peculiarly disagreeable feeling of dizziness, the so-called ocular or visual vertigo, to which we shall have occasion to refer again.

With reference to the pupillary symptoms we must keep in mind the reactions present in a normal eye: the pupil reacts directly to changes between light and darkness, contracting if light is thrown into the eye, and indirectly by sympathy, in that the pupil of one eye dilates if the other is covered; it also reacts on motions of convergence and on forced accommodation, contracting in either case. All these reactions are lost in complete paralysis of the third nerve. The pupil is moderately dilated and gives no response to the influence of light or

accommodation ; if the paralysis is incomplete, and either the sphincter of the iris or the ciliary muscle or both are intact, so that in the latter case only the extrinsic muscles do not perform their function, the size of the pupil can vary and accommodation be retained.

The reflex immobility of the pupil (Erb), also called the "Argyll-Robertson pupil"—that is, where the pupil has lost its reaction to light impressions (reflex), but has retained its power of accommodation—is very frequently observed in tabetics. Besides this, the pupil in tabes is often very small, pin-head-pupil—spinal myosis.

Paralysis of the abducens, unilateral or bilateral, which also comparatively frequently accompanies locomotor ataxia, often constituting here the only initial symptom for a long time, is to be recognized by noticing that the eye, which is slightly turned inward, can not be moved outward, while all the other movements are unimpeded. In exceptional cases this is found associated with facial paralysis. A case of this sort, in which the paralysis was congenital, has lately been reported by Möbius (cf. lit.).

Unilateral paralysis of the patheticus, which supplies the superior oblique muscle, is always difficult to recognize even when the muscular system of the other eye remains perfect, and can only be diagnosticated after an examination of the nature of the double images. When there is paralysis of the oculomotorius in the other eye a diagnosis is impossible. The examination ought to be made by an ophthalmologist in order to establish the absence of power in the superior oblique. Extremely rare is the bilateral patheticus paresis, which has been noted in some cases of tumor of the pineal gland. The anatomical conditions directly underlying it are not known (Remak, cf. lit.).

A paralysis of the patheticus, superadded to a paralysis of the oculomotorius, may be recognized by the absence of the characteristic rotation around the sagittal axis, which would otherwise occur on looking down (Wernicke).

If several muscles of one eye which are supplied by different nerves are paralyzed, or if there exist paralysis of the muscles of both eyes, we speak of an ophthalmoplegia (Hirschberg, Mauthner), and we distinguish an external ophthalmoplegia if only the extrinsic, and an internal ophthalmoplegia if only the intrinsic muscles of the eye are paralyzed (sphincter, dilator, cil-

iary muscle). The so-called ophthalmoplegia progressiva (von Graefe) will be described in the eleventh chapter of this part under the name of poliencephalitis superior (Wernicke). Quite lately our attention has been drawn to a so-called recurrent paralysis of the third nerve, of which Mauthner has analyzed fourteen instances. This disease is characterized by the fact that only one, and always the same, oculo-motor becomes affected, and that the paralysis is always complete—that is, takes in all the branches. Females seem more predisposed to the affection than males. The duration of the individual attacks varies from one, three, four, to even six months. They may recur after an interval of from four weeks to a year. Other nervous symptoms—migraine, vertigo—may or may not accompany them. The attacks may recur during the whole life of the patient, and even in the intervals traces of paralysis may remain (Möbius, Remak). Whether there are instances in which the disturbance is only functional, or whether in all cases there exists a distinct organic basis, we are with our present material unable to decide definitely, and we are equally in the dark with reference to the seat of the affection, as to whether it is of peripheral or of central origin. That there are instances where the former is true is proved by a case published by Richter (cf. lit.), where a new growth in the nerve itself was found.

In a suspected paralysis of the ocular muscles we endeavor to make out in our examination any defects in the mobility of the eyeball. For this purpose the patient is asked to follow with his eyes the finger of the examiner in different directions without moving his head. In this way every asymmetry in the movements of the two eyes can be noted. If the mobility in the direction of the action of the affected muscle is defective (" primary deviation "), nystagmus-like twitching is sometimes observed on attempts at extreme rotation in that direction. But it may happen that the paresis of a muscle is not recognized if its innervation is particularly strong; then we have in the corresponding muscle of the other eye so abnormal an innervation that in the latter the effect is excessive and we get a so-called " secondary deviation " of the sound eye. This can easily be demonstrated if the presumably healthy eye is first covered with the hand and the patient endeavors to fix with the paretic eye a point which it can not reach at all or only with the utmost exertion. If, then, the fixing eye is covered, we observe whether the healthy eye be in a proper position or not, and

shall find that the latter has been moved too far in the desired direction. If this method does not give any satisfactory results, we have to examine into the nature of the double images. One eye of the patient having been covered with a colored glass, he is asked to follow with his eyes (of course, again without moving his head) the flame of a candle which is moved to and fro. If there exists paralysis or paresis in one eye, the patient complains of seeing two flames, which become the farther apart the more the affected muscle is exerted. But if now, for instance, the patient looking toward the left complains of diplopia, this may be due to paralysis of the left external or the right internal rectus, as both of these muscles move the eyeball to the left. To determine which of these two is not performing its function properly, we must ascertain from the patient whether the double images are homonymous or crossed—that is, whether the colored picture be on the same or on the opposite side to the eye covered with the colored glass (homonymous and crossed diplopia respectively). In the former case the abducens (rect. ext.) is the nerve affected ; in the latter the oculomotorius (rect. intern.). For a minute study of the double images, the reader is referred to the plates and the work of Landolt, of Paris, which has been translated into German by Magnus (cf. lit.).

The associated lateral movements of the eye to the right and to the left may be interfered with in the following ways:

1. There may exist a so-called conjugate deviation of the eyes—that is, a permanent fixation of both eyeballs to one side, which can only be overcome, and then but temporarily, by the strongest effort. We shall refer to this symptom again in our account of hemiplegia.

2. Motion of both eyes toward one side may be permanently lost. In this case we have a paralysis of the abducens of the one and paralysis of the internal rectus of the other side, and the eyes are turned not toward the affected, but toward the opposite side. In such cases the lesion is situated in the lateral portion of the pons, near the abductor nucleus. If the centres of both sides which lie close together are paralyzed, the eyes which are fixed in the middle can be moved neither to the right nor to the left, but only upward and downward, the upper eyelid moving normally (Wernicke).

3. The upward and downward motion of the eyes may be lost and only the lateral motion be possible. This form of the

associated ocular palsy, in which also both upper lids may be paralyzed, is caused by a lesion of the centres situated in the central gray matter of the third ventricle and the aqueduct of Sylvius—that is, in the region of the oculo-motor nucleus. If this be accompanied by a hemiplegia, we are justified in diag-nosticating a lesion of the pyramidal tract at the level of the upper corpus quadrigeminum, the posterior commissure, and the adjoining portion of the optic thalamus (Wernicke).

The treatment of the ocular paralyses is very problemati-cal, and rarely produces unquestionable results. Usually a trial is made with iodide of potassium, a course which may be justified if there is a history of syphilis; but this drug is frequently of no avail whatever. Electricity is used either by applying one electrode over the closed lid of the diseased eye and the other over the base of the neck, so as to pass the current through the whole course of the eye muscles, or by allowing the current to pass transversely through the head from one mastoid process to the other. Medium-sized elec-trodes should be used and a weak current be applied about four times a week, each session occupying from one to two minutes. Now and again after prolonged galvanization we are really fortunate enough to perceive an improvement in the paralysis, or even to see it disappear. That much of this is to be attributed to the treatment seems doubtful, if we remember that it is utterly impossible to stimulate the ocular muscles with the current; for the same reason an electrical examina-tion in ocular palsies is impossible (cf. Erb, Electrotherapie, page 428).

Passing over the different spasms of the eye muscles which occur in some brain diseases, we shall pay attention here only to one form with which the neurologist ought to make himself familiar, viz., nystagmus. This consists in a to-and-fro motion of the eyeballs in a certain plane, usually horizontal (*nystagmus oscillatorius*), which continues on voluntary movements of the eyes, but which is itself not under the control of the will. These movements are usually present in both eyes, and vary quite markedly in frequency and extent, according as the pa-tient is made to fix a point or to change the direction in which he is looking. The condition is supposed to be due to weak-ness of sight of both eyes, dating from early childhood—that is, to impairment in the functions of the retina at a time when these have an important regulating influence in the establish-

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ment of the normal fixation of the eyes (von Graefe). How-
ever, there are undoubtedly cases which do not belong to this
class, for it is a well-known fact that nystagmus may be a pro-
fessional disease, as it is often observed in miners who have
to use their eyes in the dark (Schroeter, Mooren, Nieden,
Foerster, and others; cf. also Hirt, Krankheiten der Arbeiter,
Part II; Die aeusseren Krankheiten der Arbeiter, page 189,
Leipzig, 1878); and, secondly, it appears in the course of
certain nervous diseases—perhaps in connection with repeat-
edly occurring cerebral anæmia (Knoll, Ueber die nach Ver-
schluss der Hirnarterien auftretenden Augenbewegungen—
Sitzungsber. d. Akademie d. Wissenschaften in Wien, Abthei-
lung III, 1886). In both these classes of cases sight is often
not diminished at all, and some other than the one given above
must be the underlying cause; and, as a matter of fact, this
nystagmus of the miners is simply due to overstrain of the eyes
in an insufficient light, while the nystagmus occurring in the
course of nervous diseases, more especially of multiple scle-
rosis, but also of tabes and epilepsy, is to be regarded as a
symptom and attributed to the same influences as the main dis-
ease. That nystagmus, finally, may also be a symptom of hys-
teria, and may persist during the whole course of the disease,
is shown by a case lately published by myself (cf. Deutsche
med. Wochenschr., No. 30, 1887, lit.).

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CHAPTER IV.

THE DISEASES OF THE TRIGEMINAL NERVE.

THE trigeminus, the stoutest of all the cranial nerves, leaves the brain by two separate roots—an anterior small, exclusively motor, and a posterior larger, the sensory portion. Its point of exit is situated at the base of the pons, where the transverse fibres of the latter are prolonged into the middle peduncle of the cerebellum. Both roots lie in close apposition, and pass into a recess—the cavum Meckelii—formed by the dura mater, and situated over the inner end of the superior surface of the petrous portion of the temporal bone. Here the posterior root forms a somewhat crescentic swelling—the Gasserian ganglion—from which pass forward the three somewhat flattened divisions, the ophthalmic and the superior and inferior maxillary nerves, the last being joined by the smaller motor root. These three branches leave the interior of the skull by the sphenoidal fissure, foramen rotundum, and foramen ovale, respectively.

The trigeminal nerve possesses two nuclei—a motor and a sensory one. The first—the smaller—is situated in the outer part of the tegmentum, and its ganglionic cells are characterized by their relatively large size (60 to 70 μ in the greatest diameter). The larger—sensory—nucleus lies external to the motor; in its collection of gray matter there are found very small ganglionic cells (20 to 30 μ in diameter).

With regard to the origin of the two roots there exist very different views, and but little is definitely known about the subject. It can not be doubted that the motor root springs from what has been decided upon as the motor nucleus, nor that there exist a number of small bundles of fibres which arise high up in the region of the anterior quadrigeminal body, and descend outside the aqueduct to the level of the exit of the fifth nerve, where they help to form the motor root. This is the so-called descending anterior, or, as Henle terms it, superior root, the section of which, a crescentic, externally convex, internally concave figure, at once strikes the eye in frontal sections of the pons (cf. Fig. 9, Vd). That the sensory

root arises from the above-mentioned sensory nucleus is probable, but not certain. On the other hand, it must be remembered that as low down as the neighborhood of the second cervical nerve there can be demonstrated in the caput cornu posterioris a layer of longitudinal medullated fibres, the highly characteristic transverse section of which, crescentic in shape, may be followed upward, as it gradually increases in size, as far as the level of the exit of the trigeminus. Suitable longitudinal sections plainly show that this longitudinal bundle forms a large part of the sensory root of the nerve. This is the so-called large ascending root of the fifth, the position of which
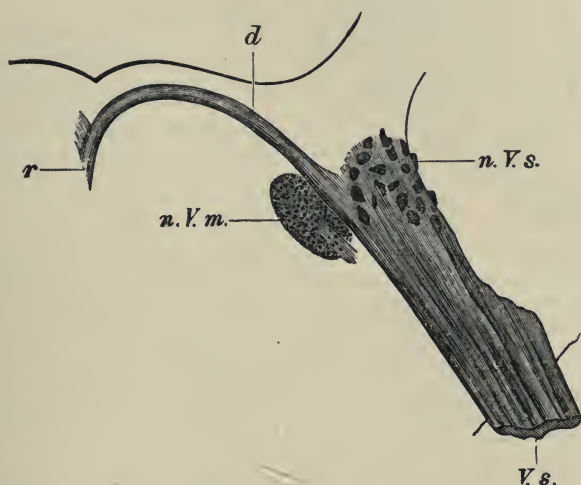
Fig. 11.—NUCLEI OF THE TRIGEMINAL NERVE. (After SCHWALBE.) *n. V.s.*, nucleus of the sensory. *n. V.m.*, nucleus of the motor root. *d*, fibres passing to the raphe. *V.s.*, sensory root.

in transverse section is represented in Fig. 12. The cortical area of the trigeminus is not definitely known as yet; still, from experiments on animals, as also from the few clinical observations which we possess, there is reason to conclude that at least so far as the motor portion of the nerve is concerned, it is located in the region of the anterior portion of the fissure of Sylvius; as regards the sensory portion we know nothing.

We shall divide the affections of the trigeminus into central and peripheral. In the first class we recognize cortical and bulbar diseases; in the second class we have to deal with either intra- or extra-cranial lesions. The trigeminus being a mixed nerve, containing in by far its larger portion only sen-

sory, but in its third branch important motor fibers, we are obliged, as there may exist in any case conditions of irritation or of paralysis, to distinguish clinically between hyperæsthesia (neuralgia, neuritis) and anæsthesia of the sensory part of the nerve, and between hyperkinesis (spasm) and akinesis (paralysis, paresis) of the motor portion.
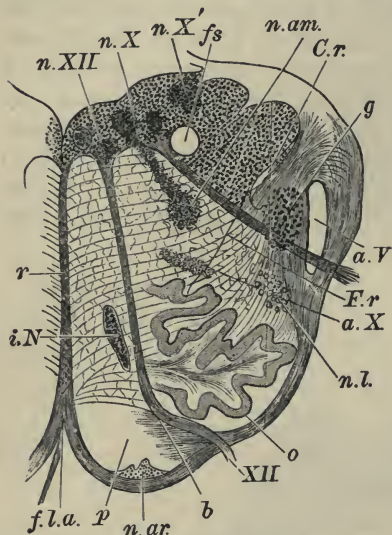
Fig. 12.—Cross-section through the Medulla Oblongata. (After Schwalbe.) *a.V*, ascending root of the fifth. *n.XII*, nucleus of the hypoglossus. *n.X* and *n.X'*, nucleus of the vagus. *XII*, hypoglossal nerve. *fs*, solitary funiculus (respiratory fasciculus). *p*, pyramidal tract. *o*, olive. *i.N*, pyramidal nucleus. *f.l.a.*, anterior longitudinal fissure. *n.am*, nucleus ambiguus.

I. The Central Affections of the Trigeminus.

In dealing with the cortical affections of the trigeminus we discriminate between irritative and destructive lesions of the cortical centre. In the former case we get spasm, in the latter paralysis of the muscles of mastication.

Spasm of the muscles of mastication (trismus, masticatory facial spasm—Romberg) occurs frequently as a part of general convulsions (Senator, Petrina, Seligmüller), and much more rarely independently, unaccompanied by other spasms (Lépine, Pfungen, v. Langer). There are two forms—a tonic, in which the teeth are pressed firmly together and the muscles of mastication, usually of both sides, are hard as wood to the touch; and a clonic, in which the lower jaw is moved to and fro horizontally or vertically, and spasmodic masticatory movements are induced. In a case in my practice, in an old gentleman who had suffered from repeated slight apoplectic attacks, the patient for several hours every day goes through well-marked chewing movements without eating anything, which at times are so vigorous that he often while smoking bites through his cigar unintentionally. The origin of the disease is often of a reflex nature. Toothache, periostitis of the inferior maxilla, face-ache, may give rise to it. Sometimes, it may be, a cortical affection lies at the bottom of it, but for

this there is at present no evidence furnished by post-mortem examinations.

Paralysis of the muscles of mastication is, on the whole, less frequently observed than spasm. Barlow, Oulmont, and Kirch-hoff report cases of it, recording in some only cortical lesions, but in others changes in deeper-lying portions of the brain as well. All the cases had this one anatomical feature in common, viz., that the cortical lesions always occurred bilaterally, thus in every case involving both centres. The first instance in which a unilateral lesion of the cortex was found was published by myself (cf. lit.). It confirms the supposition that the cortical motor area of the trigeminus includes the lower third of the anterior central convolution and the adjoining portion of the second and third frontal convolutions, and demonstrates that a unilateral lesion of the cortex (in this case it was left-sided) is sufficient to paralyze the muscles of mastication on both sides. The lesion was due to the presence of a psammoma the size of a filbert, which was situated upon the dura and cortex at the spot indicated, causing a depression and softening of the latter. The paresis of the muscles of mastication had reached a high degree, and was the more interesting from the fact that it was accompanied by periodical attacks of pain in the face and spasm in the area of distribution of the left facial nerve. Paresis and paralysis of the muscles of mastication are occasionally observed among the symptoms due to progressive bulbar paralysis and to pseudo-bulbar paralysis. The idea that these may develop as the result of a peripheral affection in an isolated disease of the motor portion of the third part of the trigeminus can not *a priori* be considered as impossible, but there have been up to the present no such cases observed. The differential diagnosis between a central and peripheral affection could be made only by means of an electrical examination. The lesion is central if there are neither quantitative nor qualitative changes in the reaction to the faradic and the galvanic currents. If such changes, however, exist—for instance, if there be the "reaction of degeneration"—the lesion is peripheral.

Only the latter form of the disease is amenable to treatment (by electricity), and then with but slight chance of success. Against the central variety we are absolutely powerless. With regard to the affection of the nuclei and roots of the fifth nerve in the pons, the anatomical relations of which are, as we have seen, not as yet sufficiently well understood, we know little

or nothing. Whether they ever occur independently, or, as is more likely, only as concomitants of diseases of other bulbar nerve centres, has not been determined. However, the supposition seems justified that the centres in question, in the course of certain general diseases of the nervous system—for instance, in multiple sclerosis and particularly in tabes—are affected relatively early. Thus Erben reports (Wiener med. Blätter, Nos. 43, 44, 1886) that he has observed very troublesome paræsthesias of the sense of taste in tabetics occurring in paroxysms, beginning in the pharynx. These were especially pronounced at the anterior edge of the tongue, and were accompanied by anæsthesia in the second branch of the fifth. This condition is presumably to be considered a disease of the nerve of taste, being analogous to the so-called gastric crises which are attributed to an affection of the vagus centre. A central anæsthesia of the trigeminus may also occur. In its symptoms it would not differ from the peripheral except that it may be bilateral. The central nature of the trouble one would infer from the simultaneous participation of other nerves, both sensory and motor (Romberg). The interference with conduction may take place at the base of the brain.

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II. Peripheral Affections of the Trigeminus.

In its peripheral part the nerve may become diseased inside as well as outside of the cranium. If the lesion is one of the stem, and extends to all three branches, it may be difficult, indeed impossible, to determine its exact site, because we do not as yet possess any means which enable us to definitely decide whether the nerve is diseased centrally from the Gasserian ganglion, whether the ganglion itself, or, finally, whether the

three individual branches are all affected at their exit from the skull.

An affection of the nerve stem to the central side of the Gasserian ganglion can only be assumed with any degree of certainty if the nerve is diseased in its whole sensory distribution and if extensive trophic disturbances are also present. The affections of the Gasserian ganglion itself (inflammation, neoplasm, softening) have as yet but little practical importance. If the disease does not take in the stem, but only the terminal portions of the individual branches, it is easy to determine the seat, and while we have in the intracranial diseases to consider those of the finer branches of the nerve in the dura, in the extracranial we have the branches distributed to the face and those to the nasal cavity.

A. Intracranial Diseases.

Headache—Cephalalgia—Migraine—Hemicrania.

Since it is very likely that there are only exceptional cases of idiopathic headache in which the fifth or its terminal branches in the dura (cf. page 3) are not implicated, it seems advisable to speak of headache here. At the same time we must expressly state that we are forced thus to take up different diseases together, which are ætiologically as well as pathologically to be strictly separated from one another.

The main point to decide in a given case will always be whether the headache is to be regarded as merely a symptom of another disease or as an affection by itself. Nobody ever would assume a headache which occurs at the onset of a severe illness—for instance, an acute infectious disease, or in association with organic brain disease (e. g., tumor), or during grave disorders of nutrition, anæmia, and chlorosis—to be an affection by itself and treat it as such. These headaches will always be considered as a mere symptom of the underlying disease ; but when we find an otherwise healthy person suffering from protracted or paroxysmal headache, while on repeated careful examination we are unable to discover any other disease, then we are forced to assume an independent affection and we have to endeavor to determine the following points : (*a*) The seat of the headache ; (*b*) its peculiarities and its course ; (*c*) its ætiology ; (*d*) its appropriate treatment.

Pathology.—The anatomical situation of the headache can

hardly ever be determined ; but we are justified, since we do not know what part the brain substance takes in it, in believing that the sensory terminal branches of the trigeminus in the dura (the dura receives at least two branches from the trigeminus) are always implicated, and are thus in some measure the seat of the headache. Under what conditions these nerve endings are thrown into a state of irritation—a state upon which the headache depends—is not well understood, and all we know about this question is more or less hypothetical. The most probable explanation is that the amount of blood in the brain or its membranes at the time being is an important factor in the production of the morbid condition, whether there be a permanent increase or decrease or frequent, perhaps very slight, changes in the amount. An increase constitutes what is called cerebral hyperæmia, a decrease cerebral anæmia ; and we assume the former condition if full-blooded individuals, who are liable to rushes of blood to the head, complain of paroxysmal headache ; the latter, if it occur in pale, anæmic patients who are subject to fainting spells. However, we do not know anything positive, and we shall have occasion to deal more in detail with this in another place. Of the greatest interest, and perhaps of the most common occurrence, are the fluctuations in the intracranial blood pressure, which possibly are the cause of the irritation of the terminal branches of the trigeminus in the dura and pia. If such fluctuations appear frequently, so as to give rise to an unequal distribution of the blood in the two halves of the brain, the irritability of the sensory endings may become abnormally increased, so that slight causes are sufficient for the production of the pathological condition. The clinical observations even go to show that without any demonstrable cause from time to time there may develop an increased irritability of these terminal branches of the fifth, associated with simultaneous fluctuations in the blood pressure. If the attack of headache thus produced is accompanied by vaso-motor symptoms, either of a paralytic or of an irritative nature, it is designated as migraine or hemicrania, the latter name being given to those not very common cases in which the pain is strictly confined to one side of the head. Owing to the vaso-motor disturbances just mentioned, some have been inclined to locate the seat of the disease in the sympathetic system, without being able, however, to show that the symptoms referable to the sympathetic are not perhaps only a secondary result of the pain, and therefore reflex

in nature (Möbius); and until this is actually demonstrated not to be the case we are justified in looking upon migraine as belonging to the affections of the trigeminus. In some, as it seems, quite exceptional cases, the seat of the headache is to be referred to certain muscles, which present at their origin and insertion as well as in their course points of tenderness (O. Rosenbach, cf. lit.). Among these, besides the frontal occipital and temporal muscles, are the sterno-cleido-mastoid and the upper part of the trapezius. This myalgia, which is occasionally produced by an unnatural position during sleep, and which is easily diagnosticated on careful examination, is said under certain circumstances to be the cause of headache.

Symptoms and Course.—With regard to the peculiarities and the course of the headache connected with affections of the trigeminus, we know that in its character as well as in its situation it presents no inconsiderable number of variations: thus, while one patient complains of a dull, boring ache, another describes his pain as sharp and burning; while in the one it is worst in the forehead, another refers it chiefly to the occiput, vertex, or temples, etc. In some instances the patients designate sharply circumscribed places of the hairy scalp as the seat of their pain. The headache also varies much in degree—from a dull sensation of pressure to a pain which allows of no sleep. In some cases the suffering is increased by a touch or a tap on the head, while in others it is soothed by a firm bandage around the temples. But seldom do we find a headache lasting for days, weeks, or even months without interruption; usually there are times when it is less severe or when it ceases completely. There is no regularity or uniformity in the occurrence or duration of the attacks. Two cases are scarcely ever alike, and almost always each presents certain peculiarities of its own: thus in the one slight febrile movements, absent in another, may occur; one patient enjoys a splendid appetite during the most violent pain, while another is unable to eat a thing, etc. The so-called attack of migraine has, at least in some cases, certain characteristics of which we shall now speak. It is usually for several hours preceded by prodromal symptoms, general lassitude, chilly feelings, tendency to yawning, buzzing in the ears, and the like. If the regular attack is going to begin in the morning the patient wakes up repeatedly during the night, and is thus able to predict the attack with certainty. The headache is sometimes, as we have remarked above, confined to one side,

and, according to statistics, the left seems to be the one pre-
ferred; but the seat often changes during the attack, so that
the patient complains now of the left, now of the right side of
the head. Sometimes a distinct pallor is noticeable on one side
during the attack, associated with dilatation of the pupil and
increase in the salivary secretion, while in other instances one
half of the face is flushed and hot, the arteries pulsating strong-
ly, and the pupil contracted. In the first case we designate
the hemicrania as spastic (sympathico-tonica, connected with
stimulation of the sympathetic); in the latter as paralytic (con-
nected with paralysis of the sympathetic). The former has
been described by Du Bois-Reymond, the latter by Möllen-
dorf, in both cases after observations made upon themselves.
But these conditions are not constant either, and if one has
seen many attacks of migraine he knows full well that the pa-
tients often change color—they are now pale, now flushed,
now complain of a feeling of heat in the head, now of cold.

If the pain is very violent the patient shows general consti-
tutional symptoms. In a bad attack he lies for hours com-
pletely apathetic, meeting every question and every source of
disturbance with unmistakable signs of disgust. He refuses
nourishment entirely, owing to a feeling of utter discomfort
and an almost uncontrollable desire to vomit. Only after copi-
ous vomiting of bile-like mucoid masses does his condition
gradually improve, the amelioration beginning with a violent
desire for food and a polyuria following the attack, which is
finally ended by a refreshing sleep. When vomiting does not
occur the patient suffers for a longer period of time. Some-
times the eyes participate, and photophobia, flitting scotomata,
even hemianopia, have been observed during the attack. These
are instances of what Féré calls *migraine ophthalmique*, a form
which he carefully discusses in his admirable monograph on
the subject. It is not uncommon for the attacks not to reach
their full development, but only for certain symptoms—flitting
scotomata, vomiting, vaso-motor disturbances, or the like—to
appear. Such isolated symptoms may be called " hemicranic
equivalents " (Möbius).

The duration of the attack varies from a few hours to a
whole day; it rarely lasts longer, and if it does, this fact should
always make us doubtful as to the diagnosis. In the intervals
the patients as a rule feel well; still, if the attacks are very se-
vere and frequent, occurring, for instance, as often as once or

twice a week, the after effects may be so lasting that the sufferers never can enjoy perfect health. Fortunately, such a rapid succession of the seizures is uncommon. Once a month or six or eight times a year is the rule, not counting slight, abortive attacks.

The course of migraine is always extremely tedious, sometimes lasting through a whole lifetime. In women the climacteric period occasionally, but by no means always, exerts a beneficial influence. At the time of menstruation the attacks seem to be especially apt to occur; but, even if no definite attack makes its appearance, women who are subject to migraine complain of more or less severe headache at such periods. Not infrequently the disease has an unfavorable influence on the disposition and appearance of the patients; they become peevish and ill-tempered, and even in the intervals between the attacks are by no means amiable or sociable. They are wont to restrict themselves considerably in their social intercourse —for one reason, because they rarely are able to make engagements for definite times, on account of the possibility of the occurrence of one of their attacks. The trophic disturbances which are sometimes superadded, as, for instance, the premature gray hairs, make such patients look older than they really are; on the other hand, there are individuals who, notwithstanding the severity of the attacks, retain for a long time their youthful freshness and vivacity. This may especially be hoped for if in the intervals the patients enjoy sound and healthful sleep. Unfortunately, in the majority of cases they are deprived of this, and are forced to resort to artificial means to procure it, of which the bromides are the most popular. It is not always easy to understand the cause of the sleeplessness (*agrypnia*) in migraine, and for that matter in all nervous diseases; it is especially difficult to do so when this is the only symptom and absolutely nothing else can be detected, when individuals otherwise healthy are wholly or almost wholly deprived of sleep for weeks; and yet it is just the discovery of this primary cause that is of the greatest importance, as it will guide our action in the treatment; and only when this is found can we reasonably hope for improvement from our efforts. Sometimes we have to deal with a gastric catarrh which until this time has been overlooked, a hyperæmia of the liver, and the like, and after the successful treatment of these by Carlsbad water, etc., sleep, which in spite of all bromides and morphine

had in vain been sought, returns of its own accord. Some-
times a high grade of anæmia may lie at the bottom, easily
recognizable by the pallor of the skin, the small pulse, and
the cold extremities. In such cases cod-liver oil, iron, and
quinine are more serviceable than the usual hypnotics, which
are rarely well borne. In all nervous patients suffering from
insomnia it is advisable to examine the thoracic and abdominal
as well as the sexual organs, and only to treat the sleeplessness
symptomatically when repeated examinations have given nega-
tive results. This symptomatic treatment consists above all in
the careful use of massage, which should be supervised by the
physician, a practice from which we have obtained very grati-
fying results. Next comes the systematic galvanization of the
brain, for the technical details of which the reader is referred
to Erb's book on electro-therapeutics (page 333, Leipsic, 1882).
As a last resort we have the administration of quieting, calm-
ing, and sleep-producing drugs, among which, notwithstanding
all the new hypnotics, morphine still holds the first place. Be-
sides this, chloral, paraldehyde, urethan, hypnone, coniine, lupu-
line, and amylene hydrate (tertiary amyl alcohol), which has
recently been recommended by von Mering, may be tried.
The latter is best given in doses of three and a half to four
grammes ($\mathfrak{m}$ l–lx) in one dose once in twenty-four hours, and
seems often to have a favorable action. On account of the bad
taste of this drug the addition of correctives—for instance, the
oil of peppermint, which somewhat masks the taste—is to be
recommended. (Amylene hydrate, 7.0 ($\mathfrak{m}$ cv); aq. menth. pip.,
40.0 (℥ x); ol. menth. pip., 1.0 ($\mathfrak{m}$ xv); syrup. simpl., 30.0 (℥ j).
Sig.: Half to be taken at night.) The sleep after it is deep and
quiet, and unpleasant after-effects are rare. Nevertheless, it is
well to be careful in its administration, as symptoms of intoxi-
cation may appear, as Dietz has reported (Deutsch. Medicinal-
Zeitung, 1888, 18). Migraine is frequently complicated by
stomach troubles, and more especially by cardialgia (gastral-
gia), and the peculiar form of nervous dyspepsia which Ross-
bach has called "nervous gastroxynsis" (γαστήρ ὀξύς). Of car-
dialgia we shall speak in discussing the neuroses of the vagus,
and it suffices here to mention that it is often associated with
slowing of the pulse, meteorism, flatulence, and vomiting. The
gastroxynsis, which depends upon an abnormal increase in the
production of free hydrochloric acid, begins with an extremely
disagreeable feeling of acidity in the stomach, with severe, at

times intolerable, headache, and ends with the vomiting of large strongly acid masses. That the headache in these cases does depend on the acidity of the stomach is evident from the following reasons (Rossbach): (1) It ceases when the acid masses are thrown off or if they are diluted by copious draughts of warm water; (2) in persons suffering from gastroxynsis it often appears after indulgence in acid food—e. g., salads. It may in general be put down as a fact that by far the greater proportion of sufferers from habitual headache or migraine have at the same time some trouble with the stomach; the frequent belching, the loss of appetite, the foul breath, speak in favor of this, and no one of these three symptoms is scarcely ever absent in patients of this class.

Ætiology.—Ætiologically headache and typical migraine have much in common; heredity, above all, the influence of which is so striking in migraine, plays an important part. It need not be of necessity the parents who have been affected; often the grandparents or their sisters or brothers or the uncles and aunts may have suffered from it; and we should therefore, in getting the family history of the patient, carefully inquire into the health of such relatives. Only in patients with hereditary tendency have we observed migraine in childhood—in girls of from six to seven years of age—as in other cases it develops as a rule during puberty. Young, rapidly growing individuals who have to do hard mental work (preparation for examinations, etc.) are frequently sufferers from severe migraine (*hemicrania adolescentium*). The influence of the stomach on the development of the disease has already been spoken of. As a matter of fact, habitual headache or typical migraine is often developed after repeated excessive indulgence in indigestible food; more frequently, it is true, the stomach and head symptoms are developed together. Quite recently Legal has called attention to the fact that, especially in cases where the headache is referred to the temples or to the occipital region, the underlying cause is frequently to be found in diseases of the middle ear and the pharynx. He advises in such cases, after a careful examination, the use of Politzer's inflations.

The ætiological importance which has to be attributed to errors in accommodation or refraction, more especially to hypermetropic astigmatism, has been pointed out by A. Bickerton (cf. lit.). There is no doubt but that in very exceptional cases migraine has to be looked upon as a reflex neurosis,

which is caused by chronic uterine or intestinal disturbances (worms, tænia), and we should take this possibility into consideration in our search for the cause. Exceptionally, migraine-like attacks have been observed in the course of gout, and it looks as if they were also in some way connected with the excretion of uric acid, since it has been found that before the attack no uric acid can be detected in the urine, while after it the amount is very markedly increased and, later on, for a time considerably diminished. The polyuria which occurs frequently after the attack, and lasts for several hours, where an acid urine, light yellow, almost as clear as water, of a very low specific gravity—1.005 to 1.007—is voided, has before been mentioned, and to the fact that migraine-like attacks may also occur in the initial stage of tabes, and may be of importance for the diagnosis and prognosis, we shall have to refer later.

Prognosis.—It is difficult, indeed at times impossible, to give a reliable prognosis in the cases now under consideration. So far as life is concerned it is always favorable if the case is one of a purely functional disturbance—that is, where the headache or migraine exists by itself as an independent affection, and where it is not to be regarded as a symptom of another disease. Life in such cases, as we said, is never in danger; the patient recovers from his severest attacks comparatively readily, and even after frequent repetitions of them it is exceptional that the digestive disturbances and the loss of strength which these entail give rise to a serious condition. But is the prognosis for recovery as good as for life? To this question we must answer without reservation, No. One can not deny that the outlook for a complete recovery is on the whole very bad, and that the chances, *cæteris paribus*, are so much the worse the longer the disease has lasted, and the more difficult it is to find any tangible cause. The worst cases are those where the trouble is inherited; in these recovery is very exceptional. At any rate the prognosis in all cases should be guarded, and little should be promised. There is hardly any other disease which is so liable to injure the physician's authority and the patient's faith in him and his medical skill as migraine and habitual headache. On the other hand, spontaneous recoveries are not unheard of, a fact which we ought to remember if all our drugs leave us in the lurch.

Treatment.—The treatment of habitual headache, and especially of migraine, is generally very tedious, and puts to a severe

test the perseverance, not only of the patient, but also of the physician. It is, therefore, an absolute necessity before undertaking to treat a patient of this kind to lay down, after a most careful and minute examination, a definite plan of treatment, which must be rigorously adhered to. It is not sufficient to use to-day this, to-morrow that drug, of which we have possibly read in the last journal as being effectual against migraine, and with which we may accidentally obtain a transient good result, but the treatment must rather be systematic, and be the outcome of certain well-considered conclusions, which we shall now briefly discuss. In the first place, we have to decide whether there exists another disease which causes the migraine. If, as is frequently the case, stomach symptoms are present, a stay at Carlsbad or Kissingen may do much good. If the acidity of the gastric juice is increased, the regular ingestion of alkaline drinks or lukewarm water is indicated. In all cases much attention is to be paid to the diet, and patients should especially be warned against overloading their stomachs at night. The regulation of the bowels is effected by the use of large enemata of water, or of small ones of pure glycerin (5 to 6 cc. ($\mathfrak{m}$ lxxx–c) at a time), or by vegetable aperients, such as rhubarb. Any degree of constipation may be attended with bad consequences. Diseases of the middle ear or the pharynx should be treated by a specialist. If the patient have a gouty diathesis, the use of lithium and the regulation of the diet should constitute the main treatment. The eyes should be examined for errors of accommodation and refraction which may exist, and these when found should be corrected by proper glasses. Cases which had resisted all other treatment have been cured in this manner (Bickerton, Brailey, Weir Mitchell, etc.).

If no other co-existing diseases can be detected, our chief efforts must be directed to building up the general constitution. From the cold-water treatment, general faradization (according to Beard and Rockwell), franklinization with the Holtz machine, systematic gymnastic exercise at home—from any one of these measures we may, under certain circumstances, obtain the desired result. In some cases lasting advantage has been seen from a change of climate, traveling, a stay in the mountains or at the sea-side.

With regard to the combating or the shortening of the attack we may say that, although there are quite a number of drugs more or less warmly recommended, we should not ex-

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pect too much from them.   Two or three times they may be
useful, but after repetition they lose their effect.   If the above-
mentioned vaso-motor changes point to the existence of a
pathological contraction or dilatation of the blood-vessels we
may, in the former case—in that of contraction—resort to the
careful administration of nitrite of amyl, three to five drops of
which are put on a handkerchief and given the patient to in-
hale, or to the internal use of nitroglycerin (three times a day
one drop of a one-per-cent alcoholic solution).   Great care has
to be exercised in the exhibition of the latter drug, and, if the
pulse indicate it, we ought to begin with minimum doses.   Such
a precaution is more especially necessary if the pulse is full
and the arterial wall tense, in which case a quarter or half a
drop is sufficient as an initial dose (Trussewitsch).   It is, more-
over, not advisable to continue its administration any longer
than one or two weeks, as it is liable to give rise to cerebral
symptoms (buzzing in the head, vertigo).   In the second
case—that of vaso-dilatation—ergot is indicated, which may
either be used in the form of hypodermic injections of ergotin
(ergotinum dialysatum, 1.0 (grs. xv); aquæ destill., 4.0 (ℨ j). Sig.:
Half a syringeful), or by the mouth (extr. secal. corn. [Denzel],
2.0 (♏ xxx); aquæ cinnamomi, 180.0 (℥ vj). Sig.: A tablespoon-
ful every two hours).   Lately Kraepelin has recommended the
hypodermic use of cystisin (0.003 to 0.004 (gr. $\frac{1}{20}$ to gr. $\frac{1}{16}$).—
Neurol. Centralblatt, 1, 1888).   If no such indications are fur-
nished by the condition of the blood-vessels we have to try
which medicine does the most good, and may begin with the
citrate of caffeine (0.15 (about 2 grs.) three times a day), which
we have in all cases found to be more effectual than the aethoxy-
caffeine recommended by Filehne.   If the latter is given in
doses of 0.25 (about 4 grs.), as Filehne advises, it will induce
violent vomiting, and the patients are worse than before.   If,
on the other hand, it is given in small doses, it is of no use—at
least in no single case have we seen lasting benefit derived from
it—whereas caffeine often does good service for months.   The
pasta guarana in 2.0 (grs. xxx) doses twice a day gives similar
results, but often interferes with digestion.   Salicylic acid is in
many cases, especially at the onset, followed by surprising re-
sults, but its continued use is disagreeable to the patient on
account of the bad after-effects.   I myself have never seen any
remarkable results from antipyrine, which has recently been
recommended, among others by Germain Sée (5.0 (grs. lxxv)

in two hours). The action which antifebrine has in migraine has not as yet been carefully studied, but this drug certainly should be tried. Application to the painful spot of an alcoholic solution of menthol (three to twenty) is often both agreeable and refreshing to the patient, the migraine pencils also prepared with menthol having a similar effect. If painful points can be discovered on the scalp or on the muscles (*vide supra*), a slight pressure and kneading of the same, later a more energetic massage to the head, is advisable.

Electricity may be used (1) in the form of the constant current passed longitudinally or transversely through the head or by applying it to the cervical sympathetic, and (2) in the form of the faradic current. In this case it is best for the physician to apply his own hand, previously moistened, to the forehead of the patient, this taking the place of one electrode. In the other hand he holds one of the electrodes, the other being placed on the back of the patient's head, the sternum, or some other indifferent point. With this mode of application, which is called the "faradic hand," only very mild currents should be used (cf. Erb, Handbuch der Electrotherapie, page 327, Leipzig, 1882).

Numerous as are the means at our disposal for combating the disease, quite as numerous are the patients who, after hundreds of unsuccessful trials, give up all medicines and all physicians. They retreat at the beginning of the attack from the world and from their families, darken their rooms, lie down quietly, and take simple domestic remedies, among which Russian tea with lemon juice has obtained a prominent place. Absolute rest is what always does most good to all these patients. Finally, we should not forget to deprecate, especially here, the use of all hypnotics, more particularly morphine, as they never do any good, and are often capable of producing serious harm.

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B. EXTRACRANIAL LESIONS.

The extracranial lesions are, on account of their great fre-
quency, of an eminently practical significance and of scientific
importance, inasmuch as valuable information about physiolog-
ical questions—that is, the course of the trophic and the gusta-
tory fibres—may be gained from them if the individual cases
are carefully observed and accurately recorded. We shall
have to treat of (1) the diseases of the facial branches of the
nerve, and again separate in our consideration the paralytic
from the irritative affections.

1. Trigeminal Neuralgia—Fothergill's Face-ache—Tic Douloureux
—Prosopalgia.

Variable in its degree of intensity, beginning with a moder-
ate, dull, boring, but always distressing and uncomfortable feel-
ing of pain, and sometimes reaching a pitch of severity experi-
enced in no other kind of neuralgia, tic douloureux forms one

of the most common affections of the fifth nerve. It appears, as a rule, unilaterally, and by preference fastens upon the first and second branches. The sensory division of the third branch seems, at least by itself, rarely to be the seat of the pain, whereas it is not uncommon for all three divisions to be simultaneously attacked.

Almost every patient gives a different description of his pain. One declares that it feels as if a red-hot wire was being driven into the bone, another as if the face was buried in a heap of stinging nettles, a third fancies that the nerves are being pierced with a sharp instrument, etc. Almost every case presents its own peculiarities as regards the frequency and duration of the paroxysms, between which there are often intervals free from pain, but the course of the disease follows no hard and fast rules. The tender points of Valleix (*points douloureux*) can almost invariably be demonstrated. Almost always there is one on the supra- and another on the infra-orbital foramen, a third over the exit of the subcutaneus malæ, a fourth over the mental foramen, and a fifth in front of the ear, where the auriculo-temporal passes over the zygomatic arch. The so-called palpebral point on the upper eyelid, the parietal point on the parietal eminence, the labial point on the upper lip, and many others are not always present. Firm pressure on these points is always, even in the intervals between the paroxysms, disagreeable to the patient and even liable to produce an attack.

The neuralgia of the first division of the fifth nerve is mostly an affection of the supra-orbital nerve, with pain in the forehead, the nose, the upper eyelid, and the eyeball (ciliary nerves). The other terminal branches are hardly ever affected. The neuralgia of the second division attacks the cheek, the lower eyelid, the nose, and the upper lip, often also the upper row of the teeth (n. alveol. sup.) and the palate (spheno-palatine branch). The branch most commonly affected, sometimes also by itself, is the infra-orbital. The neuralgia of the third division embraces the lower jaw, the chin, the cheek, sometimes also the auricle and the external meatus. The lower row of teeth, the tongue, and the mucous membrane of the mouth are often the seat of intense pain. As we have said, an isolated affection of this third division is comparatively rare. The inferior alveolar branch, however, forms an exception to this, as it is not rarely attacked separately, and the consequent tooth-

ache has often induced patients to have one tooth after another extracted—of course, however, without any improvement.

That the vaso-motor and the trophic fibres of the nerves are also at times implicated is evident from certain symptoms, viz., intense flushing, hyperidrosis, strong pulsation of the temporal artery on the diseased side, together with increased secretion of tears and saliva, affections of the hair, which has a tendency to turn gray and fall out, especially over the most painful places. Such symptoms are not uncommon. Herpetic eruptions, especially zoster ophthalmicus and frontalis of the affected side, have repeatedly been described.

Pathology.—Of the pathology little can be said with certainty. The thickening and swelling of the neurilemma, the degeneration of the Gasserian ganglion and of the nerve trunks, the small inorganic concretions which have occasionally been seen on the neurilemma—all these are changes which have sometimes been observed, but which quite as often were absent. At all events no particular importance can be attributed to them. Whether cortical lesions and affections of the nuclei and the roots are capable of bringing on the disease we do not definitely know, yet such possibilities can not be excluded (cf. the case published by myself in the Berl. klin. Wochenschr., 1887, 27).

Course.—The course of the disease is on the whole extremely tedious, and attacks which harass the patient to the end of his days are to be observed here as in migraine, the only difference being that in the disease under consideration the sufferings of the patient are still more unbearable. The disease throws a shadow over his whole existence far more gloomy than in migraine, and so we can well understand why again and again he tries all sorts of remedies and frequently even the most heroic measures to relieve his pain at a time when a migraine patient would have given up all medicine and all doctors.

Treatment.—Unfortunately, here also therapeutics is often powerless, as has already been indicated by the remark that the disease is often of life-long duration. Hope of recovery is only justifiable in cases where we have an underlying disease, as, for instance, malaria, in which case the neuralgia is to be regarded as a symptom, or where local causes exist—for instance, bone diseases, the presence of foreign bodies, or neoplasms which can be removed, etc. Such cases will repay the efforts of the

physician, and a cure can be effected by proper internal medication or by surgical interference. In cases, however, where a primary cause, which would furnish us with data for our treatment, can not be discovered—where we, therefore, are forced to experiment with the nervines and the so-called specifics—let us beware of raising our expectations too high, for too often all our efforts will be in vain. Arsenic, zinc, quinine, the bromide and iodide of potassium, asafœtida, castoreum, valerian, and many other medicines now completely obsolete have been tried, and still to-day sometimes are tried at random. The one uses this, the other that drug; under favorable circumstances each one does good once, but rarely is the improvement lasting. The most confidence may be placed in iron, if chlorosis be present (best administered in the form of Blaud's pills), quinine, arsenic, and iodide of potassium; if these leave us in the lurch we can resort to salicylate of sodium, 4.0–6.0 (3 j–3 jss.) a day, in capsules, or to salol or gelsemium, giving the latter in the form of the tincture, and pushing it perhaps until slight symptoms of intoxication appear (twenty drops every two hours). I have used corrosive sublimate, 0.05 (five-sixths of a grain a day) in pill form, several times successfully. Of butyl-chloral I am unable to say anything favorable (butyl chloral hydr., 7.5 (3 jss.–3 ij); glycerin., 20.0 (3 ss.); aquæ, 130.0 (3 iv). Sig.: A tablespoonful every ten minutes). In all my cases it proved very unsuccessful; the same holds good for bichlorate of methylene, which is applied externally in the form of a spray. Other anæsthetics, chloroform above all, do at least as much good, and the narcotics are decidedly better, as Trousseau has already upheld, who declared large, or we should rather say huge, doses of opium or morphine to be the only effectual treatment. Whatever we may think about morphine, in cases of tic douloureux, especially in severe instances, we can not dispense with it. The combination of morphine with atropine, or the alternate use of the two separately, has been recommended (Althaus); chloral hydrate alone, 4.0–6.0 (3 j–3 jss.) a day, is uncertain in its action, but in combination with morphia often acts very well; antipyrine, which has lately been recommended for all sorts of affections, may also be given a trial. The so-called revulsives (daily repeated cold-water enemata (Gussenbauer), cold or warm poultices, sinapisms, superficial cauterization, the electric brush) often act splendidly where we want to produce temporary amelioration of the pain; but, as we say,

unfortunately this is only transient. The same is true of electricity, which may be used according to the polar method (steady application of the anode over the painful place, cathode at some indifferent place, weak increasing and decreasing currents being used), or according to the method of direction of the current (descending steady current). Galvanism to the neck has also been advised. The constant current may be given a trial, combined with the action of chloroform, as Adamkiewicz has proposed in his paper on cataphoresis. I have several times used the "diffusion electrode," which he recommends, quite successfully (cf. lit.). The faradic brush, the unpleasant action of which may be somewhat mitigated by putting moist blotting-paper on the skin, is often very satisfactory, and I can recommend the strong cutaneous faradization very highly even during the paroxysms. Operative interference (neurectomy) has recently more and more, and justly so, fallen into disrepute. The results are often entirely negative, and where some success has been obtained with it this did not prove lasting. Baths, especially at the non-medicated hot springs, a stay at the seaside or in the mountains, cold-water treatment, and vapor baths may be advised, but we are unable to state definitely which of these modes of treatment are indicated in any particular case.

Ætiology.—About the ætiology we know little worthy of mention. That heredity and exposure to cold have something to do with the disease we must admit; but this is not peculiar to neuralgia of the fifth. However, anatomical changes (exostoses, narrowing of bony canals due to syphilis, etc.) are here frequently of moment. Age, sex, occupation, do not seem to exert any particular influence; still, the disease is on the whole very rare in small children, and if present it is always inherited.

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2. *Anæsthesia of the Trigeminus—Paralysis of the Trigeminus.*

Paralysis of the sensory branches of the trigeminus is on the whole less frequently met with than neuralgia of the face. Only exceptionally are all the divisions (the motor portion of the third included) affected equally ; but most observations go to show that, as a rule, only individual branches suffer, and these not in their whole extent, but only within certain areas. The smaller the number of fibres in the distribution of which anæsthesia obtains, the more peripheral is the seat of the cause (Romberg), and we may assume an affection of one whole branch to exist " where the loss of sensation is found not only in certain areas of the surface, but also in the corresponding cavities of the face " (Romberg). Whether the branch is affected before or after leaving the skull we have no means of deciding.

A lesion of the first division also causes anæsthesia of the surface of the eyeball. Since, in consequence of this, influences from outside (foreign bodies, dust, traumatism) are not perceived, not infrequently a keratitis, which begins in the lower segment of the cornea, is developed. This may run into an inflammation of the whole ball and bring about destruction of the eye (*ophthalmia paralytica*). That to explain this condition we must not assume a lesion of special trophic nerves has been shown by experiments on animals (Senftleben). An affection of the second division deprives the nasal branches of their function, and the nose becomes not only insensible to external touch, but certain pungent smells—as, for instance, that of snuff—are no longer recognized on the affected side. In lesions of the third division the corresponding half of the tongue, but only in its anterior two thirds, loses its sensation, and the patient has no longer any sense of taste in this area (n. lingualis) ; since, however, loss of taste in the anterior portion of the tongue has been observed in some cases where the function of the third division of the fifth was found to be perfect (Heusner), we can not exclude the supposition that the fibres of the chorda tympani (or at least a considerable part of them) join the facial from the second division of the fifth. Certain it is that the fibres which pass to the chorda return again to (the

second and third branch of) the trigeminus after having prob-
ably run with the facial as far as the geniculate ganglion.
From Fig. 13 the distribution of anæsthesia over the skin of
the face may be learned. Vaso-motor changes, subjective sen-
sations of heat and cold, sensations of being swollen, and dis-
turbances in the movements of mastication, difficulty in open-
ing the mouth (paresis of the external pterygoid and the ante-
rior belly of the digastric), are sometimes met with (Müller).
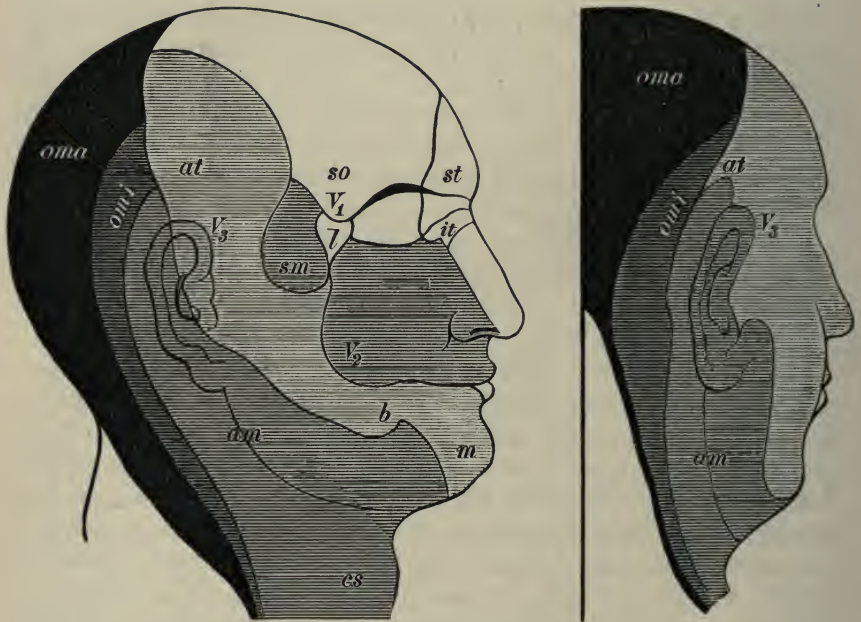
Fig. 13.—DISTRIBUTION OF THE SENSORY CUTANEOUS NERVES ON THE HEAD. V_1, V_2,
V_3, the three branches of the trigeminus. *at*, auriculo-temporal. *so*, supraorbital. *st*,
supratrochlear. *it*, infratrochlear. *l*, lachrymal. *m*, mental. *b*, buccinator. *am*,
auricularis magnus. *sm*, subcutaneus malæ. *oma* and *omi*, occipitalis major and mi-
nor. *cs*, superficial cervical.

The course of the disease depends upon the seat of the
lesion. In peripheral affections the prospect for recovery is
usually comparatively favorable; yet this is frequently only
partial, and several of the qualities of sensation remain per-
manently lost, the sensibility in general is dulled, and tactile
paræsthesias persist—in a word, recovery is imperfect.

The treatment chiefly consists of excitants, among which
the most efficient is the application of the faradic and the
galvanic brush to the skin. Transient improvement may be

thus obtained after a short while in the peripheral affections. This electric brush is the best excitant for the skin, and is to be preferred to all liniments and the like, which are supposed to act in much the same way. Internal treatment, provided there be no definite underlying disease, is absolutely superfluous.

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3. Trigeminal Cough.

Finally, we may call attention to a reflex neurosis, which was first described by Schadewald, and then studied by Wille. This is a paroxysmal cough which, occurring in individuals whose respiratory organs are perfectly sound, is entirely due to an irritation of the trigeminal fibres distributed to the nose, pharynx, and the external auditory meatus. The just-mentioned writers distinguish accordingly a nasal, a pharyngeal, and an auricular trigeminal cough, and declare the first (nasal) to be the most frequent variety. According to them also, this neurosis is by no means rare, and the possibility of its existence ought always to be thought of where we have to treat cases of an obstinate paroxysmal cough, which is liable to be produced by the action of pungent odors and by changes of temperature, and which is accompanied by hypersecretion of the nasal mucous membrane. The treatment consists in the use of the nasal douche, the application of weak induction currents directly to the nasal cavity, and the administration of potassium iodide. Further observations are still needed to decide whether we actually have to deal in these cases with a neurosis of the trigeminus, or whether the vagus has not something to do with the affection, or whether finally, as Hack suggested, the erectile tissue of the nose is responsible for it.

Quite lately it has been claimed that peripheral irritation of

the trigeminus (by inhalation of pungent vapors, new growths, etc.) may reflexly give rise to sensations of dizziness ("nasal vertigo," Joal). Until more confirmatory evidence is brought forward, it would be well to suspend judgment on this question.

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CHAPTER V.

THE facial nerve emerges at the base of the brain from the medulla oblongata by the side of the abducens and behind the trigeminus on the posterior margin of the middle peduncle of the cerebellum. The auditory nerve is situated close behind it, and between the two a separate bundle of fibres is placed—namely, a second root of the facial, the so-called nervus intermedius or portio intermedia Wrisbergii. With the auditory nerve the facial then passes forward and outward into the internal auditory meatus, at the bottom of which it enters through a small opening the Fallopian canal (cf. Fig. 16). In the hiatus of this canal it makes an almost rectangular turn (genu nervi facialis), passes backward and then downward, and leaves the skull through the stylo-mastoid foramen to divide inside of the parotid gland into the terminal branches, the temporo-facial and the cervico-facial, which form together the plexus anserinus major. At the so-called genu the nerve forms a gangliform swelling—the ganglion geniculi—from which the larger superficial petrosal nerve is given off (cf. diagram, Fig. 16). These are the fibres which communicate with the trigeminus, and have the function of gustatory fibres for the anterior two thirds of the tongue (cf. page 73).

The nucleus of the facial, a group of large multipolar ganglionic cells, lies four millimetres and a half beneath the floor of the fourth ventricle, in the region of the formatio reticularis, and above the upper olive (cf. Fig. 14). From this illustration it is also apparent that the ascending root of the trigeminus has the emerging portion of the facial root to its mesial side, while the anterior root of the auditory lies external to it. The axis cylinder processes of the ganglionic cells of the nucleus are united in a larger fasciculus, forming the first part of the root (Ursprungsschenkel of Krause), which at the floor of the fourth ventricle becomes a compact bundle, the intermediate portion (VII, a). At the anterior end of the eminentia teres this is bent at right angles (genu cerebrale), and becomes the emerging portion (Austrittsschenkel) of the facial (VII), which

reaches its point of exit, before mentioned, through the transverse fibres of the pons.

Quite lately experiments on animals by Mendel have shown that in rabbits and guinea-pigs the facial branches to the eyes take their origin in the oculo-motor nucleus. Whether this is the case or not in man our present pathological observations do not allow us to decide with certainty.
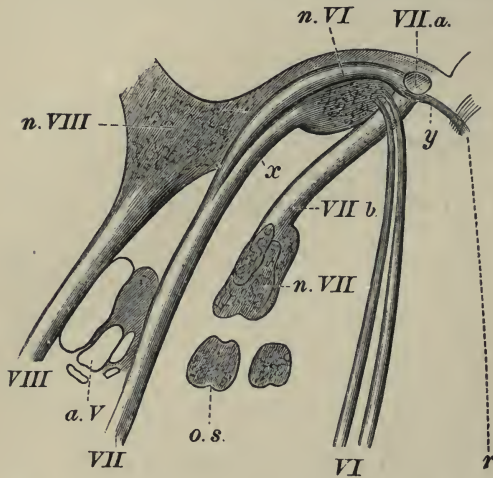
Fig. 14.—DIAGRAM SHOWING THE COURSE OF THE FACIAL FIBRES IN THE PONS. (After SCHWALBE.) *n. VII*, facial nucleus. *VII b*, root-bundle of the facial nucleus. *VII a*, intermediate portion (cross-section). *VII*, emerging portion of the facial. *n. VI*, abducens nucleus. *n. VIII*, nucleus, and *VIII*, root of the auditory nerve. *y*, fibres coming from the raphe. *x*, fibres coming from the abducens nucleus. *o. s.*, upper olive. *a. V*, ascending root of the trigeminus.

Just as in the case of the trigeminus, so in the facial, we must distinguish between central (cortical and bulbar) and peripheral (intra- and extra-cranial) lesions, which, owing to the purely motor functions of the facial, may give us, clinically, spasm or paralysis. We shall discuss each class separately.

1. FACIAL SPASM—" MIMIC FACIAL SPASM "—" TIC CONVULSIF."

Lesions which give rise to facial spasm may be central or peripheral in their situation. In the first case either the cortex or the nucleus (or the root) of the nerve in the medulla oblongata is concerned. According to our present ideas the cortical area for the facial is located in the lower half of the anterior and the lower third of the posterior central convolutions, and it is also supposed that the posterior halves of the two lower frontal and the anterior part of the supramarginal convolutions have some, although a less important, connection with it (Exner).

It is not known whether stimulation of these centres can produce a facial spasm, or, in other words, whether there exists a real cortical facial spasm. At least, we are unable to find

anything in the literature to warrant such an assumption. The possibility that those cases of tic associated with involuntary explosions of obscene words, which have been reported by Gilles de la Tourette, Dana, and Wilkin, may be due to stimulation of the facial and speech centre in the cortex, is, perhaps, not to be excluded. It seems, however, well established that the disease can be produced by reflex stimulation of the facial nucleus (cf. the case of Berger and its treatment). Undoubtedly, disease of the peripheral portions of the nerve is the most common, in which, just as in trigeminal affections, either the whole facial area or only individual branches may be affected. We distinguish a clonic and a tonic variety of spasm.

A patient suffering from clonic diffuse facial spasm has lost control over his facial muscles, either on one or, more rarely, on both sides. The muscles affected are in irregular motion, so that against his will the patient makes the oddest faces, wrinkles his forehead, raises the alæ nasi, screws his eyes up, etc. When the attack has passed he has a temporary respite, yet often enough the pause is very brief, and even during remissions spasms flash across his face, so that his features are never for any time entirely at rest. On the slightest provocation, by speaking, often also by eating, quite violent paroxysms are excited, so that the patient would fain cover up his distorted face.

If the spasm is tonic, the affected side of the face is singularly rigid and takes no part in the facial movements, but is distorted. The muscles are distinctly hard to the touch, the corner of the mouth is pulled toward the diseased side, the mouth firmly closed, the eyebrow drawn up—signs sufficiently marked to distinguish it from facial paralysis, in which also the affected side does not take part in the movements of expression. Vaso-motor and trophic changes are, as a rule, absent.

In cases where the spasm is confined to some branches of the facial only, we find that the muscles around the eyes are almost always the ones affected. The eyelid is attacked by a clonic or tonic spasm, and conditions are developed which go under the names of spasmus nictitans and blepharospasm.

The spasmus nictitans consists of spasmodic blinking, in which not only the eyes are rapidly closed and opened, but also the neighboring muscles (frontalis, zygomatici) participate in the spasmodic movements. In a mild form this spasm is seen in many people where it is only to be regarded as a bad habit.

Blepharospasm consists of a paroxysmal spastic contraction of the orbicularis palpebrarum, lasting a few seconds or minutes, which completely closes the lids. In rare cases the attacks follow each other so quickly and are so prolonged that the patient has to be treated as a blind man. The attacks appear unexpectedly and quick as lightning. They are often precipitated by voluntary firm closure of the eyes, eye-strain, or by the action of light, and the patient is utterly unable to raise the lid until the attack has passed. The physician, however, will succeed at times in cutting short the paroxysm if he be able to discover any of the so-called pressure points, which, according to von Graefe, who first discovered them, are often present. More or less firm pressure exerted at these points is capable of producing an interruption of the spasm and a cessation of the attack. Unfortunately, however, such points are often entirely absent, and when they exist their position is so uncertain and changeable that they may only be accidentally discovered. One of the few which is present with some constancy corresponds to the supra-orbital foramen. We should, however, look for them over the whole distribution of the trigeminus, over the spinous and transverse processes of the cervical vertebræ, and even in the region of the brachial plexus. It is our duty to make a frequent and untiring search for them, as we may thus be able to afford our patients very great relief.

Course.—The course of the disease, be it in the form of a total or a partial spasm, is usually very tedious, and a prognosis for recovery must be very guarded. The outlook is especially unfavorable when the affection is complicated with other motor disturbances, as I have observed, for instance, in two cases where the facial spasm was associated with writer's cramp. Such complications, which are, according to my experience, not common, render, as we said, the prognosis far more gloomy, and in such cases we must beware of too easily awakening hopes in the minds of our patients.

Ætiology.—We know little about the ætiology of blepharospasm. That it may be of reflex origin can not be doubted. The most varied diseases of the eyes, affections of the trigeminus, especially tic douloureux, carious teeth, intestinal worms, uterine troubles, may lie at the bottom of it, and the origin of the disease is cleared up only if, after removal of some primary cause, the spasm suddenly ceases.

I saw a case of blepharospasm, which had persisted for

years and was considered hopeless, cured after a coexisting flexion of the uterus had been materially improved. Diseases of the blood-vessels can, furthermore, produce the spasm, as we see from the case of Buss (cf. lit.), where an atheromatous artery, and from that of F. Schultze, where an aneurism of the left vertebral artery by pressure upon the facial nerve brought on the spasm. Finally, hysterical conditions can lead to it, as is shown by the latest communications of Charcot on the so-called hemispasmus glosso-labialis, which has been described by Marie in the Progrès méd. (June 6, 1887).

Treatment.—All these we must keep in mind in deciding upon a line of treatment, and not imagine that we can cure a facial spasm, whether it be total or partial, clonic or tonic, with indiscriminate galvanization, for without system we shall only meet with success in rare cases, and then only by good luck. The most promising plan of treatment is the application of the anode to pressure points if such be present, while the cathode is placed on some indifferent region, the back of the neck or the sternum. Weak currents applied for one or two minutes, with careful avoidance of make and break, give the best results. The application of the anode to the back of the head, keeping it at the same point, also sometimes meets with success (Berger), but too often leaves us in the lurch ; and this will hardly surprise us if we remember that even when the anode is placed on the back of the neck the abnormally stimulated reflex centre in the medulla is by no means always reached by those curves of the current which really do penetrate deeply.

Cures, such as that reported by Berger, undoubtedly depend upon a happy coincidence of circumstances. The medulla oblongata, above all, where in such an astonishingly small space a number of the most important nuclei lie close together, seems to be the most unfavorable place for local electrization (Duchenne), by which we aim at affecting individual nerves or nerve roots. We may reach all or none, no matter whether we use small or large electrodes. Still, even this method ought to be tried, since we have no positive remedy. Should the galvanic treatment fail, the internal treatment is still more vague, and it is well to inform the patient of the uncertainty of this procedure. Of course, the usual nervines and antispasmodics are to be given. As a last resort, neurectomy of the supraorbital or stretching of the facial nerve (Bernhardt, cf. lit.) has to be considered, yet even from this we can expect no lasting success.

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2. Facial Paralysis—Mimic Facial Paralysis—Hemi-plegia Facialis—Prosopoplegia.

Facial paralysis is an affection the relative frequency of which makes it of the greatest practical importance.   In this more than in any other nervous disease any layman can easily judge just how much the art of the physician has accomplished in a certain time in a given case, and on this account it especially behooves us at our first examination to be very careful in making a positive statement as to the prospects of recovery or the probable duration of the disease.   Both these points depend chiefly on the seat of the lesion, which, as in tic convulsif, may be central or peripheral.

A. CENTRAL FACIAL PARALYSIS.

Symptoms and Diagnosis.—Central facial paralysis may be produced either by a cortical lesion (cortical paralysis) or by a lesion of the facial fibers in the brain between the cortex and the pons (intracerebral paralysis *par excellence*); or, finally, it may depend upon a disease of the nuclei and nerve roots in the pons.   Cortical facial palsies may be caused by tumors, abscesses, or chronic inflammations in the region of the motor centres.   Those of intracerebral origin may be produced by syphilitic arterial disease or by rupture of a vessel in the region of the internal capsule and the crus cerebri.   The third form, that originating in the pons, is found in Duchenne's paralysis and, more rarely, in tabes.   Whether the facial paralysis, which appears and disappears in the course of hysteria is to be regarded as of central origin or not, can not be decided.

The clinical picture differs but little in these three forms, and only at times do the accompanying symptoms make a differential diagnosis possible.   Thus, for instance, the intracerebral paralysis often appears with an apoplectic attack, and is accompanied by hemiplegia and speech disturbances, while if

facial paralysis is found in connection with spinal disease it is always of nuclear origin.

All three forms of central paralysis have usually, however, two features in common which can almost be regarded as pathognomonic and which distinguish them from the peripheral paralysis, namely: 1. The presence of a normal electrical excitability in the nerves and muscles to both currents. 2. The escape of the upper facial branch. While in peripheral paralysis all three divisions are equally affected, we find in the central form the upper branch usually intact, and the patient can wrinkle his forehead and close both eyes.

We say usually, not always, because there are undoubtedly exceptions, where we meet with a central paralysis in which the upper branch has not been spared. It is quite probable that the naso-labial and the orbiculo-frontal fibres of the facial have a separate cortical origin, and we can well imagine that if the cause of the paralysis—e. g., a small focus in the cerebrum—is situated above the union of those two branches, one remains intact (in the large majority of cases the upper), whereas if it is below their point of union both branches are affected.

A further guide to localization is the condition of the movements of expression (Bechterew). If these are lost while the voluntary innervation of the facial muscles is intact, we have to assume a focus in the optic thalamus, the centre for facial expressions, or close to it (Bechterew), while a facial paralysis with retained power of facial expression allows us to exclude a lesion in the thalamus and its coronal connection with the hemispheres. In the case of Rosenbach (Neurol. Centralblatt, 11, 1886) there was an isolated paralysis of mimic expression in the left facial and right-sided bilateral hemianopia, and the lesion was taken to be in the right thalamus.

In differentiating between a cortical and a bulbar facial paralysis the following points must be taken into consideration: That the lesion is cortical is probable if the facial alone without the corresponding half of the body is paralyzed (monoplegia facialis), and if the affection is confined to the lower branches of the nerve, while the normal reaction to the electrical current remains undisturbed. It is easy to understand that the hypoglossus often takes part in the lesion if we remember in how close proximity the centres of the two nerves are situated in the cortex, and in a given case an examination of the mobility of the tongue will show whether we actually have to

deal with a so-called monoplegia facio-lingualis. Sometimes a disturbance of speech points at once to this combination. In every case in which we assume a cortical lesion, the sensation in the distribution of the facial and the hypoglossus ought to be tested, because it is just in these cases that we find not infrequently sensory changes—e. g., analgesias and anæsthesias. Of these "cortical" sensory disturbances we shall speak further in the next part, where we shall discuss cortical lesions of the brain.
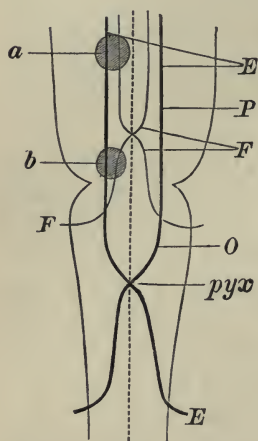
We shall be led in a facial paralysis to think of an affection of the pons when not only the nerve, but with it one whole half of the body is paralyzed; and there are two types of pontine facial paralysis according as the lesion is situated in the upper or lower part of the pons. In the first case (focus *a* in Fig. 15) the facial and the same, in the second (focus *b*) the facial and the opposite half of the body are affected (hemiplegia alternans, Gubler, 1859), because the facial fibres cross in the pons and we may have a lesion above or below this crossing, and in both cases this will be situated, of course, above the crossing of the fibres going to the extremities (cf. also what is said further on about lesions of the pons).

Fig. 15.—Diagram showing the Decussation of the Fibres going to the Extremities, and of those going to the Face, in the Pons and Medulla Oblongata. *F*, facial fibres. *E*, fibres going to the extremities. *P*, pons. *O*, medulla oblongata. *pyx*, decussation of the pyramidal tracts. *a*, a focus in the upper, *b*, a focus in the lower part of the pons (the latter is situated below the decussation of the facial fibres).

The facial paralysis caused by the lesion in the upper part of the pons, and that found in connection with hemiplegia after a lesion in the internal capsule, are in so far alike as they are both accompanied by paralysis of the extremities on the same side. But there is one point of difference which will influence our diagnosis, namely, that after pontine lesions the facial paralysis, very much as in the peripheral form, takes in all three branches of the nerve, while in a lesion of the capsule or the basal ganglia only the lower branches of the nerve are affected; but in contradistinction to what happens in the peripheral paralysis the electrical condition may, at least in some cases, remain normal.

The most striking symptom of central facial paralysis is the relaxed and expressionless appearance of the affected side. The naso-labial fold is more or less distinctly flattened, the corner of the mouth is slightly open and hangs down, the mouth seems to be drawn to the well side, the patient is unable to raise his upper lip or to whistle. On inflating the cheeks the air escapes; drinking and speaking are difficult, the latter especially, because the labial sounds are defectively formed. During eating the food gets in between the cheek and the teeth on the affected side and the patient has to bring it to the right place again with the fingers. In biting, the mucous membrane of the cheek is often caught between the teeth. The upper part of the face is in by far the greater number of cases normal; the forehead can be wrinkled well in its whole extent, and the patient can frown and close either eye perfectly.

The condition of the velum palati and the uvula varies, and is, therefore, of no value, either diagnostically or prognostically. The uvula may deviate to the sound or to the affected side, or may occupy its normal position. With our still imperfect knowledge of the innervation of the muscles concerned, any attempt to explain the different positions of the uvula must needs be hard, but we shall be less surprised at our difficulty when we consider that the levator palati is supplied not only by the facial through the large superficial petrosal, but very probably also by the vagus accessory, the tensor palati, however, by the third branch of the fifth. In other words, at least three cranial nerves are concerned in the motion and fixation of the uvula, and besides, even under normal conditions, the uvula is occasionally found to deviate to one or the other side. The only thing of which we can, perhaps, be sure is that if during phonation paresis of the velum palati and deviation to the sound side becomes apparent, the large superficial petrosal is most likely affected (paralysis of the levator palati and azygos uvulæ). Of greater importance for the diagnosis of central facial paralysis is the persistence of the reflexes, which in peripheral paralysis are often diminished or sometimes completely lost.

Furthermore, the disturbances in hearing, the alterations in taste and in the salivary secretion, so frequently observed in the latter, are almost always absent in central affections.

The existence of a bilateral facial paralysis—diplegia facialis —points as a rule to a central lesion, and more especially to a bulbar affection. It certainly is one of the greatest rarities to

have a simultaneous paralysis of both as the result of a peripheral lesion.

Prognosis.—The prognosis depends upon the anatomical basis of the disease. Lesions of the cortex and the pons often bring about facial paralyses that are incurable, while those observed in conjunction with capsular hemiplegias, especially in the early stages of the latter, frequently present a decided improvement after a time.

Treatment.—The question of treatment arises only when the primary lesion is amenable to therapeutic measures. Since this, however, is only very rarely the case, it is best, at least in the central facial paralysis, to restrict ourselves to the expectant treatment. The measures that will be recommended as indicated in the peripheral form are here of very little avail.

B. PERIPHERAL FACIAL PARALYSIS.

In its peripheral course the facial may be divided into two portions—an intracranial and an extracranial. The former is less frequently affected than the latter, which is more exposed to atmospheric influences, especially cold. For practical reasons we prefer to consider the diseases of these two segments separately.

1. The Intracranial Lesion.

This form is distinguished by the fact that besides the constant existence of paralysis of all the facial branches, we have often certain concomitant symptoms, which can only be fully understood if we picture to ourselves the exact course of the nerve. This can be done with the help of the diagram (taken from Erb) here represented, which permits an accurate localization of any given intracranial lesion.

(*a*) If the lesion be between the exit of the facial stem (from the pons) and the geniculate ganglion, we shall find a paralysis of the velum palati, abnormal acuteness of hearing, and diminished salivary secretion.

(*b*) If the facial be affected in the region of the geniculate ganglion itself, then we find in addition to the just-mentioned symptoms alterations in the sense of taste.

(*c*) A lesion between the geniculate ganglion and the stapedius nerve produces the symptoms described in *a* and *b*, but no abnormality of the velum palati.

(*d*) A lesion between the origin of the nerve to the stape-

dius muscle and the giving off of the chorda tympani gives alterations in the sense of taste and diminished salivary secretion, but no abnormality of hearing or of the velum palati.

(*e*) If, finally, the nerve is diseased below the giving off of the chorda, in the Fallopian canal, we only find paralysis in the distribution of the posterior auricular branch without any trouble with taste, hearing, the condition of the velum palati, or the secretion of saliva. We should state again, however, that in all cases from *a* to *e* all the facial branches take part in the paralysis.

Valuable as this diagram is, undoubtedly, regarded from a theoretical stand-point, yet in practice we but rarely meet with opportunities for observing cases which exactly correspond to it ; nevertheless, in every instance we should not fail to attempt to locate the lesion with as much accuracy as possible.

A physiological explanation for the appearance of the above-mentioned concomitant symptoms is not always easy. That alterations in the sense of taste are due to lesions of the chorda tympani can not be doubted, and if they are present the lesion is situated between the geniculate ganglion and the giving off of the chorda; if they are absent the lesion must be sought below this region. The disturbance in the sense of taste is limited to the anterior two thirds of the tongue, and exists, of course, only on the paralyzed side. Sensory changes in the tongue are not necessarily present. Less clear is the cause of the diminished salivary secretion. Its occurrence is said to point to a lesion above the geniculate ganglion (Wachsmuth).

Among the most common and best known symptoms are the disturbances in hearing, which consist either in an abnormal acuteness of hearing (hyperacusis, oxyacoia) or in a decrease in the power of hearing. In the first case, where we have a kind of hyperæsthesia for all musical tones, the alteration is supposed to be due to a paralysis of the stapedius muscle (which is supplied by the facial) and a consequent overaction of the tensor tympani (Lucae, Hitzig, Roux). The latter—the hardness of hearing—can be due to several causes. We may either have a disease of the middle ear and the adjoining portion of the temporal bone, which has affected the facial nerve by contiguity, or a simultaneous affection of the auditory nerve, which, in the internal auditory meatus, has been exposed to the same deleterious influence, and become affected by the same disease as the facial. Quite lately again the frequency of this combina-

tion of facial paralysis with a slight paralysis of the auditory nerve has been pointed out by O. Rosenbach (cf. lit.).
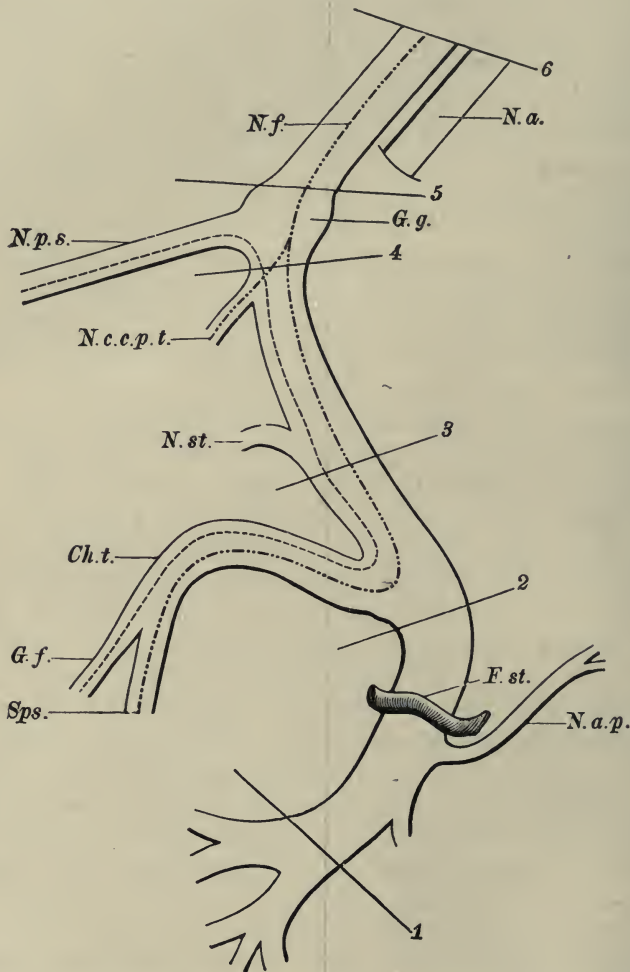
Fig. 16.—Erb's Diagram for Facial Paralysis. Representing the course of the facial trunk from the base of the skull to the pes anserinus. *N. a.*, auditory nerve. *N. f.*, facial nerve. *N. p. s.*, large superficial petrosal nerve. *G. g.*, geniculate ganglion. *N. c. c. p. t.*, communicating branch to tympanic plexus. *N. st.*, stapedius nerve. *Ch. t.*, chorda tympani. *G. f.*, gustatory fibres. *Sps.*, secretory nerve to salivary glands. *F. st.*, stylomastoid foramen. *N. a. p.*, posterior auricular nerve.

2. *The Extracranial Lesion.*

The peripheral paralysis of the facial after its exit from the skull is, as we have already said, the most common. Of this

class the so-called rheumatic form, which is attributed to the influence of cold (*a frigore*), and the traumatic, often observed after operations, gunshot injuries, etc., are the two chief representatives. When any one, heated as he is, passes from a warm room into a cold wintry night, or is exposed to drafts in the railroad cars, and finds himself a few hours later taken with a paralysis of one side of the face, this is the so-called rheumatic form which has attacked the stem of the nerve after its exit from the Fallopian canal. In these cases all three facial branches are affected, and the appearance of the patient is changed in a very material and striking manner. Even the layman notices that he now wrinkles only one half of his forehead, and that the folds and furrows generally present are obliterated on one side ; that the patient can shut one eye only while the other remains wide open and can not be closed despite the strongest efforts. If the attempt is made, the eyelids remain gaping, the eyeball is rolled inward and upward, and the pupil disappears behind the upper lid, a position which is also maintained during sleep (lagophthalmos). The inability to shut the lids prevents the tears from running into the tear ducts and interferes with the process by which foreign bodies, particles of dust and the like, are removed from the eye. It happens, then, that the tears are always running down the cheeks, and that a conjunctivitis, even an ulceration of the cornea, may be developed through the mechanical irritation caused by such foreign bodies. The appearance of the lower part of the face has already been described

Concomitant symptoms, such as we have found in intracranial lesions, are in these cases, as a rule, absent. The patient complains of nothing else than of his inability to move the facial muscles as he wishes and the (secondary) trouble with his eyes. Speech and mastication are interfered with to a marked degree only in severe cases. It has been mentioned already that the reflexes are decreased or lost.

A complication which, though not frequent, is of great interest when met with in peripheral facial paralysis, namely, herpes zoster, has been lately described by Letulle, Strübing, and Voigt (cf. lit.). Whether this is due to an inflammation of the peripheral endings of the fifth, which is transmitted to the facial (Strübing), or whether the stem of the facial contains in parts fibres, an inflammatory irritation of which may produce herpes zoster (Eulenburg), is not clear.

I have only in rare instances seen this complication, and have found that whenever it was present the cases pursued an unusually protracted course.

When a relapse occurs, as happens sometimes, but, after all, rarely, the course is equally unfavorable, according to my own observation. Möbius has reported such instances (cf. lit.), but his experience as to recovery seems to have been less unfavorable than my own. In one of my cases there intervened between the second and first attack eight years. In the first one the cure was effected after four weeks ; in the second not until six and a half months had passed.

The instances of the facial paralysis, which repeatedly attacks the same individual, form an interesting counterpart to the cases where several members of the same family suffer repeatedly from the disease, cases such as Neumann has reported from Charcot's polyclinic of the Salpêtrière (Du rôle de la prédisposition nerveuse dans l'étiologie de la paralysie faciale dite *a frigore.* Arch. de neurologie, July, 1887, xiv, 40). It seems as if the influence of cold, etc., only forms the immediate exciting cause for the disease, and that this would probably not appear unless the patient were burdened with some hereditary tendency — i. e., came from a neuropathic family (Charcot).

Duration and Course.—The duration and course of rheumatic facial paralysis are extremely variable, and it is of great importance for the physician to be able to give at the beginning an approximately accurate opinion as to the length of time necessary for recovery. This we can, however, only do if we investigate the electrical condition of the paralyzed muscles, and hence it follows that it should be our invariable rule to make an electrical examination before venturing upon any expression of opinion. The following are the chief points to guide us :

1. If we find no changes either in faradic or in galvanic excitability the prognosis is favorable ; recovery in from seven to twenty days (light form).

2. If we find the faradic and galvanic excitability of the nerve diminished, but not lost, the galvanic excitability of the muscles, however, increased, and the usual formula of contractions changed (A. C. C. > C. C. C.), then the prognosis is relatively favorable ; recovery in from four to six weeks (intermediate form of Erb).

3. If the reaction of degeneration be found—i. e., if the far-adic and galvanic excitability of the nerve and the faradic excitability of the muscles be lost, while there is an increase in the galvanic excitability of the muscles associated with qualitative changes and changes in the mechanical excitability—then the prognosis is relatively unfavorable, and for recovery two, four, six, eight, even twelve months, may be required (grave form). These are those bad cases in which secondary contractures and spasmodic twitchings of the muscles also appear, which, according to Hitzig's opinion, are to be referred to an obscure abnormal irritation in the medulla oblongata. It is well to know that, as convalescence begins, voluntary motion may return long before the electrical excitability, so that often the patient is able to perform some slight voluntary movements before faradic stimulation provokes the least contraction.

Diagnosis.—With regard to the diagnosis there is even for the beginner no more easily recognizable disease. Still there are cases where it is difficult, not to say whether there is any paralysis, but, strange as it may sound, which is the affected side. One is particularly liable to mistakes in old people, in whom the wrinkled, inelastic skin has produced a stereotyped expression, which, even when the facial muscles contract, is but little changed. Suppose now the muscles to have lost their innervation, the paralyzed side takes on the soft features of an earlier period of life, and this may go so far that the patient believes his rigid, wrinkled side to be the paralyzed, and the affected side the healthy one (Gowers). We also must remember that the non-paralyzed zygomatici pull the face sharply toward the well side, a condition which easily produces in the layman the impression of something abnormal, so that he takes the side thus distorted for the diseased one. In general, however, we may say that the diagnosis of a peripheral facial paralysis is one of the easiest imaginable in neuropathology.

Treatment.—In the treatment we may in recent cases recommend for trial steam-baths and counter-irritation to the skin; but never, unless there is a special indication, should internal remedies be advised, because in a non-complicated rheumatic facial paralysis they are absolutely superfluous. In more protracted cases the methodical use of electricity is strongly indicated, for even though it is undoubtedly true that the disease, if the prognosis is at all favorable, gets well of its own accord, and really requires no treatment at all, there can, on the other

hand, be no doubt but that the electrical treatment hastens the cure in a marked degree; therefore, electricity should be used under all circumstances. Just which method should be employed can not be definitely laid down, but we should keep in mind that not only the galvanic current is beneficial, but that the faradic brush applied to the stem and the individual branches of the facial gives good results, and the patient should, therefore, be persuaded to submit to this somewhat
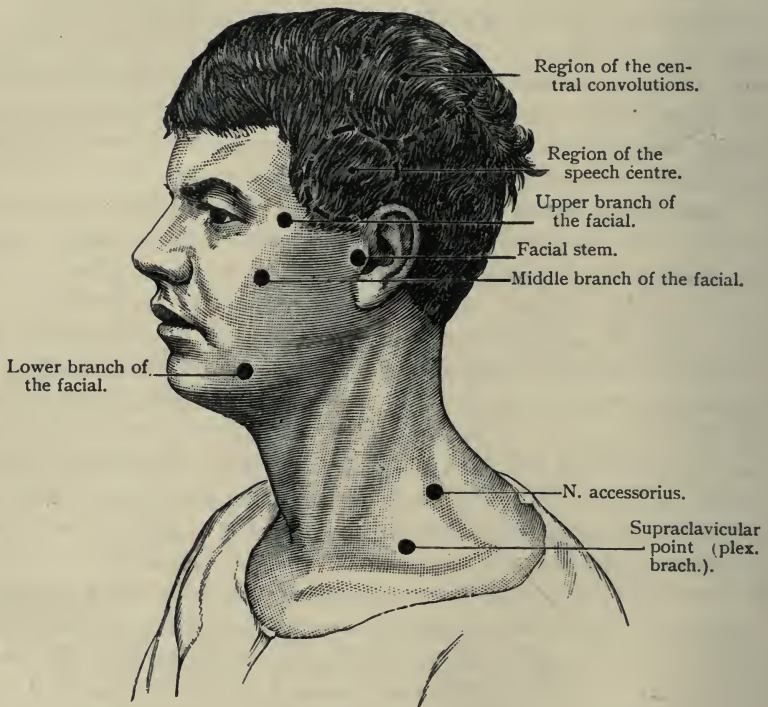
Fig. 17.—Some of the so-called "Motor Points" on the Face and Neck.

disagreeable procedure. The places from which the most important facial muscles can best be stimulated are seen in Fig. 17. At these points the motor-nerve branches to the muscles concerned lie very near the surface. They are called "motor points" (Ziemssen). In galvanization every specialist has his pet method of application and his own ideas about the strength and direction of the current. The one prefers to apply the electrode over the mastoid process, placing either the anode or the cathode on the affected side of the face; another treats at

the same time the sympathetic in the neck; a third, again, applies the anode over the affected nerve and the cathode to an indifferent point, and so forth. Whichever method we may prefer, the main thing, after all, is to produce by repeated opening and closing of the current contractions of the muscles by which the tonus of the latter will soon be improved. I should like to mention, too, that I have seen the application of the galvanic brush and the use of the combined current (de Wattewille) repeatedly attended with satisfactory results.

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CHAPTER VI.

DISEASES OF THE AUDITORY NERVE.

THE auditory nerve emerges at the base of the brain, alongside of the facial, and takes with this latter a forward and outward course. After having entered the internal auditory meatus, it divides before reaching the cribriform plate, which separates the internal meatus from the internal ear, into two main branches, an anterior inferior and a posterior superior. These nerves pass as small filaments through the openings in the plate, to be distributed respectively to the cochlea and vestibule, and are hence called ramus cochlearis and ramus vestibularis.

The cortical centre of the nerve is probably to be sought for in the temporal lobe; the fibres are said to run through the last third of the posterior division of the internal capsule, through the middle geniculate body, through the brachia conjunctiva posteriora, the posterior corpora quadrigemina, and the inferior fillet (v. Monakow, Baginsky).

About the situation of the nuclei of the auditory nerve there seems still to exist a difference of opinion among the anatomists. Usually two nuclei are distinguished, an inner or principal nucleus and an outer one situated laterally from the first. In their structure these present material differences. While the former—the inner nucleus—only contains scattered, small, slender, ganglionic cells (15 to 20 μ long), the latter contains cells of considerable size (60 to 100 μ long and 15 to 21 μ broad). The situation of the two nuclei may be understood from the accompanying diagram.

Of the two roots, the superficial terminates in the internal auditory nucleus, while the deeper one passes between the restiform body and the ascending root of the fifth, and turns toward the outer one. This, also, the diagram, which is taken from Wernicke, and which demonstrates the views of Meynert, illustrates.

Although the diseases of the auditory nerve are not, as a rule, treated of in neurological text-books, they are found sometimes so closely connected with other nervous diseases, and

are, notwithstanding their comparative rarity, of such decided practical importance, that we feel not only justified but compelled to consider them here, at least briefly.

The nerve, as we have said, rarely ever becomes primarily diseased, but diseases of the middle and internal ear—that is, peripheral affections—are by far the most common causes of
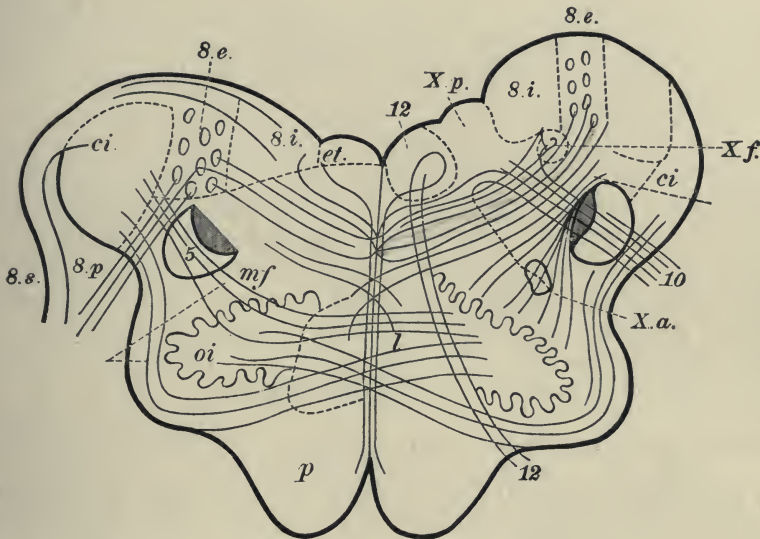
Fig. 18.—DIAGRAMMATIC SECTION THROUGH THE MEDULLA OBLONGATA IN THE REGION OF THE (LOWER) OLIVE. The right half represents a lower plane. p, pyramids; oi, lower olive; l, lemniscus; mf, motor region of the tegmentum; 5, ascending root of the fifth; $ci.$, corp. restif.; 8.$e.$, external, 8.$i.$, internal nucleus of the auditory; $et.$, emin. teres; 12, nucleus and root of the hypoglossus; 10, root of the vagus; $X.a.$, anterior, $X.p.$, posterior vagus nucleus; $X.f.$, combined root of the "lateral mixed system" (cf. p. 105); 8.$s.$, superficial, 8.$p.$, deep root of the auditory nerve.

diminution or loss of hearing. We may distinguish between conditions of irritation and those of paralysis, so that on the one hand we shall have hyperæsthesias, on the other pareses or paralyses.

I. HYPERÆSTHESIAS OF THE AUDITORY NERVE.

We speak of a hyperæsthesia of the auditory nerve where the patient experiences a painful sensation in his ear when perceiving certain sounds or noises. For instance, in excitable and nervous individuals who suffer from hemicrania or tic douloureux, such a sensation may be produced by high musical

notes, whistling, and the like. Quite a different affection is an abnormal acuteness of hearing, which is extremely rare, the so-called oxyacoia of which we have spoken in the chapter on facial paralysis.

Frequently one hears nervous patients complaining of subjective auditory perceptions, roaring, buzzing, hissing, singing, humming, and the so-called nervous tinnitus aurium, which may persist during the whole life without a sign of any other disturbances of function. This symptom may be due to a purely functional disorder or it may be the forerunner of a middle-ear sclerosis.

Therapeutically, we may, after the removal of masses of cerumen or epidermis which may have obstructed the outer canal, with benefit make use of blisters, stimulating lotions applied to the mastoid process, subcutaneous injections of morphine, the bromides, digitalis, and atropine. If abnormalities of tension in the sound-conducting apparatus and consequent rise of pressure in the labyrinth be the cause of the disorder, then the inflation of the middle ear and the rarefaction of the air in the outer canal is to be recommended.

II. The Pareses and Paralyses of the Auditory Nerve.

Analogous to the rheumatic facial paralysis we have a condition in the auditory nerve which manifests itself in either a decrease or a loss of the function of hearing, the so-called rheumatic acusticus paralysis. It is less frequent than the former, although the cause of both, namely, cold, is the same. Central paralyses are always connected with decrease of hearing power on one side only. Absolute unilateral deafness, as a consequence of a focal lesion in one of the hemispheres, has up till now not been observed (Wernicke). Whether the disturbances in hearing observed by Baginsky in railway spine are of a central or peripheral nature remains yet to be studied (cf. lit.).

Next in order we have to mention in this connection the anæsthesia and paresis of the auditory nerve, which sometimes appear quite suddenly in the course of hysteria, and often as suddenly disappear again after a longer or shorter period of time. We shall consider these forms more carefully in the chapter on hysteria.

Of interest from a pathological standpoint is the nervous deafness occurring after an epidemic cerebro-spinal meningitis. It is this form which has been so thoroughly studied by Moos.

There is hardly any doubt but that it is caused by the passage of purulent masses from the meninges along the sheath of the auditory nerve into the inner ear. The prognosis is unfavorable. A diminution of hearing, probably due to transitory circulatory disturbances, occurs sometimes after epileptic attacks. Although not common, this affection is certainly well authenticated.

Of especial interest ætiologically are the disorders of hearing which we find in engineers and firemen on the railroads as a consequence of their occupation. This must principally be attributed to the noise, aided, however, to some extent by the abrupt and severe changes of temperature and the exposure to all kinds of weather. We do not know anything positive about the relative frequency of this affection, which consists in a more or less pronounced decrease of hearing, but in the general interest of the public it certainly deserves as much attention on the part of the companies as the color-blindness which has for years been carefully looked into. Locksmiths, blacksmiths, and boiler-makers, whose auditory nerves are also being constantly overstimulated, suffer from similar disorders. In rare and exceptional cases it has been observed that mechanics who are "hard of hearing" hear better during the usual noise connected with their work than when everything around them is quiet—paracusis Willisii. This very remarkable phenomenon is probably due to a decrease in the vibratory power of the auditory ossicles, owing to which the sound is conducted with more difficulty, a condition which is obviated by a more forcible concussion (Buerkner, Roosa). We would not leave unmentioned the fact that an overtaxation of the auditory nerves lasting for years causes great nervousness, and may even predispose to mental diseases.

In the treatment, endermic inunctions of strychnine (0.1 to glycerin 10.0 (gr. jss.; glycerin, ℨ ijss.)—sig., ten drops) over the mastoid process, and fumes of sulphuric ether conducted by a catheter into the tympanic cavity to act on the distribution of the acusticus, deserve recommendation. A beneficial effect from the galvanic current can be expected only if examination assures us that the current has a modifying influence on the subjective noises or upon the power of hearing. This treatment necessitates a knowledge of the investigations of Brenner on the galvanic reactions of the auditory nerve (cf. Erb, Handbuch der Elektrotherapie, 1882, page 226).

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III. Ménière's Disease—Ménière's Vertigo—Vertigo ab aure læsa—Vertigo in General.

When we speak of Ménière's disease we mean a combination of symptoms which is made up (1) of subjective noises in the ear, (2) a feeling of dizziness, accompanied with vomiting, (3) a gradually increasing difficulty of hearing, sometimes ending in deafness.

On account of the exceptionally practical importance which has to be attributed to the so-called vertigo (*le vertige, Schwindelgefühl*), we may be allowed to make some general remarks on this before considering the special form, viz., Ménière's disease.

By vertigo we mean a subjective feeling of motion appearing suddenly or gradually without any loss of consciousness, attended by a simultaneous sensation of loss of equilibrium. The subjective sense of motion is either referred to the body or parts of it, or to surrounding objects. The motion is in different directions, sometimes in horizontal or vertical circles, revolving with their convexity sometimes forward, sometimes backward, and the older observers distinguish accordingly a vertigo titubans, fluctuans, etc., from the nutatio—that is, subjective movements in a straight line. As concomitant symptoms we note headache, especially in the back of the head, anxiety, tremor, cold sweat on the face, nausea, vomiting; in grave cases, transient loss of consciousness, as in the prodromal state of an apoplectic attack. If consciousness is completely retained, as happens in the majority of cases, the subjective sensation of movement often gives rise to objective voluntary movements, to be regarded in a measure as instinctive efforts against the threatening danger of falling. The patient plants his feet firmly on the ground, stretches out his arms into the air, seizes with his hand any object within his reach, etc., but, in spite of all, he may, notwithstanding the perfect retention of consciousness, fall, owing to the feeling of disturbed equilibrium—vertigo caduca.

If the patient is unconscious—e. g., asleep—then he experiences a sensation of falling down from a great height, down steps, out of the window; he imagines himself sinking into an opening in the ground, etc. This so-called nocturnal vertigo (*Traumschwindel*) usually torments those who suffer from vertigo when awake. Two exquisite examples of this vertigo I have observed in Bright's disease.

By far most commonly the vertigo occurs in paroxysms which appear without regularity and are of variable duration. Between the first and second sometimes hours and days, more rarely months, and indeed whole years, intervene, and only exceptionally—e. g., in cerebellar affections—do the subjective sensations of movement persist uninterruptedly, and thus render the vertigo constant.

The position of the body has rarely any influence on the vertigo, for although at times some amelioration is felt on sitting down, there are cases in which the vertigo continues even when the patient occupies the horizontal position in bed. The pathogenesis of the trouble—that is to say, the organic changes in the brain which are necessary for the production of the sensation—are but little understood. It is generally supposed that changes in the blood-pressure, due, perhaps, to stimulation or paralysis of the vaso-motor nerves, are the chief cause of vertigo, just as a lasting decrease or increase in the amount of blood in the brain can probably give rise to attacks of dizziness. Until the conditions under which vertigo can appear in otherwise healthy people are more accurately understood, our knowledge of the pathological influences at work can be only imperfect. Of great interest are the experiments of Purkinje, undertaken sixty years ago, as to the influence of swinging, and especially of circular movements, in the production of vertigo, These were published in Rust's Magazin für die gesammte Heilkunde, part 23, 1827, and have been reprinted in Romberg's Nervenkrankheiten (*loc. cit.*, p. 118) with this addition by the author: "From all these experiments we see that, taking the head as a sphere, around the axis of which the true motion takes place, an imaginary plane through it determines in every case the apparent motion of the objects in the subsequent position of the head at rest. The same holds good in attacks of vertigo."

Johannes Müller also has made experiments on vertigo, and is inclined to attribute it to the after-effects of visual impressions on the retina. That this, however, is not always the case is shown by the fact that vertigo may appear in people whose eyes are closed, and even in the blind.

We have already spoken about its occurrence in the paralysis of the ocular muscles (p. 45). Here let us add that this ocular or visual vertigo disappears if the patient closes the affected eye or holds his head in such a position that the

paralyzed muscle does not come into play during the act of seeing.

In the present chapter we shall discuss more especially how far diseases of the internal and middle ear are connected with vertigo. It has repeatedly been observed that affections of the nasal mucous membrane, swellings of the erectile tissue, have produced it. If, then, we add that it has been claimed that the intestines (intestinal worms, tænia, ascaris) and the stomach are responsible for feelings of dizziness, which Trousseau calls "*vertigo a stomacho læso,*" we can not fail to be impressed with the complexity and the lack of clearness in the ætiology of this affection. We must, however, always keep in mind, no matter where the remote cause lies, be it in the faulty movements of the ocular muscles, in the nose, in the ears, or in the stomach, etc., we must keep in mind, I say, that the influence of the cerebrum and the cerebellum is under all circumstances quite essential for the production of vertigo. Whether the characteristics of the vertigo vary or not with the different organs affected is not yet clearly known.

The disease described by Gerlier in Ferney, which shows itself by a very pronounced dizzy feeling, appearing in paroxysms—the so-called "paralyzing vertigo"—is accompanied by other symptoms, namely, a weakness, resembling a paralysis, in the extremities, drooping of the eyelids, and extraordinary lassitude without any loss of consciousness. This condition, which has been repeatedly observed in the canton of Geneva, where it occurs epidemically among laborers and herdsmen, is ætiologically mysterious. Gerlier attributes it to miasmata from marshes and stables, but this does not explain the immunity of the female sex. For this new and as yet entirely strange neurosis Gerlier has proposed the name vertige paralysant (Progrès méd., 1887, 26; Deutsche med. Zeitung, 1887, 44, 1888, 24).

Middle life and moderately advanced age (especially in the female sex, and so in them the climacteric period) seem to predispose to attacks of vertigo, which chooses by preference its victims from among vigorous and full-blooded individuals. Its frequent occurrence in advanced old age will not surprise us if we remember the atheromatous condition of the arterial walls and the consequent irregularities of the blood supply to the brain substance. Among the exciting causes, unaccustomed circular rocking movements, such as we feel on board ship, play an important *rôle*—it is interesting that children possess a

wonderful immunity (sucklings rarely or never become sea-sick)—prolonged stooping, straining at stool, an overloaded stomach, can give rise to vertigo. The dizziness experienced on looking down from a height—the "height dizziness"—which has erroneously been attributed to a fear of danger, is probably a reflex movement evoked by a wrong conception of our position in space, the result of a purely optical illusion ; for its production not only the cerebrum and cerebellum, but also the action of the retina is needed.

The prognosis in vertigo depends upon the nature of the primary disease, and Boerhaave's expression, "vertigo est omnium morborum capitis levissimus et facillime curabilis," has to be taken *cum grano salis*. In an organic lesion of the cerebellum—that is, of the vermiform process—we can expect no improvement in the vertigo, while if it is attributable to an anæmia of the brain, occurring as a symptom of a general anæmia, the outlook is decidedly favorable.

In the same way the treatment will be different in different cases according to the primary disease, which always has to be taken into consideration. For the symptomatic or prophylactic treatment, the repeated administration of mild laxatives, the frequent use of strong stimuli to the skin, such as cold douches, brushing of the neck and the back, mustard plasters, regular bodily exercise, and well-regulated diet, are to be recommended, while any overloading of the stomach, especially in the evening, should be strenuously avoided. In spite of the much-lauded remedies (cocaine, etc.) we do not possess any reliable medicinal treatment for sea-sickness and height dizziness.

After this digression we will return to the consideration of that form of vertigo which is especially connected with aural disturbances. Notwithstanding the fact that it is by no means settled that the above-mentioned combination of symptoms constituting Ménière's disease can be produced by a pure neurosis of the auditory nerve, we will take it up here, because under all circumstances this nerve plays a prominent part in the pathology of the affection.

Since Ménière in 1861 first described the disease, it has been repeatedly observed and carefully studied by German physicians. All have, however, failed as yet to give us a clear understanding of its pathology. Ménière himself believed that

an extravasation of blood or an acute exudation takes place into the labyrinth, which produces the same symptoms as occur in animals after injury to the semicircular canals. This view is in so far incorrect in that cerebral affections, accumulations of cerumen, and diseases of the middle ear, can undoubtedly produce the same symptoms; and then we have to remember that not the hæmorrhage nor the exudation, but its action upon certain parts of the membranous labyrinth is necessary before the symptoms occur (Politzer). It can easily be imagined that whenever the extravasation stimulates the nerves of the ampullæ Ménière's symptoms are produced, while they are absent if the hæmorrhage does not directly press upon the nerves of the antrum or the ampullæ (Politzer).

Quite recently Brunner (cf. lit.) has put forward a supposition which we think is worth considering, namely, that we may be dealing with a vaso-motor neurosis of the vessels of the labyrinth. According to him the pressure in the labyrinth acts in a similar way as pressure in the cranial cavity, where considerable changes are borne as long as the normal expansion of the subdural and subarachnoid space is not interfered with. He thinks, therefore, that narrowings of the labyrinth could produce a predisposition to Ménière's disease.

This hypothesis is extremely plausible, especially as the symptoms appear paroxysmally, and in the intervals the patient is apparently in perfect health. In this way also the favorable action of quinine can be explained if we suppose that it diminishes the hyperæmia in the semicircular canals, just as Horner has shown to be the case for the retinal vessels. He observed that large doses of quinine constantly produced considerable ischæmia in the latter. The question is, however, not settled, and we have to confine ourselves for the present to a most careful study of the clinical manifestations of the disease.

There are hardly two cases in which the symptoms are exactly the same, and the course is so far from being uniform that we can not be surprised if often great uncertainty about the diagnosis prevails. The onset even is very variable. Now it is sudden with loss of consciousness and apoplectiform symptoms, etc.; again it is gradual, first, subjective noises in the ears being noticed, sometimes comparable to the whistle of a locomotive, sometimes to the rustling of the leaves in the forest. Next comes a feeling of dizziness, at times only moderate, at times so pronounced that the patient in spite of all his efforts

falls to the ground. Vomiting may be present or absent. Finally, a decrease in the power of hearing, first in one, then in the other ear, becomes noticeable. Some cases show a decided progressive tendency. After short remissions the symptoms always reappear with increased severity, the vertigo gains so much in intensity that now the patient repeatedly falls with great violence, vomiting becomes more and more frequent, and the patient becomes at first incapable of following his calling, and finally is reduced to the state of a useless member of society. In rare instances periods of marked improvement, which may indeed last for years, occur. In these even the difficulty in hearing may be gradually diminished, and the prognosis becomes relatively favorable. Finally, it is at times observed that with the full development of the deafness all the other symptoms, buzzing in the ears, vertigo, and vomiting, disappear. In other words, we have what we call a relative recovery or recovery with defect. In any given case we are never in a position to predict the outcome, and have always to be very guarded in our prognosis.

Of considerable diagnostic importance is the fact that usually the examination of the drum and the Eustachian tube does not reveal any changes, and that neither cranial nor spinal nerves present any disturbances of function. Rinne's test gives variable results in Ménière's disease. This test consists, as is well known, in applying a vibrating tuning-fork with moderate pressure first over the mastoid process, leaving it there until the patient seems to hear the sound, and then as quickly as possible bringing it immediately in front of the external meatus, avoiding all contact with the head or ear. If the patient then is able to hear the sound of the tuning-fork once more, this is a sign that, as is normally the case, the conduction through the air is better than through the bone. If, on the other hand, he does not hear it, the conduction through the air must in some way be interfered with. In the diagnosis these are points to be considered.

In the treatment, above all, the action of large doses of quinine—0.7–1.0 (gr. x–xv) *pro die*—must be tried, a procedure warmly recommended by Charçot, and later used with gratifying results by Féré, Moos, and others. In many cases, as we have said, the effect is very marked and there is no need to seek further for other medication. At times, however, this will fail, and then we are forced to resort to a two-per-cent solution of

pilocarpine (nine to ten drops subcutaneously), a drug which is supposed to favor absorption. The result is often surprising. I have seen grave symptoms completely subside after three or four days' use of this medicine. The injections are to be continued every second day for three or four weeks, and, as a rule, after the fifteenth dose the treatment can be discontinued, at any rate for a time. We need not add that on administering this drug the general condition of the patient must be carefully looked after, and any symptoms of collapse guarded against by the timely exhibition of stimulants, wine, and the like.

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CHAPTER VII.

THE glosso-pharyngeal nerve leaves the brain between the root fibres of the auditory and those of the vagus, at the side of the medulla oblongata, by five or six filaments; these soon unite to form an anterior (small) and a posterior (larger) bundle; they both pass outward, under and in front of the flocculus, to the anterior division of the jugular foramen, through which the nerve leaves the skull. Whether the so-called jugular ganglion which the nerve presents while still inside the skull has to be looked upon as a special ganglion or only as a group of nerve-cells which have separated themselves from the petrous ganglion, which is seen on the nerve immediately after its exit from the skull, remains to be decided.

The glosso-pharyngeal has no nucleus of its own, but originates in a large collection of nerve cells, which are regarded as the nucleus common to this nerve, the vagus, and the accessorius. This nucleus is situated midway between the anterior and posterior spinal roots. In the manner in which its root fibres originate it corresponds partly to the motor, partly to the sensory type (Wernicke). It is therefore designated as the mixed lateral system (Deiters), and it is supposed that the glosso-pharyngeal originates in the upper, the vagus in the middle, and the accessory in the inferior portion of the nucleus (cf. Fig. 18, p. 95). The manner in which this common nucleus is composed is not yet understood, nor do we know how many modes of origin for root fibres of this "lateral mixed system" we have to assume. Exact data may be found in Wernicke's text-book, i, p. 155 *et seq.*

The glosso-pharyngeal, which, according to our present ideas, has to be regarded as the only genuine nerve of taste, is the third one which is to be taken into consideration in the examination of the functions of taste. The trigeminus (the third branch (lingual), possibly also the second branch) and the facial (chorda tympani) we have treated of, and it remains, therefore, to determine whether and if so under what condi-

tions diseases confined to the glosso-pharyngeal occur, and in what manner taste is altered by them. Since it only supplies the posterior third of the tongue with sensory fibres (ramus lingualis nervi glosso-pharyngei), it is not to be wondered at that, in determining an isolated affection of the nerve, we not rarely meet with considerable difficulties.

We know but little about central diseases of this nerve. It is supposed, however, that there exists a bulbar affection, a gray degeneration of the nucleus which is found in tabes (Erben), also that the gustatory paths may be in a state of irritation which gives rise to alterations in taste-perception analogous to the paræsthesias which occur with irritation of the paths of tactile sense in the posterior columns of the cord. Conduction anæsthesias are also said to occur, although it is impossible to decide whether only the glosso-pharyngeal or whether in addition the trigeminal and the facial paths are concerned (cf. Fränkel, Berl. klin. Wochenschr., No. 3, 1875). A central paralysis of taste manifesting itself solely on the posterior third of the tongue has never been observed. With the cortical centre of the glosso-pharyngeal we are not as yet acquainted.

Peripheral anæsthesia, anæsthesia gustatoria, ageusia (*a*, priv., γευσις, sense of taste), impairment or loss of taste produced by affections of the peripheral nerve endings, has been met with in diseases of the mucous membrane of the tongue, and has been known to be produced by the action of low temperatures (ice) or acrid substances (vinegar, chewing tobacco, red pepper). In testing for such alterations the patient is asked to close his eyes, open his mouth widely, and protrude his tongue ; then a small portion of sugar or quinine, etc., is placed upon that part of the tongue the function of which is to be tested, and the patient is to indicate with his finger where he perceives the taste before he retracts his tongue, and tell us by signs what he has tasted. The test is made with bitter, sour, sweet, and salty substances, and for the purpose any one, as long as it is not poisonous, may be selected. Further, it is possible to accurately determine the boundaries of the area with normal and that with disturbed function of the tongue by means of the galvanic current. As we know, a sour, metallic, the so-called galvanic taste is perceived if the electrode is placed upon the tongue and the current is closed ; the same taste is experienced during galvanization of the throat, the neck, or head, and is probably produced by the current acting upon the

taste nerves in their peripheral or central course. The use of the galvanic current is also to be recommended in the treatment of the affections of the nerve.

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CHAPTER VIII.

IMMEDIATELY behind the superficial origin of the glosso-pharyngeal on the postero-lateral aspect of the medulla oblongata, the vagus appears, with its ten to fifteen separate bundles, which soon unite to form one trunk. This is a flat band which, accompanied by a process of the dura, passes outward below the flocculus, together with the accessorius, to the anterior division of the jugular foramen, inside of which is to be found the ganglion of the root of the vagus, or, as it is also called, the jugular ganglion. After its exit from the skull the vagus receives a part of the accessorius, and forms the gangliform plexus or the ganglion of the trunk, which, however, only transmits a part of its fibres.

About the difference in the further course of the left and right vagus we shall have a few words to say later.

That the nucleus of the vagus is only a part of the nucleus common to it, the glosso-pharyngeal, and the accessorius, has already been stated in the preceding chapter. The cells of the part belonging properly to the vagus are spindle-shaped, multipolar, 30 to 45 μ long and 12 to 15 μ broad (hence much smaller than the cells of the hypoglossus nucleus, which we shall describe later). As another important origin of the root fibres of the vagus, a compact round nerve bundle following the longitudinal axis of the medulla oblongata must be mentioned. It has been described by Meynert as the solitary fasciculus, while Krause designates it as the respiratory fasciculus, because it connects the vagus with the origin of the most important respiratory muscles (cf. Fig. 19). The so-called nucleus ambiguus (in the diagram *n. am*) is held to be still another nucleus of the vagus. This is a collection of peculiar nerve cells situated within the formatio reticularis to the mesial side of the nucleus lateralis.

Just as most of the cranial nerves, the vagus may be diseased in its centre as well as in its peripheral course. The first class of cases are usually met with as partial manifesta-

tions of other, general, diseases (tabes, hysteria). The latter are distinct affections in themselves, which may occasionally be due to peripheral causes, such as indigestion, catching cold, or reflex influences, diseases of the intestines and the uterus. Very frequently, it is true, the seat of the disease remains ante as well as post mortem obscure, and this is not to be won-dered at if we remember that we know little or nothing about the pathological anato-my of the vagus. Among the cases hitherto observed, many were not fitted to throw any light on the symptoms mani-fested during life, as in nu-merous instances no abnor-mality at all was found in the nerve, so that we are led to assume that the disease was purely functional (i. e., a dis-ease without appreciable ana-tomical basis). The pathol-ogy of the vagus, therefore, belongs to the most obscure chapters in the pathology of the cranial nerves, and the following can only be consid-ered to be an imperfect at-tempt at giving a compre-hensive exposition of the high-ly interesting diseases con-nected with this nerve.

Fig. 19.—Cross-section through the Me-dulla Oblongata. (After Schwalbe.) *a. V*, ascending root of the fifth. *n.XII*, nucleus of the hypoglossus. *n.X* and *n.X'*, nucleus of the vagus. *XII*, hypoglossal nerve. *fs*, solitary funiculus (respiratory fasciculus). *p*, pyramidal tract. *o*, olive. *i.N*, pyramidal nucleus. *f.l.a.*, anterior lon-gitudinal fissure. *n.am*, nucleus ambiguus. *n. l.*, nucleus lateralis.

Since the symptoms may sometimes be the same whether the disease is of central or peripheral origin, we shall, so as to avoid repetition, deviate from our usual method of division, and give our attention chiefly to the question how lesions of the vagus may influence (*a*) respiration, (*b*) circulation, (*c*) digestion, functions which, as is well known, are chiefly under the con-trol of this nerve; next, we shall take up the consideration of exophthalmic goitre, an affection which possibly ought to be classed among the vagus neuroses.

A. Affections of the Air-passages due to Lesions of the Vagus.

1. The larynx, above all, interests us in this connection. This organ is innervated by the vagus and the accessorius, though it is still a matter of doubt whether all the motor fibres originate from the latter or only those that innervate the muscles used in the production of voice, while the vagus presides over the respiratory movements of the vocal cords; the sensory fibres of the larynx certainly all belong to the vagus.

The branches of the vagus, which come off in the cervical portion of the nerve and innervate the laryngeal muscles, are the superior laryngeal and the inferior or recurrent laryngeal. The former leaves the vagus at the lower end of the gangliform plexus, and divides into a motor branch, which goes to the crico-thyroid muscle, and into a sensory branch, which contains the fibres for the mucous membrane of the epiglottis and the whole laryngeal mucous membrane above the vocal cords.

The recurrent laryngeal is shorter on the right side, because, without going beyond the upper aperture of the thorax, it curls around the subclavian artery, and runs back in a groove between the trachea and the œsophagus upward to the larynx, while on the left side it has to make the long course around the arch of the aorta. Its terminal branch (R. terminalis) divides into two twigs, which together supply all the muscles of the larynx, with the exception of the above-mentioned crico-thyroid, with motor nerves, and the mucous membrane of the parts below the vocal cords with sensory fibres.

Of the laryngeal muscles, the posterior crico-arytenoids draw the vocal cords apart—that is, they are the abductors or openers—while the lateral crico-arytenoids in conjunction with the lateral thyro-arytenoids draw them together, and are therefore called adductors or closers. Of these muscles, on each side the "abductor" arises at the posterior surface of the cricoid cartilage and passes upward and outward to the end of the muscular process of the arytenoid cartilage, while the other, the "adductor," arises from the upper margin of the cricoid cartilage and is inserted at the outer side of the muscular process of the arytenoid cartilage. It moves the muscular process forward, being thus the antagonist of the abductor. The crico-thyroids provide for the elongation and tension of the vocal cords; they are assisted by the internal thyro-arytenoids, which run parallel with the vocal cords.

In the laryngeal muscles paralysis and, though comparatively rarely, spasms have been observed.

The chief forms of paralysis, which we shall here consider, are (1) the paralysis of the recurrent laryngeal, in which case all the muscles supplied by this nerve are paralyzed (or weakened); (2) the so-called abductor paralysis—that is, paralysis of the posterior crico-arytenoids, the openers of the glottis; (3) paralysis of the internal thyro-arytenoids.

Without going into the much-discussed and still unsettled question as to the mechanism of these paralyses, we have attempted to give a succinct and clear summary of the clinical symptoms, including the appearances found on laryngoscopical examination (cf. table on page 114).

The existence of a cerebral centre for the laryngeal muscles is shown by the fact that in different cerebral affections—e. g., pseudo-bulbar paralysis and certain brain tumors—paresis or paralysis of the vocal cords has been observed. In chorea adductor paresis has been noted. A most curiously perverted action of the vocal cords has been observed by Krause in the course of hysteria; on inspiration they were approached, while on expiration the glottis was wide and gaping.

Another form of central paralysis is the nuclear. In complete paralysis of one vocal cord a lesion in the accessorius nucleus of the corresponding side has been found; the usual cause of this, however, seems to be a peripheral affection of the trunk of the vagus, or of the recurrent laryngeal (by pressure, contusion, injuries, surgical operations, tumors, and aneurisms), yet we are not often in a position to speak with certainty as to the seat of the affection, and to say whether this is central or peripheral. The nature of the laryngeal paralyses which occur in general neuroses (hysteria, epilepsy), intoxications (lead), infectious diseases (diphtheria, dysentery, cholera), is quite obscure. The easiest to understand are those acquired through straining of the voice and diseases of the larynx itself (catarrh, perichondritis). (B. Fränkel on mogiphonia, cf. lit.)

The prognosis ought to be guided by the consideration of the nature of the primary affection, but we should also take into consideration the functions of the affected muscles, and not forget that, for instance, in abductor paralysis, danger of suffocation may arise at any moment. It is always unwise to predict the exact time of recovery; the course of such paralyses is usually very protracted.

The treatment of most of the cases has to be conducted by a specialist, and consists in touching the vocal cords with the

sound (Rossbach), and in the external or intralaryngeal use of electricity. Faradization of the different laryngeal muscles necessitates a dexterity which can only be attained after a thorough acquaintance with the laryngoscopical technique. The general treatment of any primary affection need not be discussed here.

Spasms of the laryngeal muscles, we have said before, are very rare, and are in general, with the exception of the spasm of the glottis, of not much practical importance. Most frequently the spasm affects the adductors, and the condition then resembles very much that of abductor paralysis, with this exception, that the spasm is generally quite transitory, while the paralysis is often of long duration. The aphonia spastica described by Schnitzler, a disturbance of co-ordination of the muscles of the vocal cords, which, on an attempt at phonation, contract spasmodically, is found occasionally in chorea and hysteria.

The spasm of the adductors, which occurs especially in early childhood, is called spasm of the glottis (laryngismus stridulus, laryngospasmus, asthma thymicum sive Millari). Its paroxysms usually occur unexpectedly without external cause. They consist in the main in a total arrest of respiration lasting from several seconds to a minute and a half, and are ushered in by a deep inspiration which is accompanied by signs of suffocation. Only rarely does the child die during the attack; usually a few deep, very audible respirations indicate the cessation of the spasm, and the child seems completely well after a comparatively short while. No definite statement can be made with regard to the number and intensity of the individual attacks, because innumerable variations can occur. The anatomical seat of the disease is entirely unknown; yet the fact that not rarely eclampsia or epilepsy complicates the affection rather speaks for the possibility of a temporary irritation of the cortical centre for the laryngeal muscles. The remarkably frequent occurrence of it in conjunction with rachitis has led to the idea (Elsässer) that we are dealing with a rachitic softening of the posterior part of the skull, which has rendered possible pressure upon the brain. Nothing definite is known about the cause. In the treatment early hardening of the child and rational nutrition play an important *rôle*. Robust, well-nourished children who can stand changes in temperature without at once catching cold, etc., are hardly ever affected with laryngis-

mus stridulus; only delicate children with a convulsive tend-
ency, who have been fed on farinaceous foods and other inap-
propriate substitutes for the mother's milk, fall a prey to the
disease. There is no medicinal treatment for the affection.
During the attacks we have to avoid the danger of suffocation
by carefully watching the epiglottis, sprinkling the body with
ice-water, brushing and tickling the soles of the feet. After
the attack we may give nervines (belladonna, bromide) and,
perhaps to avoid a too frequent repetition, narcotics (morphine,
1 to 3 milligrammes—gr. $\frac{1}{60}$–$\frac{1}{20}$ subcutaneously). The treatment
of the rachitis should never be omitted.

Sensory disturbances of the larynx manifest themselves
either in anæsthesias, or, what is less common, in hyperæsthe-
sias of the mucous membrane, and are especially found in the
distribution of the superior laryngeal. They are not rarely
combined with motor changes, paralysis or paresis of the pha-
ryngeal muscles (cf. chapter xi), but often they appear alone.
The most common form is the anæsthesia attending diphthe-
ritic paralysis; it is characterized by the absence of the re-
flex gagging and cough which normally follow touching the
laryngeal mucous membrane with the sound, the finger, or the
laryngoscope. In such cases it may happen that the food on
deglutition enters the larynx, and, through faulty closure of
the glottis, can not be removed by coughing, and thus gives
rise to dangerous attacks of choking, and even to aspiration
pneumonia. The latter does not seem to occur in cases of
purely hysterical anæsthesia.

The hyperæsthesia is found in ulcerative processes, or in
bad, acute catarrhs. Although it seems to play a prominent
rôle in hysterical patients, it is in reality not present, but is
either simulated or erroneously said to exist by patients who
are forever worrying themselves and finding out new ailments.

The anæsthesia calls for electrical treatment, galvanization
of the larynx and the palatal muscles, the faradic brush to the
throat, etc. To meet the hyperæsthesia, narcotic remedies may
be of service, but in hysterical patients often no other treat-
ment but a good sensible lecture is needed.

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TABLE OF THE MOST COMMON PARALYSES OF THE LARYN-
GEAL MUSCLES.

	Kind of paralysis.	Occurrence.	Symptoms.	Ophthalmoscopic picture.
INFERIOR OR RECURRENT LARYNGEAL.	Complete recurrent laryngeal palsy.	In compression paralyses of the vagus or the recurrent laryngeal (carcinoma œsophagi), often unilateral (left), as initial symptom of aortic aneurism. In tabes.	Voice not clear. Patient is easily tired on talking. Coughing impossible.	Vocal cords slightly abducted, the so-called "cadaveric position" (Fig. 20). In forcible phonation the healthy cord reaches beyond the middle line. Overriding of the arytenoid cartilages (Figs. 21, 22).
	Abductor paralysis (paralysis of the posterior crico-arytenoids).	In diseases of the nerve itself, the causes of which are often unknown.	If bilateral: extreme inspiratory dyspnœa; if unilateral: inspiration hampered, long-drawn, noisy. Dyspnœa on the least exertion. Speech but little affected.	Glottis appears as a narrow slit, becoming still narrower on inspiration (Fig. 23). Inability to abduct the paralyzed vocal cord (Fig. 24).
	Paralysis of the internal thyro-arytenoids.	In catarrhs of the mucous membrane of the larynx. After over-exertion of the voice. In hysteria.	Voice hoarse; speaking an effort.	Glottis does not close completely on phonation (Fig. 25). If at the same time the arytenoids are paralyzed, the glottis presents an hour-glass outline (Fig. 26). Neither anterior nor posterior portion is closed, but the vocal processes are in their normal position.
	Adductor paralysis (paralysis of the lateral crico-arytenoids).	Rarely isolated. In hysteria.	Absolute absence of voice. Power of coughing retained. "Phonic paralysis" (Türck).	Nothing characteristic.
SUPERIOR LARYNGEAL.	Paralysis of the crico-thyroids.	After diphtheria.	Voice rough; high tones impossible.	Excavation of the vocal cords. Cords do not vibrate visibly.
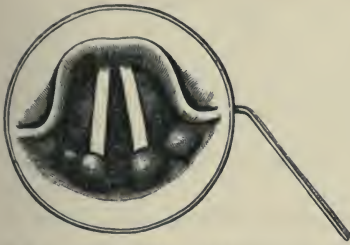
Fig. 20.—BILATERAL PARALYSIS OF THE RECURRENT LARYNGEAL. "Cadaveric position" of the vocal cords.
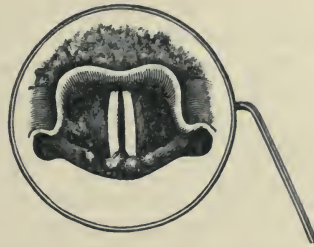
Fig. 21.—RECURRENT LARYNGEAL PARALYSIS. Overriding of the arytenoid cartilages.

Fig. 22.—PARALYSIS OF THE RECURRENT LARYNGEAL ON THE LEFT SIDE (in inspiration).
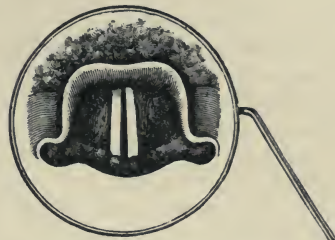
Fig. 23.—PARALYSIS OF BOTH POSTERIOR CRICO-ARYTENOIDS (in inspiration).
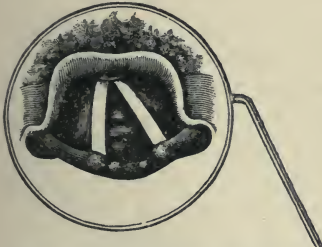
Fig. 24.—PARALYSIS OF THE RIGHT POST. CRICO-ARYTENOID (in inspiration).

Fig. 25.—PARALYSIS OF BOTH INTERNAL THYRO-ARYTENOIDS (acute laryngitis).

Fig. 26.—PARALYSIS OF BOTH INTERNAL THYRO-ARYTENOIDS, associated with paresis of the arytenoid muscle.

Figs. 20–26.—Partly after STRÜMPELL, partly after EICHHORST.

Rosenbach, O.   Ueber einen Fall von doppelseitiger Stimmbandlähmung.
    Breslauer ärztl. Zeitschr., i, 2, 3, 1880.
Hayes.   Dubl. Journal, 3 S., lxix, p. 34, January, 1880.
Rosenbach, O.   Monatsschrift f. Ohrenheilk., etc., No. 3, 1882.
Schech.   Zur Ætiol. der Kehlkopflähmungen.   Ibid., No. 8, 1883.
Dehio.   Petersburger med. Wochenschr., 22, 1883.
Aysaguer.   L'Union, 46, 1885.   (Bilateral Abductor Paralysis during Preg-
    nancy.   Two Tracheotomies.   Recovery.)
Bäumler.   Deutsches Arch. f. klin. Med., Bd. xxxvii, Heft 4, 5, 1885.   (Recur-
    rens Paralysis after Inhalation of Dust.)
Gerhardt.   Stimmbandlähmung und Icterus.   Deutsche med. Wochenschr., No.
    16, 1887.
Wegener.   Ueber Kehlkopfmuskellähmung als Symptom der Tabes.   Inaug.
    Dissert.   Berlin, 1887.
Fränkel, Bernh.   Ueber die Beschäftigungsschwäche der Stimme, Mogiphonie.
    Deutsche med Wochenschr., 1887.
Holmes, Gordon.   Paralysis of the Abductors of the Vocal Bands.   Lancet, Oc-
    tober 22, 1887.
Kidd.   Bilateral Paralysis of the Dilator Muscles of the Glottis, with subsequent
    Paresis of the Constrictors.   Lancet, July 16, 1887, p. 108.
Küssner.   Zur Kenntniss der Vagus-Symptome bei der Tabes dorsal.   Berliner
    klin. Wochenschr., No. 20, 1887.
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    Vorträge, iv, Leipzig, Vogel, 1887.
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    und Nervenkr., xix, 2, 314, 1888.

2. The lungs receive from the thoracic portion of the vagus the pulmonary or bronchial nerves, the so-called anterior branches of which, in conjunction with filaments of the sympathetic, form a plexus on the anterior wall of the bronchus, and enter with the latter the lungs, while the posterior branches, together with those coming from the four upper thoracic ganglia of the sympathetic, are distributed in the same way on the posterior surface of the bronchus. They are the motor nerves for the unstriped muscles of the bronchial tree.

The diseases of the bronchial nerves, which produce, as it seems, a faulty innervation of the circular muscles of the bronchi, give rise to the morbid condition which has lately been the subject of much controversy, and is described under the name

Bronchial Asthma, Asthma Bronchiale s. Convulsivum s. Nervo-
sum, Spasmus Bronchialis (Romberg).

Pathology.—Opinions about the nature of bronchial asthma are still divided. While some (Störk, Fräntzel) maintain that it is due to an acute swelling of the bronchial mucous mem-

brane, others (Bamberger, Wintrich) consider a tonic spasm of
the diaphragm to be responsible for it ; still others (Trousseau,
Biermer) believe it to be a vagus neurosis, supposing, in conse-
quence of a disturbed innervation (vagus), a tonic spasm to take
place in the circular muscles of the medium-sized and fine
bronchi, thus producing an acute pulmonary emphysema.
After Bert had shown, in 1870, by experiment that a contrac-
tion of the medium-sized and finer bronchi could actually be
produced by irritating the vagus, later Biermer worked out
his theory so thoroughly, and has defended it so successfully,
that we are probably justified in accepting it as correct, espe-
cially as with its help all the characteristic symptoms, the
sudden onset and the sudden disappearance of the attacks, the
expiratory dyspnœa, the low position of the diaphragm, etc.,
can well be explained. It is clear that this bronchial spasm
forms an impediment much more easily overcome by inspira-
tion than by expiration, and that this difficulty in expiration
must of necessity not only influence the alveoli, but also the
smaller bronchi, from which the inspired air can only imper-
fectly be forced out ; hence arises dyspnœa and emphysema
during expiration. On auscultation, sibilant rhonchi are heard
all over the chest. But all this does not explain the cause of
the spasm. This may be sought for in an independent affection
of the bronchial mucous membrane, a view which possibly may
be supported by the presence in the sputa of asthmatics of the
so-called " Curschmann's spirals " (spiral threads which must be
looked upon as casts of the finest bronchioles). Or we may
assume a reflex origin. Thus Leyden maintained that certain
pointed octahedral crystals which he discovered in the sputa
of asthmatics irritated the mucous membrane, and thus pro-
duced the spasm. Many observations, however, allow us to
doubt the correctness of this latter view. It has been estab-
lished, on the other hand, beyond doubt (Voltolini, Hack, Som-
merbrodt) that certain diseases of the nasal mucous membrane
(polypous growths, chronic catarrh, etc.) may give rise to asth-
matic attacks—reflex neurosis; possibly some part in the pro-
duction of these is played by the reflex dilatation of the vessels
in the bronchial mucous membrane, which was by Störk and
Weber supposed to take place in connection with the bron-
chial spasm, a theory which was afterward confirmed by Som-
merbrodt.

Symptoms.—The characteristic features of the disease are

the paroxysms of distress and dyspnœa, previous to which the patient may for days complain of general malaise, be low-spirited, and troubled with digestive disturbances, diarrhœa, etc. The attacks begin quite suddenly, usually at night, more rarely in the day-time; during them the respiration is changed, so that the breathing in inspiration, but more especially in expiration, becomes labored and accompanied by a loud wheezing. This may last only a few hours or may continue for days, and may be repeated at varying intervals. Toward the end of the attack moist râles can be heard on ascultation, and there is expectoration which contains the above-mentioned spirals and crystals. Between the attacks the patient enjoys perfect comfort.

Ætiology.—The ætiology of the disease is but little known. No doubt hereditary predisposition does exist, and persons with a neuropathic family history fall, *cæteris paribus*, more easy victims to asthma than others. Just of what nature the exciting causes of the actual outbreak are we are as yet unable to say. We have repeatedly observed that hysterical persons suffer from asthmatic conditions, which on examination of the respiratory organs, prove to be of a nervous origin. In these instances the patients are for days troubled with paroxysmal dyspnœa, their expiration is difficult and wheezing, while nothing abnormal is found on ascultation and percussion. We shall later on have more to say about this hysterical asthma.

That the inhalation of certain kinds of dust may give rise to asthma, while not a frequent, is certainly a well-authenticated observation. We may especially find this connection when the same obnoxious causes have been acting frequently and through a rather prolonged period of time, as is the case in those who follow certain occupations (millers, bakers, etc.); in the same way it is well known that repeatedly druggists have been affected regularly with asthmatic attacks while occupied with the pulverization of ipecacuanha root, and that the dust of certain kinds of grain—for instance, of oats—causes such disturbances in those engaged in thrashing (cf. Hirt, Krankheiten der Arbeiter, 1871, Bd. i, p. 12).

The asthma which develops under the influence of certain poisons has to be classed among these cases, and in this connection the so-called lead asthma (asthma saturninum) is deserving of special mention. This is a very peculiar disease, which sometimes sets in very acutely only a few minutes after the work has been taken up. Though to the highest degree dis-

tressing to the patient, a fatal outcome in it has never been noted (cf. Hirt, *op. cit.*, Bd. iii, p. 40). This trouble is, however, even among lead-workers, quite rare, so that we may assume that among one hundred affections due to working in lead two instances at most of this above-described asthma occur. As to the mode of origin, we do not know whether to refer it to the action of the poison on the central nervous system, or on the peripheral nerve-endings of the vagus.

Treatment.—We are not acquainted with any specific for bronchial asthma; the much-recommended iodide of potassium (2.0–5.0 (xxx to lxxv grs.) a day) often fails, and, as a rule, we do not accomplish much with the usual nervines, arsenic, quinine, bromide, etc. From the use of electricity we have never seen any lasting benefit. Well-conducted hydrotherapeutic measures may produce a decided decrease in the frequency and the severity of the attacks. For the treatment of the attack itself we can foremost recommend pyridin, which was suggested by Sée. It is a product obtained in the dry distillation of organic substances, a colorless fluid which easily evaporates in the air. For the therapeutic use half a teaspoonful of it has to be poured on a shallow dish, and this inhaled three to four times daily in a closed room. The smell is horrible, and often disgusting, but in many instances the action was found extremely beneficial. As soon as the pyridin evaporates the patient becomes easier, the feelings of distress are relieved, the heart's action is more regular. The effect is not always lasting; still, I have seen cases in which daily regular inhalations used for several weeks have not only cut short the individual attacks, but have also decreased their frequency. Of course, with this, as with all other remedies, we may be disappointed. From the inhalation of the fumes of burning saltpetre paper, which has recently again been recommended by Kochs, I have only seen transient, never any lasting effects. The same holds for the well-known stramonium cigarettes, for amyl nitrite, and the vapors of turpentine. More good may be expected from the administration of tinct. lobeliæ, which often works like a charm (tinct. lobel., 5.0 (℔ lxxv); aquæ laurocer., 15.0 (℥ iv). Sig.: 15 to 20 drops every two hours). With the extract of quebracho, which has been recommended by Penzoldt, I have no large experience of my own. Hyoscyamine, together with small doses of strychnine given several times a day, has been used by Walker (Lancet, August 20, 1887, p. 368).

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B. Cardiac Affections due to Lesions of the Vagus.

The superior and inferior cardiac branches are given off from the cervical and thoracic portions of the vagus; they join the cardiac branches of the sympathetic to form the cardiac plexus (superficial and deep). It has not yet been determined of what character these fibres are; there is, however, no doubt but that we have to distinguish inhibitory fibres, the stimulation of which diminishes, and accelerator fibres, the stimulation of which increases the number of heart beats. The sensory nerves of the heart are also furnished by the vagus.

Angina Pectoris.

Among the neuroses of the heart which probably are caused by a disturbance in the vagus, we shall first consider angina pectoris (stenocardia, cardiac neuralgia, nervous heart pain), a disease of the true nature of which our knowledge is as yet quite imperfect, though its symptoms have been recognized for more than one hundred years (Heberden, 1772). Its cardinal symptom is a piercing, burning, paroxysmal pain in the region of the left nipple, attended with a sensation of impending death; it often radiates into the left arm, and even down to the finger tips, and may continue for minutes or hours. It usually begins without any premonition and surprises the patient by day at his work, or wakes him up at night out of his sleep. The severity of the pain differs; in some cases it is moderate, in others it reaches an insupportable degree. Dyspnœa is not always present; the respiration remains sometimes regular and quiet, although the patient suffers from a distressing feeling of anxiety, and his skin is covered with a cold sweat. During the intervals, the patient feels perfectly well, unless there is a co-existing lesion of the heart muscle or valves.

The prognosis depends mainly upon the question whether we have to deal with a vagus neurosis, or whether some complication co-exists. If the myocardium, owing to disturbed intracardial circulation (caused, for instance, by atheroma of the coronary arteries), has undergone pathological changes, death may occur during an attack, but this has never to be feared if the heart be otherwise sound. It is impossible to give an absolutely favorable prognosis with regard to recovery, because here also we do not possess any remedy which is capable of doing away with the attacks entirely. But the same suggestions as have been made for the treatment of bronchial asthma have been made for angina pectoris, and about the same results have been obtained in both. If internal treatment can not be dispensed with, digitalis may in the first place be tried, then strophanthus, and finally arsenic, which latter may with advantage be combined with strychnine.

For the attacks freshly prepared amyl nitrite, a few drops (5 to 10), to be carefully inhaled by the patient, is the most useful treatment; besides this, inhalations of chloroform and the hypodermic injections of morphine deserve recommendation, as they relieve the patient at once from the intolerable

torments of his condition. The severe states of collapse following these measures, observed by Bamberger, are probably, after all, quite exceptional.

The ætiology of the disease is as obscure as its nature: here we must again carefully discriminate between the cases where the angina pectoris is merely a symptom of some organic heart disease (disease of the coronary arteries, fatty heart, valvular disease), and where it appears as an independent affection—i. e., where no heart lesion can be demonstrated. The latter form is disproportionately less frequent (Gauthier). Males and those advanced in age seem especially predisposed to the disease (Gauthier); yet the author has repeatedly treated cases of undoubted angina pectoris in children thirteen to fifteen years of age. Psychical disturbances, such as are found in hysterical patients, also the influence of certain poisons— e. g., tobacco—deserve some consideration. That angina pectoris is a vagus neurosis can reasonably be accepted, as the sensory fibres of the heart are furnished by the vagus, and as pain is the most prominent symptom of the trouble. Presumably the sympathetic is, however, also concerned, and some are even inclined with Lancereaux, who several times found this nerve vascularized, to regard the cardiac plexus of the sympathetic as the chief seat of the disease; but even were this so, we could not exclude some participation of the vagus. A publication of Leroux, who found at the autopsy a bronchial gland and the right vagus grown together in a case where anginal seizures had existed until just before death, appears also to speak in favor of an implication of the latter nerve. Frequently no anatomical lesion was found.

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Nervous Palpitation of the Heart.

Secondly, we have to speak of the so-called nervous palpitation of the heart, palpitatio s. hyperkinesis cordis, by which term we designate a paroxysmal increase in the frequency and strength of the heart's action, which is not only objectively noticeable, but also subjectively felt by the patient. Pain is absent, and in pure cases at least there is no dyspnœa. Palpitation appears more frequently as an independent affection than angina; the attacks usually begin suddenly, often at night. If the patient be lying on his left side, he is seized with a feeling of oppression and anxiety, the pulse is accelerated, and its rate may be increased to more than two hundred beats to the minute; sometimes the second heart sound is curiously clicking (cliquetis métallique) and the first extraordinarily weak, the carotids throb, the radial pulse becomes hard and full. Dehio (cf. lit.) has examined the pulse curves by means of a Dudgeon sphygmograph, and found the pulse waves higher, the decline steeper, the first elastic elevation decidedly nearer to the base line of the curve, and the dicrotic elevation lower than normal. He attributes this condition to an increase in the frequency of the beats, and a decrease in the duration of the individual ventricular contraction. Besides the palpitation, the patient complains of ringing in the ears, dizziness, and faintness. The attacks usually pass off in a few minutes, disappearing as suddenly as they came on, and the patient soon feels perfectly well. Their frequency is extremely variable; they may appear once, twice, or more often daily, or only after long intervals of weeks or months.

That here we also have to deal with a neurosis of the vagus seems only a rational assumption. The seat varies; it may be either central or peripheral, but in most cases we are unable to positively say which it is. Sometimes we are justified in assuming that such conditions depend upon a central, bulbar nuclear affection, just as we may probably refer a temporary diminution of the vascular tonus to a transient paresis of the vaso-motor centre in the medulla oblongata (Dehio).

It is very important in these cases of palpitation to look for further co-existing affections, after the removal of which the nervous palpitation often disappears suddenly, and never recurs. To this class belong chiefly the anæmias of the young, cardiogmus juvenilis, habitual constipation, gout, and malaria,

and accordingly we are able to bring about a marked improvement in the palpitation, which in such cases is only symptomatic, by improving the condition of the blood, by proper regulation of the bowels, by promotion of the excretion of uric acid, and by combating malaria by means of quinine, according to the indications in each. If such indications for therapeutic measures are wanting, we have to fall back upon the narcotics and nervines, unreliable as they are in their action. In hysterical persons certain mechanical manipulations, pressure on the abdomen, momentary compression on the neck, and the like, may be of service. Application of the ice-bag to the cardiac region may occasionally be beneficial; the psychical treatment of the patients, repeated assurances that these attacks are never fatal, and that they are quite amenable to treatment, is not to be underrated; in the case of children especially this has been found very effectual.

The ætiology is, unless the palpitation is secondary to an underlying disease, quite obscure. Under what conditions individuals in other respects quite sound, with a good family history, and who present no symptoms of neurasthenia, can be attacked by such transient pareses of the vagus we do not know. In suspicious cases we should think of masturbation.

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Tachycardia.

In rare cases, in people otherwise healthy, but more frequently in those affected with heart disease, we meet with a transient acceleration of the heart's action (tachycardia), which usually lasts for several hours, after which the pulse rate again becomes normal. These attacks are accompanied by a feeling

of great anxiety, and are ushered in by vaso-motor disturbances —e. g., circumscribed flushings.. The number of the pulse beats may reach 150 or more. Pressure upon the vagus in the neck, a draught of cold water, or similar stimulation of the peripheral ends of the vagus often may cut short an attack against which we possess no other remedy. Whether in a given case irritation of the accelerators or a paroxysmal paralysis of the vagus is responsible for the attacks has, according to Nothnagel (Wiener med. Blätter, i, 2, 3, 1887), to be decided in the following way : A great increase in the frequency of the pulse, accompanied by a weak heart-beat, and, perchance, another disturbance of some nerve path belonging to the vagus, speak for paralysis of this nerve ; whereas a strong impulse, fullness of the peripheral arteries, with high tension, associated with other symptoms of vaso-motor irritation, is in favor of stimulation of the accelerators. Traube assumes that some cases are due to a temporary anæmia in the medulla oblongata, in consequence of which a paresis of the inhibitory nerves ensues. To this class seems to belong the case related by Dehio (cf. lit.). The mode in which nicotine acts upon the vagus is of great interest, and certainly deserves a closer study than has been given to it hitherto.

Chronic nicotine poisoning, as it is found in smokers, and only occasionally in tobacco workers, is not always well adapted to throw much light on this subject, for, whereas it is well known that the nicotine when brought into direct contact with the nerves paralyzes them rapidly, it is by no means common to find paralysis of the vagus in the course of nicotine intoxication. As a rule, it is true that the heart's action is increased, yet cases occur in which there is a slowing, so that we are led to think of a stimulation of the vagus, such as happens after drinking cold water, where the pulse rate may be reduced to thirty or twenty beats. Owing to the miserable arrangements for ventilation in tobacco factories, we have from time to time occasion to study the action of nicotine in those employed in them, although the disease is, as has been said, by no means frequent.

Cases which, in consequence of a vagus neurosis, present a simultaneous disturbance in the circulatory and respiratory apparatus, occur, but are rather uncommon. A case to the point has been published by Tuczek (Deutsches Arch. f. klin.

Med., 1877, xxi, 1), and two others by Kredel (ibid., 1882, xxx, p. 547). For the respiratory apparatus acute emphysema, with dyspnœa and symptoms of catarrh, were noted; they were associated with tachycardia (asthma cardiacum, according to Kredel), and the existence of a paralysis of the vagus fibres regulating the heart, in conjunction with a stimulation of those presiding over the lungs, whereby spasm of the muscles of the bronchi was produced, was assumed. At the autopsy the cause was found to be pressure exerted upon the vagus trunk by a rapidly swelling lymph gland. The attacks lasted from twelve to thirty-six hours. Some of the patients had organic heart disease.

<div align="center">LITERATURE.</div>

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C. The Disturbances of the Digestive Organs due to Lesions of the Vagus.

The vagus forms two gastric plexuses; the one—the anterior—situated on the anterior surface, and the other—the posterior—situated on the posterior surface of the smaller curvature of the stomach. The first plexus is formed by the left, the second by the right, a somewhat stouter nerve. The branches of these plexuses associate with fibres from the sympathetic which accompany the ramifications of the coronary arteries; a part of the fibres which appertain to the right (posterior) vagus go on to the cœliac plexus, and can in a careful dissection be traced to the spleen, the liver, the kidneys, and small intestine.

The muscles of the œsophagus and stomach are also innervated by the vagus; its sensory fibres conduct the impulses concerned in the reflex actions of deglutition, sobbing, and vomiting.

One of the digestive disturbances attributable to an affection of the vagus, the so-called gastroxynsis, has been mentioned in the consideration of migraine; another one, which

occurs paroxysmally—namely, the gastric crisis (Charcot), which depends presumably upon an affection of the nucleus of the vagus—will be discussed in the description of tabes. It remains, therefore, to speak of three groups of symptoms, two of which are quite frequently met with, constituting in themselves separate diseases—namely, cardialgia and nervous dyspepsia—while the third one, the so-called œsophagismus, is more frequently observed as a symptom of other neuroses, especially hysteria.

Cardialgia.

Cardialgia (gastralgia, gastrodynia) is a disease of the sensory nerves which occurs mostly in paroxysms. Romberg, distinguishing two forms, assumed the one to be due to a hyperæsthesia of the vagus branches going to the stomach (" gastrodynia neuralgica "), the other to a hyperæsthesia of the solar plexus (neuralgia cœliaca). There have been, however, cases coming under notice which can not be classed under either of these heads, and even more which do not permit of a decision as to which of the two forms we are dealing with.

The characteristic symptoms of gastrodynia are violent paroxysmal constricting pains, starting in the region of the stomach and radiating to the back; the face becomes livid, hands and feet cold, the pulse smaller and intermittent, and a feeling of unutterable anguish and distress takes possession of the patient. If in the presence of these symptoms careful examination has excluded the existence of any organic stomach lesion—e. g., acute or chronic catarrh, gastric ulcer or tumor —if there is no evidence of gall stones, and the patient has previously at times been subject to neuralgia in other parts of his body, we make our diagnosis with some amount of certainty. But in all cases this can only be done after careful and repeated examination before and after meals; not uncommonly we find that pain, which is present while the stomach is empty, is relieved by the ingestion of food, and the patient states that uniform firm pressure on the epigastrium has often a beneficial alleviating effect, both conditions not generally observed in organic diseases of the stomach.

In the treatment of these cases we must first of all endeavor to remove any primary cause, and in this connection mental and physical overstrain, excesses in venery, masturbation, uterine affections, must be thought of. Besides the ex-

ternal application of blisters to the epigastrium, arsenic given for several weeks is to be recommended. During the attack morphine can often not be dispensed with. The diet has to be carefully regulated, but not restricted ; on the contrary, it is advisable for the patient to take four or five times daily substantial but easily digestible food.

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Nervous Dyspepsia.

The disease known as nervous dyspepsia is an extremely common neurosis of the vagus, especially in females. It is characterized by a loss of appetite, painful sensations in the region of the stomach, frequent vomiting, and still more frequent belching ; besides these the patients generally suffer from other nervous symptoms—dull headache, vertigo, palpitation—they are easily tired, complain of a lump in their throat (globus hystericus), at times have a voracious appetite, and obstinate constipation is seldom absent. In rare cases periodical spells of vomiting have been noted (twenty to thirty in the twenty-four hours), accompanied by acute circumscribed swellings of the skin (angio-neurotic œdema, Strübing, Quincke). Although the patients feel very poorly, their state of nutrition remains, nevertheless, for a long time remarkably good ; only in a few cases do we observe a rapidly increasing and marked anæmia. It is still doubtful whether the condition is essentially a disease of the peripheral nerves of the stomach or a general neurosis (neurasthenia dyspeptica, Ewald). We would refer the reader to a most interesting and comprehensive article which has been written on this subject by Leube (Berl. klin. Wochenschr., No. 21, 1884).

In making our diagnosis we are brought face to face with no inconsiderable difficulties. The claim of Leube that we are, in the presence of the above-described symptoms, justified in thinking of nervous dyspepsia if a stomach-washing six to seven hours after the meal shows the stomach to be empty, has been opposed by Ewald and others. These have shown that, on the one hand, the stomach may be empty seven hours after a meal in cases of ulcer, and, on the other hand, may contain remains of food in nervous dyspepsia after the same

time. To be sure, an increase of hydrochloric acid (hyperacidity) is a common condition in gastric ulcer. The results of stomach-washing are, however, certainly not always pathognomonic, but we must rather for the purpose of diagnosis take into account the course of the disease and the general condition of the patient. But in spite of the greatest care experienced men not seldom in these cases are led into error. Under certain circumstances the hyperemesis nervosa, a motor neurosis of the stomach occurring in pregnant women, especially in the first months of pregnancy, may closely simulate this disease.

In the treatment our attention has chiefly to be directed to the proper nutrition of the patient. Of medicines, arsenic, quinine, chloral (1.0 (grs. xv) several times a day), should be resorted to. Saline purgatives, Carlsbad water, as well as the use of electricity, are of no avail. A stay in the mountains, hydrotherapy, sea-baths, all should be tried in succession, and last, but not least, the possibilities of psychical treatment must not be forgotten.

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Œsophagismus.

Spasmodic dysphagia, known as œsophagismus (spasm of the gullet), is an affection which sometimes follows dyspeptic symptoms and protracted vomiting, sometimes irritation of the fauces by hot food, irritating substances (mushrooms, red pepper, etc.). Sometimes the spasm is seen to occur reflexly in consequence of uterine diseases, and quite frequently in hysteria. As an independent affection it is rarely ever observed. In all cases it is characterized by the fact that the patient from time to time (periodically) experiences great difficulty, or is even unable, to swallow his food, that when it reaches a certain point it is regurgitated, and that the sound which is intro-

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duced for the purpose of examination is stopped at the same place; if this point is situated in the upper portion of the œsophagus, usually violent pain is experienced on the ingestion, more especially of cold food, a circumstance which makes the patient object to taking his nourishment, and consequently leads to emaciation, although the loss of flesh is here much less considerable than in stenosis of the œsophagus caused by new growths, because in the former case the patient is able at times to swallow his food without any difficulty.

Predisposed to œsophagismus are nervous, easily excitable, hysterical persons in whom the affection often suddenly makes its appearance after some emotion without the previous existence of any symptoms referable to the œsophagus.   It has often followed the suppression of the menses, or has appeared during pregnancy and lactation.   Sometimes no other ætiological factor could be discovered than injuries to the gullet years previous to the spasm—burns, injury by sulphuric acid, etc.   No definite statement is warranted as to the duration and the course of the disease, as both vary greatly, but this much may be said with certainty, that in pure cases the prognosis is always good, that complete recovery is almost always effected by the repeated use of the sound and by the application of the faradic brush.

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D. Graves' Disease—Basedow's Disease (*Glotzaugenkrank-heit, Cachexie exophthalmique*)—Exophthalmic Goitre—Tachycardia strumosa exophthalmica.

Symptoms.—The three symptoms which are regarded as characteristic of Graves' disease are (1) an excited, accelerated action of the heart, with visible pulsation in the arteries of the neck; (2) enlargement of the thyroid gland; (3) exophthalmos. As a rule the heart symptoms are the first to appear. The increase in the frequency of the pulse is variable. We may count from a hundred to a hundred and fifty beats a minute, and not infrequently the intensity of the heart beat is more forcible than normal, a circumstance which adds much to the discomfort of the patient. Auscultation does not always reveal abnormalities. Occasionally a systolic souffle is audible, but this is often absent. Enlargement of the heart also has been observed. The extraordinarily strong pulsation in the carotids, which is very conspicuous and easily felt, is in remarkable contrast to the smallness of the pulse wave in the radial artery (Parry).

The swelling of the thyroid is rarely very great. It is usually symmetrical. In the gland itself pulsation can be easily seen, and on palpation a distinct thrill is communicated to the hand. I may say that I have repeatedly seen cases in which the volume of the gland changed from time to time, and that this change became perceptible in a comparatively short time, sometimes even in a few hours.

The exophthalmos, which is probably always bilateral, also differs in degree in different cases. In the majority, however, it is so marked that the protruding eyeballs can not be completely covered by the lids during sleep. This gives to the patient an appearance which to the layman is both peculiar and repulsive, and is still more aggravated if the upper eyelid does not follow the downward motion of the ball, and thus allows a zone of the sclerotic, 1 to 2 mm. in width, to become visible above the cornea. This defective co-operation of lid and ball (Graefe's symptom), which happily does not occur very often, makes the patient almost an object of horror to those who surround him. The almost complete absence of the involuntary winking of the lid (Stellwag's symptom) is quite conspicuous, especially since the voluntary movements can be made as well as before. We can then easily understand that

our patients, particularly when they are ladies of the better classes, avoid as far as possible the contact with friends and acquaintances, as well as with strangers.

Ophthalmoscopically only one characteristic sign has been noted—namely, the spontaneous pulsation of the retinal vessels, discovered by O. Becker. This is not confined to the disk, but can be observed in the retina as well. With this exception there are no changes in the fundus and eye-sight, accommodation, and pupils are entirely normal. Only on the cornea we occasionally find a decrease in sensibility, probably due to the want of moisture on the ball, the normal quantity of the lachrymal fluid not being sufficient on account of the undue evaporation which takes place, because the two lids are far apart, and winking only rarely occurs.

Insufficiency of convergence, a symptom first described by Möbius, is sometimes observed. If the patient be asked to look at a near point, one eye will soon be found to deviate outward.

Among the subjective symptoms, in addition to the annoying palpitation already mentioned, a tendency to free perspiration may be noted. Even slight exertion produces a feeling of heat, more especially in the head and neck, so that the patient preferably remains in cool, shady places, and sleeps with as little covering as possible, etc. An actual elevation of temperature is, however, not always objectively demonstrable. This tendency also accounts for the blushing evoked by the least bodily exertion or mental emotion. Both symptoms I have not infrequently seen to occur unilaterally. Trousseau has mentioned the fact that the most gentle stimulation of the skin of the face and neck produces a deep-red mark, designated by him as *tâche cérébrale*, a phenomenon, however, which can apparently not always be evoked. All these symptoms are attributable to asthenia of the vaso-motor nerves, as is also the decrease in the resistance which the skin offers to the electrical current, first observed by Charcot, the saturation of the skin with fluid resulting from the dilatation of its capillaries rendering it a better conductor than it would naturally be in the dry state. In a healthy individual using an electro-motive force of from ten to fifteen volts the resistance amounts to from four to five thousand ohms, while in the course of this disease it only measures from three to six hundred ohms, and only increases when the patient improves.

Various nervous disturbances often accompany Graves' disease, among which the copious vomiting of a watery bile must be considered the most important. It may be of such persistency as to seriously reduce the strength of the patient. Vertigo, buzzing in the ears, sleeplessness, occasional transient dyspnœa, have also been observed. Falling out of the hair of the head and eyebrows is not rare, and I have seen a case of a peasant woman, thirty-eight years old, who, toward the end of the disease, when she was extremely emaciated owing to the persistent diarrhœa and vomiting, had become completely bald.

Complications with chorea, epilepsy, and diabetes mellitus are said to occur. Oppenheim has described a case in which it accompanied Addison's disease (Neurologisches Centralblatt, i, p. 29, 1888).

Course.—We should keep in mind that remissions may occur during the course of the disease; nay, that it often may remain stationary for months or years before further deterioration takes place which leads to death. For our prognosis a knowledge of the fact that such remissions can occur is of great importance. Cases which pursue a rapid course from the very beginning are exceptional.

The onset of the disease may be either brusque or quite gradual. In the first case twelve to fourteen hours are sufficient time for the development of the three cardinal symptoms; in the latter these appear gradually—first the palpitation, then the swelling of the neck, and finally the protrusion of the eyeballs.

Of great interest, because relatively frequently met with, are the cases in which the disease does not reach its full development; only certain symptoms are well marked, while others may be hardly perceptible or even absent. Trousseau calls these instances "*formes frustes*" (*fruste*—abortive), and attributes much importance to them. P. Marie has subjected them to a careful study in his excellent monograph (cf. lit.), and has shown that the goitre as well as the exophthalmos may be wanting, in which case we shall only find the tachycardia, very often accompanied by a symptom to which he has given special attention—namely, the tremor. This tremor shows a great regularity of rhythm, and consists of about eight or nine oscillations in a second. Ernst Cramer, in his observations made in my wards, was generally able to confirm Marie's results. (Ueber das Wesen des Zitterns. Inaugural Dissertation. Breslau, 1886.)

Prognosis.—The prognosis seems to be less favorable in the male sex and in advanced age than in female patients, the latter always having a better chance, especially if pregnancy interrupts the disease (Charcot).

Ætiology.—Ætiologically the affection is obscure. Pelvic diseases, cold, emotions, hereditary tendencies, have been thought of in this as in other neuroses, but their causative action does not seem even probable.

Pathological Anatomy.—What has been found on the autopsy table does not give us any key to an understanding of the disease, for all that has been observed in the cervical sympathetic is very inconstant, and although an enlargement and thickening of its ganglia has several times been met with, we are not justified in drawing any conclusions from such alterations, especially as there are cases published in which the examination of the sympathetic gave entirely negative results. The changes in the heart and thyroid were equally unsatisfactory. Only one thing more I should like to mention, namely, that often immediately after death the exophthalmos is markedly smaller than during life, or at times entirely disappears.

Pathogenesis.—To explain, then, the pathogenesis of the disease is difficult. Certainly, however, the explanation of Köbens, afterward defended by Trousseau, Fletscher, Reith, Eulenburg, Guttmann, and others, that the disease was essentially one of the sympathetic, is, to say the least, doubtful. Without taking into consideration the fact that we should in this case be obliged to assume a constant state of irritation of certain groups of fibres in the sympathetic, the goitre and exophthalmos can hardly be explained by lesions in this nerve. For these reasons Sattler proposed to assume a circumscribed lesion in the region of the vagus centre, by which the inhibitory action on the heart is diminished or suspended. He does not consider that the absence of other vagus symptoms can be held to disprove this, as this inhibitory action actually can be suspended and the other functions of the nerve remain intact. Filehne, who cut the restiform body in its anterior quarter, and thus produced symptoms of Graves' disease, is also inclined to make a lesion of the pneumogastric responsible for at least some of the symptoms. Goitre and exophthalmos are, according to him, due to a vasomotor paralysis. Broadly the whole disease is, in his opinion, of a paralytic nature.

All attempts to fix the anatomical seat of the disease have

this disadvantage in common, that they only explain a part of the symptoms. It seems therefore, in view of our defective knowledge, more reasonable to collect and analyze more observations before adding a new hypothesis to those already existing.

Sattler attributes Graefe's symptom to a lesion of a co-ordinative centre which governs the co-operation of the levator and orbicularis palpebrarum on the one hand, and of the muscles which rotate the eye round its horizontal axis on the other. Stellwag's symptom he explains by a lesion in the centre which communicates reflexes, starting from the retina and the sensory fibres of the conjunctiva and cornea to the motor apparatus of the lids.

Treatment.—The treatment in most instances is not attended with much success. In so many cases, which received no treatment whatever, improvement has been noticed, that our therapeutic measures, although at times giving apparently good results, are not too much to be relied upon. As a rule, internal medicines are the least efficient. Iron and quinine are those generally in use, but little is accomplished by them. The same must be said of the iodide and the bromide of potassium. Opiates and ergot have also been tried, but without result. More may be expected from hydrotherapy, whether it be in the form of Priesnitz's cold packs or of cold douches, which latter should be used for a considerable time. Patients of the better classes should remain several months of the year in properly conducted hydrotherapeutic establishments, as most of them will generally feel more comfortable there than at home. The best success, after all, is obtained, according to my own observations, from systematic galvanic treatment. The cathode is applied to the angle of the lower jaw, while the anode is held at the lower cervical vertebræ (on the opposite side). The current should be weak, and only applied for a short time, from a minute to a minute and a half. Not infrequently after the tenth or fifteenth *séance*, not generally until after the twentieth or thirty-fifth, we are able to observe a steadily progressing improvement which may last for years (Erb, Benedict, Guttmann, Mor. Meyer, and others). Whether this is due to the action on the sympathetic or on the vagus can not be decided, as both nerves are influenced by the electricity.

That some cases of Graves' disease have a peripheral origin

is shown by the communications of Hack (cf. lit.), who repeatedly succeeded in removing some symptoms of the disease—for instance, the exophthalmos—by destruction of the swollen erectile tissue of the nose by the galvanic cautery. On the operated side the eye at once went back to its normal size, a fact which should impress us with the necessity of always making a rhinoscopic examination.

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CHAPTER IX.

THE DISEASES OF THE ACCESSORY NERVE.

THE accessorius consists of two parts, both of which have a separate origin and exit. The upper one belongs entirely to the vagus, emerges with it, and is hence called accessorius vagi. The lower one begins at the level of the first cervical nerve (cf. Fig. 27), and can be traced as far down as the level of the sixth, sometimes even of the
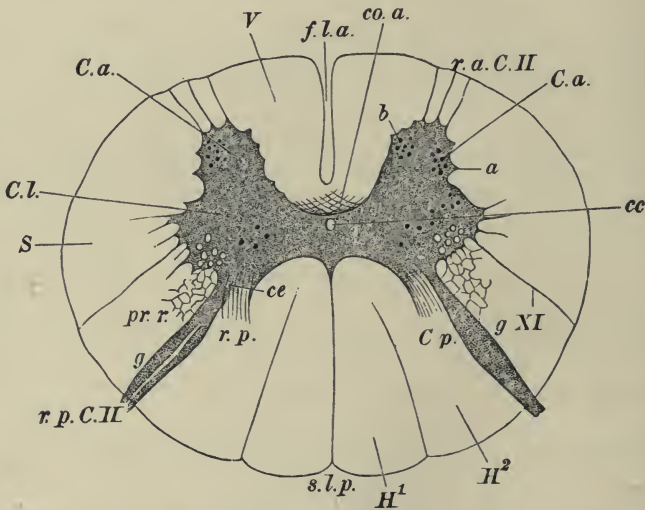
Fig. 27.—CROSS-SECTION THROUGH THE CERVICAL CORD. *r. p. C. II*, posterior root of the second cervical nerve. *XI*, fibres of accessorius. *C. a.*, anterior horn. *C. p.*, posterior horn. *C. l.*, lateral horn. *H¹*, Goll's column. *H²*, Burdach's column. *S*, lateral column. *V*, anterior column. *g*, substantia gelatinosa of posterior horn.

seventh cervical nerve roots; this is the spinal portion, the accessorius spinalis. After having passed upward to the foramen magnum, close to the cord, it unites inside the skull with the portion belonging to the vagus to form for a while a common trunk, the accessorius communis, which, soon after leaving the skull through the jugular foramen, again divides into two branches, the accessorius vagi then becoming the inner, the accessorius spinalis the outer branch.

The nucleus of the former portion has been described in the two previous chapters; that of the spinal portion is in the region of the anterior horn of the cervical cord. Since the latter is provided with motor ganglionic cells, the purely motor nature of the spinal portion is evident (Schwalbe). According to Dees (Allgem. Zeitschr. f. Psychiatrie von Laehr, Bd. 43, Heft 45, 1887), the nucleus of the accessorius is divided into three portions, the upper being situated in the centre of the anterior horn, just above the first cervical nerve; the middle at the lateral border of the anterior horn from the second to the fourth cervical nerve; and the lowest at the base of the lateral horn from the fourth to the sixth cervical nerve. The large multipolar (motor) ganglionic cells which form the nucleus are arranged like a rosary.

We may have central as well as peripheral diseases of the accessorius, and, as is the case in other motor cranial nerves, the diseases may be of a paralytic or of an irritative nature (hyperkinesis, spasm—akinesis, paralysis).

Accessorius Spasm, Spasmodic Wryneck, Spasmodic Torticollis (*Tic Rotatoire, Nickkrampf*).

Since the accessorius supplies the sterno-cleido-mastoid and the trapezius (with its posterior larger portion), it is these two muscles which present disturbances in affections of the nerve. Either of them may be affected by itself, by a clonic or a tonic form of spasm; hence there exist quite a variety of clinical pictures, especially as the disease may also be unilateral or bilateral. The sterno-cleido-mastoid is about as often the seat of a clonic spasm as the trapezius, whereas the tonic form is very rarely seen in the latter muscle.

By the rhythmical contractions of one sterno-cleido-mastoid the head is moved to one side in a very characteristic manner; the chin is turned toward the opposite (well) side and is elevated, while the ear is approached to the clavicle. Contracture of this muscle (the tonic spasm) fixes the head in this position—caput obstipum spasticum. If both sterno-cleido-mastoids are affected, the head is drawn alternately first to the one, then to the other side (clonic form), or it is pulled strongly forward and bent toward the chest (tonic form of the spasm).

Contractions of the trapezius draw the head backward and toward the diseased side, elevate the shoulder, and approach the scapula to the vertebral column. A tonic spasm in the same locality entails fixation of the head in this position.

A simultaneous spasm of the sterno-cleido-mastoid and the trapezius of the same side, in which the facial muscles also sometimes take part, is known to occur more frequently than an affection of both trapezii or of both sterno-cleido-mastoids alone. The directions of the movements and the positions which result from such spasms can be made out from what has been just said.

The occurrence of such affections is either in paroxysms or else we have permanent contractions, only ceasing or abating during sleep. Recovery is exceptional. All therapeutic measures, not excluding the electrical and chirurgo-orthopædic treatment, are usually unsuccessful. The thermo-cautery may be tried. Any internal medication would have to be conducted according to the principles described in the treatment of facial spasm.

The causes of the disease differ widely. Cerebral tumors, meningitis, foci of softening, as well as caries of the cervical vertebræ, new growths in the medulla oblongata, may give rise to central, while external influences, cold, etc., may give rise to peripheral affections of the nerve. There are, moreover, well-authenticated cases on record of reflex spasm in the distribution of the accessorius arising from irritation by worms, uterine trouble, fright, and other emotions. As a rule, no ætiological factor can be detected. An epileptic who came under my observation, a single woman, twenty-seven years of age, presented at times a spasmodic torticollis, the contractions being extremely violent, sometimes lasting for weeks, and again being almost entirely absent for the same length of time.

ACCESSORIUS PARALYSIS.

This very rare disease may take in one or both of the above-named muscles. Unilateral paralysis of the sterno-cleido-mastoid produces a wry position of the head, in which the chin is somewhat elevated and directed toward the diseased side. Turning of the head is difficult but not impossible, as other muscles are brought into play. Bilateral paralysis of the muscle causes the head to be held straight, and is characterized by the absence of the prominence which the normal muscle produces.

Unilateral paralysis of the trapezius allows the scapula to sink downward, causing the distance between its internal margin and the vertebral column to become greater. As a consequence, the arm falls forward, the clavicle becomes more

prominent, the supraclavicular fossa more marked, and the posterior upper angle of the scapula can be distinctly felt. Voluntary elevation of the shoulder and the motion of the scapula toward the spinal column is interfered with, and becomes only possible with the aid of the levator anguli scapulæ and the rhomboidei. The arm can not well be raised above the horizontal position in spite of the serratus which acts normally, because the action of the upper third of the trapezius is lost. The paralysis of both trapezii allows both shoulders to sink outward and forward, so that the back appears more curved ; the ability to support the head in the upright posture is sometimes interfered with. Simultaneous paralysis of the sterno-cleido-mastoids and the trapezii gives a combination of both clinical pictures. If, in addition, the inner (smaller) portion of the nerve takes part, the resulting paralysis of the laryngeal muscles, the velum palati, and the pharyngeal muscles manifests itself by hoarseness, the nasal tone of the voice, and difficulty in swallowing. The increase in the frequency of the pulse, which has in such cases been observed by Seeligmüller, must be attributed to the simultaneous affection of the cardiac branches of the vagus. Prognosis and treatment are the same as in the spasmodic affections, and little more can be said about the ætiology. Yet we should add here that these forms of paralysis have been known to occur in consequence of certain occupations (water-carriers, whose occupation obliges them to carry buckets suspended from their shoulders, and who are exposed to wettings, are apt to suffer—Seeligmüller), and in the course of tabes it has been seen as a bulbar affection. We may also imagine an injury to the nuclei of the nerve during difficult labor, and in symptoms of paralysis in the muscles of the neck in new-born children, who for the first few years of life are unable to hold the head straight, such a possibility ought not to be forgotten (Gowers).

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CHAPTER X.

THE ten to fifteen bundles of fibres of which the hypoglossal nerve consists, as it emerges from the medulla oblongata in the groove between the anterior pyramid and the olivary body, unite to form two larger bundles, which leave the dural space separately, and, after their entrance into the hypoglossal canal, the anterior condyloid foramen, become a single stem, which leaves the cranial cavity by this canal. Outside the base of the skull it passes along to the mesial side of the vagus, at first obliquely downward and forward, then obliquely upward, runs on the outer surface of the hyoglossus muscle, and soon reaches the region where the genioglossus muscle radiates into the tongue. There are various communications between the hypoglossus, the vagus, the anterior branches of the upper cervical nerves, and the lingual branch of the trigeminus. The superficial origin of the hypoglossal nerve and of the other cranial nerves and their points of exit from the brain-substance and the base of the skull are represented in Figs. 28 and 29.

The cortical area of the hypoglossal nerve is found, according to Exner, in the lower portion of the anterior central convolution and the adjoining portion of the inferior frontal convolution, as shown in Fig. 30. Its nucleus is situated in the floor of the fourth ventricle (Fig. 37), where its very large ganglionic cells, which measure up to 60 μ in diameter, closely resemble the large multipolar ganglionic cells of the anterior horn in the cord.

The root fibres of the hypoglossus (Fig. 31, 12) certainly arise in part from the nucleus of the same side. To what extent the nucleus of the opposite side, as well as the group of ganglionic cells situated in its neighborhood and the above-mentioned nucleus ambiguus (Fig. 31, Xa), can be considered sources of origin for them, and, moreover, whether direct fibres of the hypoglossus have their origin in the cerebrum, is still undecided.

While in certain of the cranial nerves—for instance, in the facial—peripheral affections occur at least as frequently as cen-

tral, in the case of the hypoglossal this is not true. Often as its nuclei take part in the most diverse diseases, especially of the cord and medulla oblongata, it is rare that a peripheral affection comes under observation. That, in a given case, the
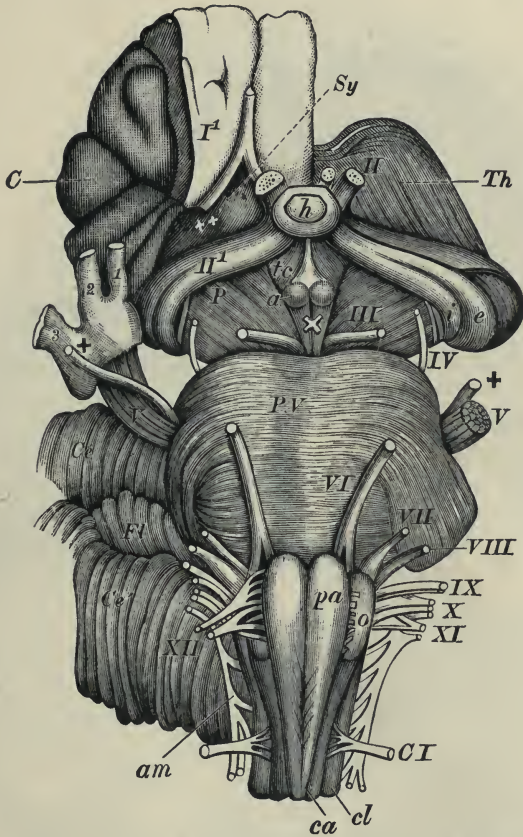
Fig. 28.—Superficial Origin of the Cranial Nerves. *I–XII*, the twelve cranial nerves. *CI*, anterior root of the first cervical nerve. *ca*, anterior column of the spinal cord. *cl*, lateral column. *pa*, anterior pyramids. *o*, olivary body. *P.V.*, pons Varolii. *i*, internal. geniculate body. *e*, lateral geniculate body. *tc*, tuber ciner. *h*, pituitary body. *P*, cerebral peduncle. *Sy*, region of the fissure of Sylvius. *a*, corpora albicantia. *C*, island of Reil. *Th*, optic thalamus.

disturbance is peripheral and not central, more especially not bulbar, we may conclude from the absence of other symptoms of bulbar disease, and from the possible presence of complete reaction of degeneration, as Erb (cf. lit.) has done in his recent-ly described case. The symptoms otherwise are the same as in the central disease.

Central paralysis of the hypoglossus may be, in the first place, of cortical origin. According to Exner, as has been stated, the cortical area for the tongue is situated at about a point where the middle and inferior frontal convolutions join
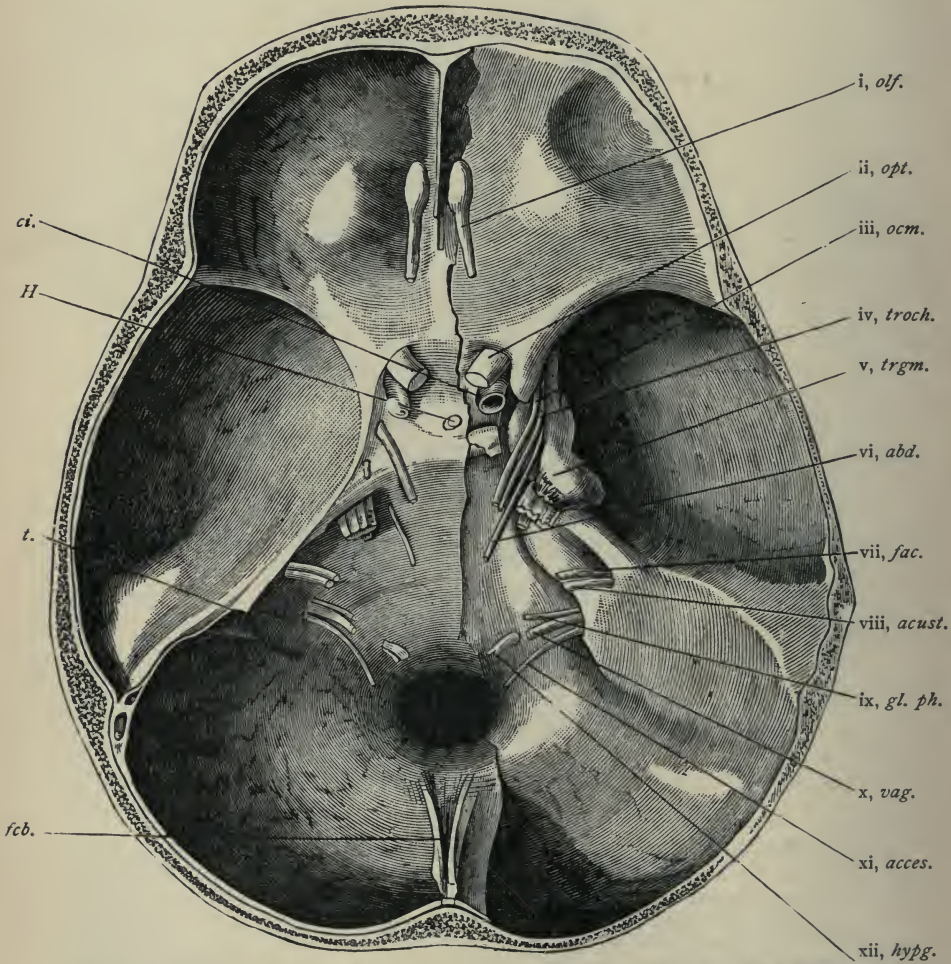
Fig. 29.—Base of the Skull with the Emerging Cranial Nerves. (After Henle.)
ci, internal carotid. *H*, pituitary body. *t*, part of tentorium. *fcb.*, falx cerebelli.

the anterior central convolution, and it is very probable that injury to this region causes a motor disturbance in the tongue. *A priori*, no objection can be made to this conclusion; *a posteriori*, it has not yet been confirmed, as proofs from autopsies are still wanting. It is impossible, therefore, to make even a

surmise with regard to the relative frequency (or rather infrequency) of the affection.

The bulbar lesion of the nerve or rather its nucleus is some-
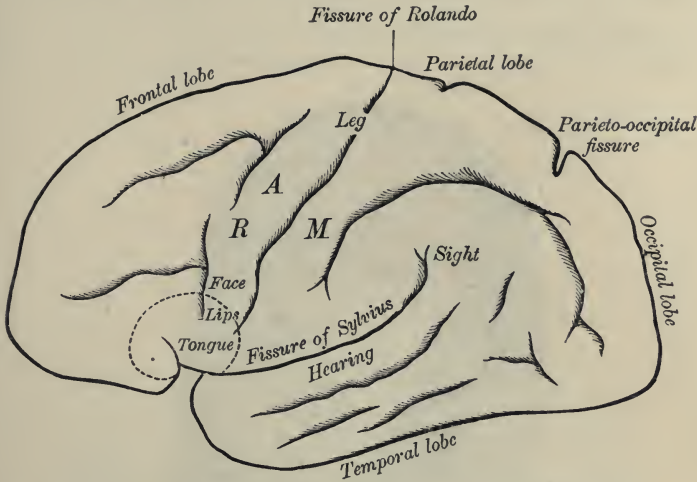
Fig. 30.—CORTICAL CENTRES OF THE LEFT HEMISPHERE. (After GOWERS.)

what better understood; it has undoubtedly been observed, if not frequently, at least repeatedly, that this lesion can occur
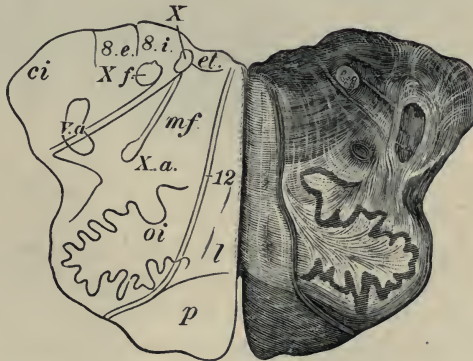
Fig. 31.—CROSS-SECTION THROUGH THE MEDULLA OBLONGATA. (After WERNICKE.) 12, root-fibres of the hypoglossal nerve. *X. a.*, nucleus ambiguus (anterior vagus nucleus). *X*, posterior vagus nucleus. *X. f.*, common root of the "mixed lateral system." 8. *e.*, external, 8. *i.*, internal auditory nucleus. *ci*, restiform body. *oi*, olive. *V. a.*, ascending root of the trigeminus. *p.*, pyramid.

unilaterally. There is then an atrophy of the nucleus, in which the ganglionic cells and the medullated fibres become

10

decreased in number or disappear entirely, while the roots appear as fine threads. In such cases (see especially Fig. 32) the tongue is protruded, not straight, but deviates toward one side, and be it remembered toward the affected side (m. genioglossus and geniohyoideus); it shows fibrillary twitchings, and an atrophy of the diseased side—hemiatrophia linguæ—which

Fig. 32.—HEMIATROPHIA LINGUÆ (personal observation).

in such a case looks flabby and shrunken in comparison with the full and firm healthy half; it is wrinkled, contracted, and much smaller than the latter (cf. Figs. 32 and 33, showing my two cases). The electrical examination shows either normal reaction or reaction of degeneration; that the latter may also occur in central lesions has been demonstrated by one of my cases, which, however, did not come to autopsy. Speech, mastication, and deglutition often suffer considerably; on the other hand, the healthy half of the tongue may develop so satisfactory and vicarious an activity that little disturbance is observable.

Unilateral paralysis of the hypoglossal nerve has been observed as a result of traumatism ; further, also, in diseases of the vertebral artery, as the result of new growths in the medulla oblongata and in cases of embolic softening in the region of the nucleus (Hirt). Whether it can be also of saturnine origin seems to me to be doubtful, in spite of the report of

Fig. 33.—HEMIATROPHIA LINGUÆ (personal observation).

Remak. In a recently published article by Koch and Marie (cf. lit.) may be found all the cases observed up to the present time collected and minutely analyzed.

In bilateral paralysis of the hypoglossal the tongue, atrophic, wrinkled, and shrunken, lies almost motionless on the floor of the mouth ; the patient can not protrude it, and has entirely lost control over it. Speaking and chewing are rendered difficult, even quite impossible. This sad picture is seen not infrequently in Duchenne's progressive bulbar paralysis, occasionally in progressive muscular atrophy, very rarely in tabes. The hemiatrophy of the tongue, too, occurs much less frequently in the course of tabes than, to judge from the communications—

for instance, those of Ballet (cf. lit.)—would seem to be the case.

The peripheral form of the affection may yield to electrical treatment (faradization and galvanization); the central, as far as we know at present, is not amenable to any treatment.

Hypoglossal spasm occurs sometimes unilaterally, somewhat more frequently bilaterally. It is an exceedingly rare affection, in regard to which there have been but few good publications. There is a paroxysmal, involuntary spasm of the tongue, by which it is protruded and retracted, rolled violently around in the mouth, and so roughly pressed against the teeth that it may be quite severely injured. In some instances there occur short rhythmical twitchings in the whole tongue which disappear at times. Berger observed an aura before such an attack, which consisted in a sensation of tension and swelling of the tongue. In Dochmann's case the attacks occurred especially at night, and were so violent that the patient, a woman, was waked up by the sudden spasmodic protrusion of the tongue. In one of my own cases the muscles of mastication took part in the affection in such a way that before the actual hypoglossal spasm occurred, the lower jaw was for a half or a whole minute spasmodically jerked to and fro, up and down. After these movements had ceased the mouth remained half open, and the turning and rolling movements of the tongue commenced and lasted for about one minute. These attacks recurred ten to twenty times a day; they came on for the first time three days after an epileptic fit, and have lasted unaltered ever since (for three years). The patient is otherwise perfectly healthy, and has a good family history. The pathogenesis of the disease, its anatomical seat (irritation of the hypoglossus centre? cortical or bulbar?), is obscure. As an accompanying symptom of chorea and hysteria it is by far more common than as an independent affection. The treatment is the same here as in paralysis of the tongue.

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CHAPTER XI.

AFTER having thus considered the lesions of the individual
cranial nerves, it remains for us to inquire under what condi-
tions several of them may be simultaneously affected, and into
the symptoms thus produced. According to the observations
collected up to the present time, an affection of this kind may
have its seat in the peripheral or in the central course of the
nerves, as well as in the cortical or nuclear centres. Only cer-
tain of the affections of this latter kind are to be regarded as
independent diseases, while the peripheral lesions are always
only partial manifestations of other diseases. In rare cases
a simultaneous peripheral lesion of several cranial nerves may
occur in consequence of traumatism, operative interferences,
etc. A case in point, in a patient operated upon by Israel, has
been published by Remak (Berl. klin. Wochenschr., 7, 1888). A
cavernoma of the neck was extirpated, and by the operation
the accessorius, the hypoglossus, and the sympatheticus were
injured, or rather resected. The symptoms caused by the acci-
dent were accurately described by Remak. Other instructive
cases, due to traumatism, have been described by Möbius (cf. lit.).

Among the general diseases in which multiple cranial nerve
lesions may occur are chiefly tuberculosis and syphilis.

Tubercular meningitis attacks, by preference, the mem-
branes at the base, and implicates most of the cranial nerves
emerging in that region, as we have seen in our account of the
diseases of the meninges. Lately Kahler (cf. lit.) has again
directed attention to the fact that, in consequence of syphilis,
a peripheral neuritis of the cranial nerves sometimes develops,
and that we may, besides general cerebral symptoms, have a
progressive slow paralysis, which attacks one cranial nerve
after the other in irregular succession.

After diphtheria peculiar forms of paralysis are observed, which chiefly take in the muscles of the soft palate and the pharynx. Since these muscles are innervated by certain of the cranial nerves, and the disease is unquestionably—e. g., when the paralysis is unilateral—often of peripheral origin (central diseases can not in all other cases be excluded), we shall devote a few lines to the consideration of their nerve supply.

The innervation of the palatal and pharyngeal muscles is by no means one of the clearest chapters in neurology. We do not know exactly which of the cranial nerves are concerned, nor their mode of distribution. Of the palatal muscles the levator palati is the most important. This receives motor fibres through the large superficial petrosal (of the trigeminus) from the spheno-palatine ganglion, which come from the facial and which also innervate the azygos uvulæ. Whether or not, however, the vago-accessorius and the glosso-pharyngeus are also concerned in the innervation of these muscles, as Gowers, for instance, seems to think, basing his arguments upon clinical observations, is not as yet decided (cf. page 85). With regard to the pharyngeal muscles, it is generally assumed that the stylo-pharyngeus and the middle constrictor are supplied by the glosso-pharyngeal nerve, and that the palato-pharyngeus, the superior and inferior constrictors, are innervated by the vagus. The participation of the accessorius is doubtful (Schwalbe). We see then that the nerves concerned in a paralysis of the pharynx are the facial, the glosso-pharyngeal, the vagus, possibly also the fifth and the accessorius.

Pharyngeal paralysis may be either unilateral or bilateral. The unilateral form can only be diagnosticated if the patient is made to move the soft palate, for instance, in saying "Ah!" While during rest it appears to be perfectly symmetrical, the base of the uvula deviates somewhat on motion towards the affected side, so that on that side a little way from the median line there is a slight depression not present on the well side; sometimes also the soft palate is a little lower on the paralyzed side even during rest. In the bilateral complete paralysis of the soft palate, the latter hangs down flaccidly and the uvula appears elongated; on deep respiration and on phonation it remains motionless, and the reflex movements evoked by tickling the mucous membrane are lost. Speech becomes markedly altered, the voice acquires a nasal tone, due, of course, to the cavity of the nose not being shut off during phonation; hence also the pronunciation of the explosive consonants "P" and "B" becomes impossible, owing to the imperfect com-

pression of the air; they sound like " M." Closure of the anterior nares removes, as Duchenne has shown, this disability. From the same cause also fluids are regurgitated through the nose on attempts at swallowing, and deglutition in general becomes difficult.

The prognosis in post-diphtheritic paralysis is not unfavorable if the velum palati alone is paralyzed. If, on the other hand, the muscles of the œsophagus also take part, the outlook becomes graver on account of the inability of the patient to take nourishment, and all the more so if feeding by the stomach-tube is not constantly and carefully practiced. If this is not done, aspiration pneumonia or inanition may bring about a fatal issue.

The electrical treatment ought to be begun as early as possible. It consists in the direct faradization or galvanization of the velum and the frequent excitation of reflex movements of deglutition by stimulation of the throat. The uvula, the pillars of the pharynx, etc., are directly touched and repeatedly stimulated by means of the curved button electrode (cf. Fig. 34). The movements of deglutition are obtained if the anode is placed on the neck and the cathode (button electrode) is quickly drawn over one of the lateral surfaces of the larynx, six to ten cells sufficing for the purpose. These gymnastics of the pharyngeal muscles constitute an excellent remedy which can not be replaced by any other. It often leads rapidly to recovery.

Fig. 34.—PHARYNGEAL AND LARYNGEAL ELECTRODE WITH ARRANGEMENT FOR MAKING AND BREAKING THE CURRENT. (After ERB.)

Central diseases of several cranial nerves at the same time may also occur, and that, too, not only in their intracerebral course—which for but few of them is known, and for those only imperfectly—but also in the centres themselves. As a

matter of fact, our knowledge about the centres situated in the cortex is also very incomplete, since we must again confess our comparative ignorance of the anatomy. Still, we shall not go too far if we assume that extensive cortical lesions may implicate several centres together, and there is no doubt but that they may be affected after or rather during an apoplectic attack by " indirect action."

Of eminently practical importance are the nuclear affections of the cranial nerves. Referring the reader to the preceding chapters for the anatomical position of the individual nuclei, we will only remind him of the fact that these nuclei are situated in the gray matter, partly of the mid- and 'tween brain, partly in the medulla oblongata. The portion situated above the latter extends from the posterior wall of the infundibulum in the third ventricle to the level of the nucleus of the abducens, and embraces the nuclei of the eye muscles (Wernicke). The other nuclei belong to the lower portion.

Clinical observations now teach us that either of these portions may be affected by itself, and we may with Wernicke call the disease of the upper, polioencephalitis superior ; that of the lower, polioencephalitis inferior. According to the course, we distinguish in either case an acute and a chronic form, so that there are altogether four clinical pictures of these nuclear affections.

Polioencephalitis superior acuta has only been observed in very few instances. The best observations we owe to Wernicke. According to him, this is essentially an acute inflammatory disease of the nuclei of the ocular muscles, and proves fatal in from ten days to a fortnight, the focal symptoms consisting in an associated paralysis of the eye muscles, the general symptoms being grave disturbances of consciousness. The walk presents a peculiar combination of spasm and ataxia. Anatomically, foci of acute softening are found in the region of the nuclei, which are either due to obstruction of the blood-vessels or to inflammatory infiltration of the tissues. Ætiologically, the abuse of alcohol may be mentioned.

Polioencephalitis inferior acuta (acute bulbar paralysis, acute bulbar myelitis) is also an extremely rare affection. It is equally rapidly fatal after the quick development of all those symptoms which we shall describe shortly in our account of chronic bulbar paralysis. Microscopically, inflammatory changes are found in the lower nuclear region.

Polioencephalitis superior chronica was described in 1868 by von Graefe, and called by him ophthalmoplegia progressiva. The first published case presented, according to von Graefe (Berl. klin. Wochenschr., 11, 1868), a peculiar clinical picture:

"Gradually all the muscles concerned in the movements of the eye become paralyzed, causing first a diminution in the range of sight, and finally resulting in complete immobility of the eyeballs. The levator palpebræ superioris is wont to be implicated, although the consequent ptosis is rarely as marked as that occurring in complete oculo-motor paralysis. It is remarkable that, on examination for reaction to light and accommodation, the sphincter pupillæ as well as the ciliary muscle present no changes. This condition, which we very rarely find in other extensive oculo-motor paralyses, seems here constant and characteristic of this disease. Another feature which distinguishes this form from other associated paralyses in the distribution of the third, fourth, and sixth nerves is the progress of the disease *pari passu* in the antagonizing muscles. Thus we never find a marked strabismus divergens owing to a dominating oculo-motor paralysis, because here the external rectus loses its functions sufficiently to neutralize the tendency to deviation, and the sight of the patient is therefore, in spite of the associated paralysis of the eye muscles, affected much less than in simple oculo-motor or abducens paralysis. . . . Still, a certain degree of asymmetry in the affection of the different muscles of one eye, as well as in the development of the whole disease in the two eyes, may at times be found." (Cf. Wernicke, *loc. cit.*, vol. iii, page 463.)

With the exception of this associated ocular palsy, which, developing progressively, may remain stationary without being completely symmetrical, the patient enjoys good health and complains neither of headache nor of symptoms of increased intracranial pressure. In isolated instances bulbar paralysis has been known to be later superadded, and in others the disease was complicated by multiple sclerosis. Anatomically, the affection depends either upon a primary disease of the nuclei of the ganglionic cells or upon a diffuse sclerotic process in which the nuclei of the nerves take part. In exceptional cases, which in their nature are as yet entirely obscure, no organic changes whatever have been found, although the clinical picture corresponded exactly to that described by von Graefe. (Eisenlohr and Oppenheim.)

Not less interesting, and at the same time of by far greater practical importance because relatively far more frequently met with, is the fourth and last of the affections under consideration—a disease the first accurate description of which we owe to Duchenne, of Boulogne, and which after him has been carefully and successfully studied by German investigators (Wachsmuth, Kussmaul, Leyden)—the chronic progressive bulbar paralysis.

PROGRESSIVE BULBAR PARALYSIS.

Paralysis of the Tongue, the Soft Palate, and the Lips (Duchenne, 1860), Glosso-labial Laryngeal Paralysis (Trousseau), Chronic Progressive Bulbar Paralysis (Wachsmuth, 1864), Atrophic Bulbar Paralysis (Leyden), Bulbar Nuclear Paralysis (Kussmaul), Polioencephalitis Inferior Chronica (Wernicke).

Duchenne's Disease.

Symptoms and Course.—In the majority of instances the onset of progressive bulbar paralysis is very gradual, and only rarely do we meet with cases in which it is ushered in by an apoplectiform attack. After having complained for weeks, perhaps months, of drawing, tearing pains in the neck and the back, the patient discovers of his own accord or from the remarks of his relatives that the enunciation of certain words, especially those containing *l*, *r*, and long *e*, has become very difficult. If he happen to use a word containing all these letters (for instance, reel), he becomes painfully conscious of his indistinct enunciation. In vain he attempts to repeat the troublesome words over and over again in order to correct his mistake. He only becomes more convinced that the movements of his tongue have become clumsy, and that he has lost his former ease and fluency of speech; and, in truth, it is the oncoming paresis of the lingual muscles which is the main cause of the disturbance.

The tongue, which can not be raised to the normal extent, can no longer be approached sufficiently to the hard palate, and thus the long *e*, for the pronunciation of which the movement is necessary, can only be pronounced with difficulty. In the same way all the finer muscular movements required for the formation of the linguals are imperfect, and consequently the enunciation of these sounds is bad. The disease progresses and the articulation becomes worse and worse, the less perfectly the lingual muscles are innervated, and other letters, *s*, *t*, *g*, finally also *d* and *n*, begin to suffer, so that conversation

with the patient becomes very uncomfortable, as certain words are almost unintelligible and others at least difficult to understand.

The lips also begin to do their duty badly, so that the enunciation of the so-called labials—*o, u, a, b, p*—gradually becomes indistinct.   The presence of strangers with whom he has to converse excites the patient, and, avoiding all society, he prefers the quiet monotony of the family circle, where nobody seems to pay much attention to the change in his speech ("alalia and anarthria"). Moreover, a change in the features of his face, at first slight and only noticed by the patient himself, but later more perceptible and evident also to his friends, gradually manifests itself, which serves as an additional reason for

Fig. 35.—FACIAL EXPRESSION IN PROGRESSIVE BULBAR PARALYSIS. (After LEYDEN, EICHHORST.)

seclusion (Fig. 35). When laughing, it appears to him as if a certain tension in his lips prevented the usual play of the mouth.   In the attempt to whistle the lips can not be puckered as well as formerly ; the muscles of the cheek have become more rigid and inactive, and as the disease progresses the whole lower half of the face assumes a characteristic appearance—a peculiar lachrymose and astonished expression—which, as is easily seen, is due to the drooping of the lower lip and to

the deepening of the naso-labial fold. The upper half of the face, the forehead and eyes, do not take part in the change, but remain entirely normal. Nevertheless the patient's face is much disfigured, and later on in the disease may have become almost unrecognizable.

While thus quite gradually symptoms have arisen which make the patient a very pitiable object, and which are bound sooner or later to interfere with his position in society, the sad truth dawns upon him that even the functions absolutely necessary for the existence of life are failing. Eating, in which up to this time no trouble was experienced, he now finds difficult. It takes a longer time to swallow the food, and in a later stage even mastication becomes impaired. Not only do the movements of the lower jaw become weaker and less energetic, owing to paresis of the muscles of mastication, but, since the powerless tongue is unable to get the food from between the cheeks and gums into the region of the pharyngeal muscles, the formation of the bolus is impossible. Spoons, fingers, and the like, have to be used instead, or the patient has to hold his head far back to get the food to slide down. Even drinking causes much discomfort, as the liquid may get into the larynx and thus give rise to violent coughing, or may be regurgitated through the nose, either condition being due to weakness of the pharyngeal and laryngeal muscles.

The implication of the larynx is very distressing, and may indeed become dangerous. The voice at times fails, speech becomes irksome, and the tone is monotonous; production of the higher notes as in singing becomes impossible; later on a marked hoarseness and finally aphonia follows, so that the patient can only express himself in whispers, which, owing to the above-described motor changes, are quite unintelligible. At the same time the absence of a firm closure of the glottis, and therefore the inability to cough forcibly, gives rise to various disturbances in the respiratory apparatus, owing to the disability to dislodge mucoid masses which may have collected in the air passages.

Another symptom which, though not constant, is frequently met with is the marked increase in the secretion of the saliva. This occurs usually rather early in the disease, and not infrequently such patients are seen going around constantly holding their handkerchiefs to the mouth to prevent the saliva from trickling away. On examination, the secretion is found to be

viscid. This flow of saliva is due to an actual increase in the amount secreted, as several careful investigators have shown, though they do not agree as to the exact amount.

Two, three, even five years may pass before any new symptoms are added to those just described. These, however, progressively gain in intensity, and it is especially the change in the features which becomes more accentuated, owing to the constantly increasing atrophy in the muscles of the lips and the cheeks; the palatal reflexes become markedly decreased and finally lost; the tongue, shrunken and distinctly smaller, lies immobile on the floor of the mouth, and can neither be protruded nor moved in any direction. Fibrillary tremor is then not uncommonly marked. On the electrical examination (which is, by the way, very hard to make), we may find reaction of degeneration in the lingual as well as in the pharyngeal muscles (cf. page 146).

The inability to take food properly is usually the cause of death; the patient pines away, and gradually dies from inanition without having the blissful benefit of a dulled conscious-
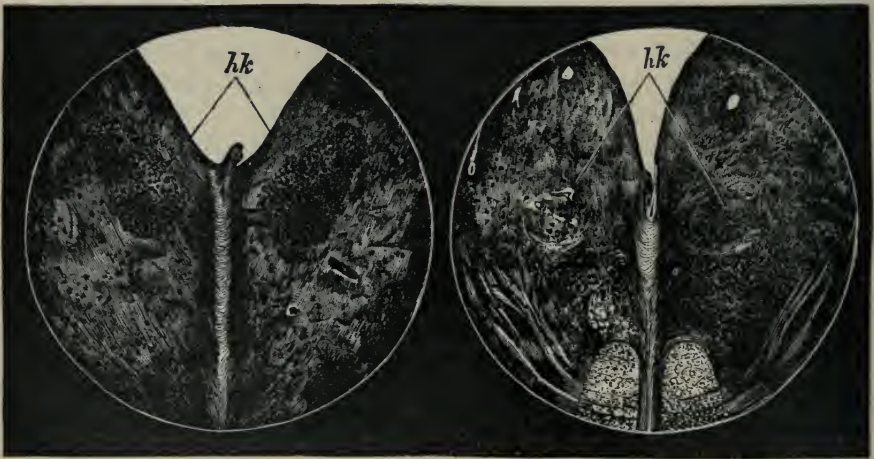
Fig. 36.—CROSS-SECTION THROUGH THE UPPER PORTION OF THE MEDULLA OBLON-GATA. On the left the healthy, on the right the diseased medulla. *hk* on the left the normal, *hk* on the right the diseased hypoglossus nucleus (the ganglionic cells are almost entirely absent on the right side).

ness to guide him insensibly through his tormenting sufferings. Only in occasional instances disease of the respiratory organs, caused by aspiration of food, hastens the termination (aspiration pneumonia).

Pathological Anatomy.—There is hardly another disease of the nervous system with the anatomical basis of which we are better acquainted than bulbar paralysis. Duchenne pronounced the process to be a primary pigmentary degeneration, and atrophy of the large ganglionic cells in the nuclei of the medulla oblongata, an assertion which has received complete confirmation from all subsequent investigators. Microscopical examination shows atrophy of the ganglionic cells. This is shown in Fig. 36 in the nucleus of the hypoglossal; the cells have in this case completely disappeared, having previously diminished in size and lost their processes. At the same time we find the connective tissue increased, the walls of the vessels in the nucleus thickened. Similar changes are found in the nucleus of the vagus accessory and the glosso-pharyngeal nerves (the so-called lateral mixed system, cf. page 95). Since, as we have said before, the upper part of the face always remains normal during the disease, we have to assume that the fibres innervating these muscles arise from a special centre. This is supposed to be a part of the abductor nucleus (Meynert), which has therefore come to be designated by the composite name of facial-abducens nucleus. (For the position of the nuclei,

Fig. 37.—The Posterior (Dorsal) Aspect of the Medulla Oblongata. 1, posterior commissure. 2, peduncle of pineal gland. 3, corpora quadrigemina. 4, superior peduncle. 5, middle peduncle. 6, inferior peduncle of cerebellum. 7, striæ acusticæ. 8, funic. teres. 9, obex. 10, funicul. gracil.

cf. pages 42 and 77.) This and the remaining nuclei, with the exception of those mentioned above, were always found to be intact. The atrophy also extends to the root fibres, which, to the naked eye, often appear smaller and of a grayish color. From the topographical position of the nuclei below the floor of the fourth ventricle, as it is approximately represented in Fig. 37, we can easily understand how, on the one hand, the

pathological process, after having attacked the hypoglossus, next implicates the neighboring vagus, and, on the other hand, how the motor part of the trigeminus usually remains unaffected, so that paralysis of the muscles of mastication is very rare. But why the auditory is constantly exempt and the facial partially affected are circumstances which need to be further investigated. A complete counterpart to bulbar paralysis is found in the so-called progressive muscular atrophy, a disease in which, as we shall see later on, the gray anterior horns of the spinal cord and their ganglionic cells are affected precisely in the same way as the bulbar nuclei in the disease we are now discussing. The ganglionic cells of the anterior horns constitute the trophokinetic centres for the muscles supplied by the spinal nerves, an office which the bulbar nuclei fulfill for those supplied by the cranial nerves. In both diseases there is atrophy and decrease in the functional power, and in both the disturbance is strictly motor, while sensation is absolutely intact. This essential similarity between the two diseases explains why not rarely one is associated with the other—in other words, why they may complicate each other. We may, indeed we frequently do, meet with cases in which bulbar paralysis is accompanied by atrophy of the muscles of the extremities, while, on the other hand, in progressive muscular atrophy, bulbar symptoms, disturbances in deglutition and speech, may be found.

Another analogy exists between bulbar paralysis and amyotrophic lateral sclerosis, a disease in which not only the ganglionic cells of the anterior gray horns, but also the motor tract in the lateral columns of the spinal cord is affected. All these diseases, viewed from an anatomical standpoint, if not identical, certainly are closely related to each other, and only differ in the position of the lesions; it is, therefore, advisable to consider and study them from a common point of view, as the understanding of the individual symptoms will thus be much less difficult.

Diagnosis.—As to the diagnosis, we need not be doubtful if we always remember that the disturbances are confined to the motor functions of the nerves governing the muscles of the lips, tongue, pharynx, and larynx, to which disturbances all the symptoms described above may be attributed. If we find any well-marked sensory changes, if the patient complains of pain or paræsthesias and the like, we either have to give up the

diagnosis of bulbar paralysis, or we have to search for some complication. The peculiar facial expression, the increased flow of saliva, the tremulous atrophic tongue partially or even completely immobile as it is, the disturbance in speech and deglutition, when taken together are so characteristic that, if intelligently observed and studied, they will make our diagnosis clear.

There is only one case in which we may be doubtful; certain foci of disease in the brain may produce symptoms simulating bulbar paralysis, so much so indeed that the name pseudo-bulbar paralysis has been given to the condition (which later on will be described more at length); nevertheless, with due carefulness we can avoid a mistake. The most important point to observe in the differential diagnosis is the course of the disease. While in progressive chronic bulbar paralysis this is slow, but always progressive toward the fatal end, in the spurious form remissions may occur, so that for years the patient may be improved, though he finally also succumbs to the disease. Besides this, pseudo-bulbar paralysis is often attended with cerebral symptoms, headache, apoplectiform attacks, etc. More about this will be found under the head of pathological diagnosis in the chapter on encephalo-malacia.

Prognosis.—The prognosis, as we should expect after what has been said, is altogether unfavorable. There is, according to our present knowledge, no cure for the true bulbar paralysis, and one ought to be careful, therefore, not to deceive the family with promises. As soon as the diagnosis is made they ought to be informed of the unfavorable outlook.

Treatment.—The only treatment from which any success may be expected, if begun early, is the systematic use of electricity: faradization and galvanization of the threatened muscles, especially of those of the tongue and pharynx, frequent excitation of the movements of deglutition, according to the method described on pages 151 and 152, are the only measures which deserve confidence. With the exception of this local treatment, there is nothing that affords even a temporary benefit. I have never seen any lasting effect from hydrotherapy, but still this treatment is very frequently advised just at that stage of the disease when electricity might do some good. Internal remedies are of no avail; the occasional symptomatic use of atropine ($\frac{1}{2}$ to 1 milligramme ($\frac{1}{120}$ to $\frac{1}{60}$ gr.) daily) to diminish the salivary secretion may be indicated. It scarcely

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needs to be mentioned that the chief duty of the physician in the later stages of the disease is to pay the most careful attention to the general nutrition of the patient.

Ætiology.—The ætiology is still obscure. It is true that there are patients affected with the disease who, owing to their occupation, have made rather excessive use of the muscles of the tongue and palate (glass-blowers, musicians). These cases, however, are so rare that it would seem very forced to attribute any ætiological importance to this factor. The same may be said about syphilis, the truth being that, in most cases, the cause is absolutely obscure, and all we can say is that males and persons advanced in life seem to be more frequently attacked by the disease than others. Heredity but rarely plays a part, and the influence of cold remains, in connection with this disease, as obscure as with all other nervous affections.

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PART III.

DISEASES OF THE BRAIN PROPER.

WHEN we have reason to suspect a brain lesion in a patient, the questions which arise are: (1) Where is the seat of the lesion? (2) What is its pathological nature? To the physician both of these questions are of interest; to the patient, more especially the latter.

The examination which searches for the seat of the lesion will give us the topical diagnosis ($\tau o\pi o s$ = place); the examination concerning the nature of the lesion, the pathological diagnosis.

The endeavor to localize cerebral lesions—that is, to make a topical diagnosis—has only of comparatively late years received attention, and much of the work so far done can not be called more than an attempt, in many cases indeed only a weak one. The celebrated discovery of Broca (1861), that certain disturbances of speech were often found associated with lesions in the third left frontal convolution, the discovery of Fritsch and Hitzig (1870), that stimulation of certain areas of the cortex produces contractions in certain definite groups of muscles on the opposite side of the body—these and various other, pathological, observations, to which reference will be made later, make it most probable, nay, almost certain, that definite parts or areas of the cortex are always connected with certain functions of the brain; in other words, that these functions can be localized; and, notwithstanding the many uncertainties and numerous contradictions between the results of experiments on the one hand and those obtained from clinical observations on the other, it is this doctrine of cerebral localization which, though still undeveloped, must be considered as the basis of all further investigation in the field of cerebral pathology.

Equal stress must, however, be laid upon the examination into the nature of the lesion. A certain symptom—for instance,

a persistent hemiplegia—is always the result of a lesion of the motor tract; a lesion, however, which can be produced in quite a variety of ways. It may be due to cerebral hæmorrhage, to a tumor, an abscess, etc. It is therefore, especially with regard to the prognosis, of the greatest importance to determine the exact nature of the lesion in a given case, but both questions ought always to be investigated with equal care if we wish to arrive at as exact a diagnosis as circumstances permit. In the following pages these two modes of diagnosis will be considered separately, and we shall first speak of what is known about cerebral localization, while in a later chapter the pathological side will be discussed.

I. The Study of Cerebral Lesions with Reference to their Seat—Topical Diagnosis—Doctrine of Localization.

Two classes of symptoms produced by cerebral lesions must be distinguished—first, general or diffuse (Griesinger); and, secondly, local. The former, so far as they concern the subjective feelings of the patient and the disturbances of the vegetative functions (temperature, pulse, respiration, condition of urine), are to be observed and described in this connection in the same way as in diseases of other organs. The latter—the local symptoms—may be divided into two classes —namely, the direct and the indirect. We call those symptoms direct which are produced by a persistent disturbance in the functions of a certain part of the brain. They are also called focal symptoms (Griesinger). By indirect symptoms we mean those which are only produced by transient conditions —changes in circulation, by compression, etc., and which are in a way concomitant symptoms of the former. They may be entirely absent; on the other hand, they may be so prominent as to make a topical diagnosis impossible.

Destruction of a circumscribed area in the brain gives rise to symptoms of paralysis, or less frequently to symptoms of irritation. The former, where we have to deal with a loss of function, are also (after Goltz) called symptoms of destruction (*Ausfallssymptome*), and if the function is not lost but is only impaired, symptoms of impairment (*Hemmungssymptome*). The latter—namely, the irritative symptoms—are usually due to a so-called indirect action.

It is not always possible to say whether a symptom is of a

direct or of an indirect nature. For instance, if we find a patient in an unconscious state with a hemiplegia, this hemiplegia may be a direct focal symptom or it may have been produced indirectly. In the latter case it will disappear in a few hours or days, in the former it will be persistent. Or, if a patient suffering from the consequences of a cerebral hæmorrhage presents, as is often the case, disturbances in speech, this may again be a focal symptom or not. If, after consciousness has been regained, the speech becomes gradually but steadily better, then the aphasia was produced indirectly. If, however, speech remains unintelligible for months or years, it is clear that we have to do with a focal symptom. Therefore, in acute lesions we can only after a certain time has elapsed discriminate between direct and indirect symptoms.

The irritative symptoms consist either of general epileptiform convulsions or of partial, involuntary movements of the extremities (hemichorea, athetosis), of tremors, contractures, or forced movements of the whole body. We shall repeatedly have occasion to refer to these phenomena.

Not all of the symptoms have an equal value for the localization of a lesion. It is important first to note their mode of onset, whether this is sudden or gradual ; whether several symptoms have made their appearance at the same time or one after the other, and so on, for in acute lesions, for instance, only those symptoms which appear synchronously are of importance. If a patient who has a hemiplegia presents a paralysis of the oculo-motor of the opposite side, and we learn that this latter has existed before the onset of the hemiplegia, nobody certainly will think of connecting the two or look upon them as being symptoms due to one focal lesion. This would only be allowable if both had set in at the same time (after an acute lesion).

But, even apart from the mode of onset, the symptoms are not of equal value in the localization. Some, it is true, as hemiplegia, together with contra-lateral oculo-motor paralysis, are almost pathognomonic (for a lesion in the crus), and their simultaneous appearance is therefore extremely important ; while others, as the conjugate deviation in severe hemiplegia, are found in different lesions, and are therefore less significant ; still others, as optic neuritis and all the general symptoms (headache, vertigo, unconsciousness), are absolutely valueless.

We see, therefore, that by no means all cases can be used
for the study of the topical diagnosis, but that only those in
which the affection, in the first place, remains chronically sta-
tionary; secondly, in which it is circumscribed and isolated
(Nothnagel); and, thirdly, where the surroundings of the focus
are as little as possible implicated.   These three conditions are
best fulfilled in instances of hæmorrhage or embolus, or rather
in the cases of softening produced by these accidents, and the
largest contingent of cases which permit a topical diagnosis is
therefore made up of these.   They are rendered more suitable
for our purpose the longer the time that has elapsed after the
first onset (according to Nothnagel, six to eight weeks), as only
then, as we stated above, are we able to separate the direct
from the indirect symptoms.   In other cerebral affections—e. g.,
meningitis, encephalitis, and especially tumors—a local diagno-
sis should only be attempted with the greatest circumspection,
and even then errors can not be altogether excluded.

Symptoms Referrable to Cortical Lesions.

In speaking of cortical lesions, "surface lesions," it must
not be forgotten that the clinical meaning of the term is differ-
ent from the anatomical one.   Anatomically, it implies that the
medullary layer situated below the cortex is intact, while clin-
ically we speak of cortical lesions even if the white matter
takes part in the pathological process as well; but so little at-
tention has been paid to this difference in the autopsy reports,
which have been published, that it seems an almost hopeless
endeavor to distinguish whether the symptoms reported in a
given case were due to changes in the cellular elements of the
cortex itself, or to changes in the fibres of the medullary layer
situated immediately beneath.

We possess quite a considerable amount of material, but it
is by no means easy to make a judicious and successful use of
it.   Certain methods have to be employed in order to arrive at
correct conclusions, methods which have been developed in
such an excellent way by Exner (cf. lit.).   It would, for exam-
ple, be incorrect to assume a certain cortical area to be the
centre for the motor function of an extremity simply because
in many cases a lesion of this area was found where a paralysis
in that extremity had existed during life.   This "method of
positive cases," as Exner has called it, is therefore uncertain,
because there are quite different circumscribed cortical regions,

a lesion in which gives rise to the same symptoms; and since, moreover, facts go to show that such a method may lead us to wrong conclusions, it ought to be discarded. Much more pref-erable, however, is the so-called "method of negative cases" (Exner), according to which "we have to mark out the lesions, found in all the cases in which a given function was not inter-fered with, and unite them on one hemisphere." If the number of cases is sufficiently large, while on the remaining parts of the

Fig. 38.—RIGHT HEMISPHERE. (After EXNER.) The portions shaded in represent those parts of the cortex which can be injured without giving rise to sensory or motor dis-turbances; the blank areas are motor and sensory.

cortex we find markings indicating lesions, the area for the functions in question will remain free.

Still better results are obtained by the method of percent-ages (Exner). The cortex is divided into arbitrary fields, and for each of these fields we determine, first, how often it has been diseased in a given number of cases; secondly, in how many of the cases the symptom which we are studying was present. The ratio between these two results is best given in percentages. Only through this indispensable, although some-what tedious, method can we ascertain that the fields of the right cortex are different from those of the left, and that cer-

tain areas exist of which a lesion always, and others of which a
lesion not always but frequently, produces a certain symptom.
The former Exner calls "absolute," the latter "relative," corti-
cal areas.

We do not always find cortical lesions at the autopsy in
cases in which certain symptoms, which would have led us to
suspect their existence, have been noted in life. On the other
hand, they are found in cases where we have hardly felt justi-
fied in expecting them. There is no doubt that no inconsider-

Fig. 39.—LEFT HEMISPHERE. (After EXNER.) This diagram illustrates that the motor
and sensory areas are of a greater extent on the left than on the right hemisphere.

able part of the cortex can be diseased without giving rise to
any symptoms. It is this part which has been called the cor-
tical area of latent lesions (Exner), and it is certainly a matter
worthy of note that the extent of this area is smaller on the
left than on the right hemisphere, whereas the motor area—
that is, the area in which a lesion is followed by motor disturb-
ances—is larger and more developed on the left than on the
right side (cf. Figs. 38 and 39). The first represents the right,
the second the left hemisphere. On both all the lesions are
indicated which have produced neither sensory nor motor dis-

turbances. The blank fields are therefore sensory and mo·
tor—their greater extent on the left side is at once ap-
parent.

Before we go into the description of the individual lesions
of the cortex we will briefly refresh our memory on the anat-
omy of the parts.

The thin gray covering which surrounds the white matter, and
which has been called brain cortex, presents on each hemisphere
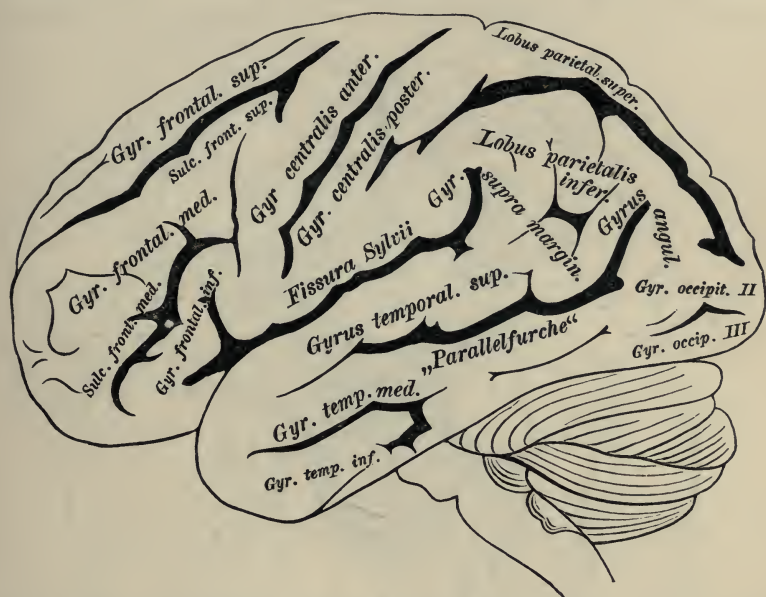
Fig. 40.—Convolutions and Fissures of the Lateral Aspect of the Brain.
(After Ecker.) Parallelfurche = parallel fissure, or first temporal fissure.

three surfaces—a lateral, a basal, and a median. The two lateral
form the convexity, the two basal the base of the cerebrum. The
cerebrum is divided into lobes, which can again be subdivided into
convolutions or gyri. To be able to localize and correctly describe
cortical lesions we must be thoroughly familiar with the position, as
well as the names, of the different convolutions. The following
illustrations are intended to facilitate the study of the convolutions
and the fissures or sulci separating them. Fig. 40 represents those
on the lateral surface (convexity), Fig. 41 those on the basal, and
Fig. 42 those on the median aspect of the cerebrum.

In Fig. 40 are included the frontal, parietal, temporal, and oc-
cipital lobes, so far as their convolutions and fissures belong to the

lateral surface, in other words, belonging to the frontal lobes, the three frontal and the anterior central convolution (gyrus centralis anterior, *pli frontal ascendant*), and belonging to the parietal lobe, the posterior central convolution (gyrus centralis posterior, or *pli pariétal ascendant*); between the two last is seen the fissure of Rolando. Further, a part of the upper and the entire lower parietal lobe are shown, which latter is subdivided into the supra-marginal convolution in front and the angular gyrus (*pli courbe*) behind; belonging to the temporal lobe we have the three temporal convolutions, of which the first (uppermost) lies between the fissure of Sylvius and a very deep fissure running parallel to it, the so-called
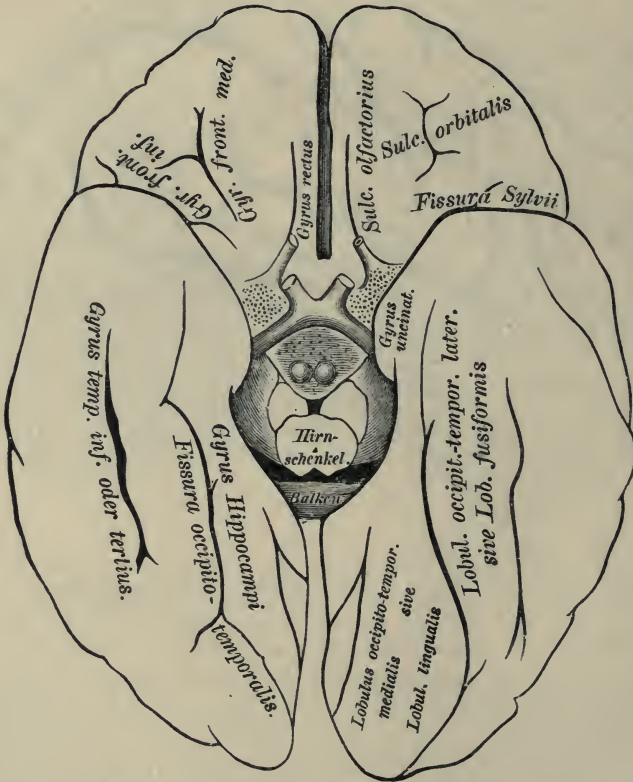
Fig. 41.—Convolutions and Fissures at the Base of the Brain (diagrammatically after Ecker). Hirnschenkel = crura cerebri. Balken = corpus callosum.

parallel fissure or first temporal fissure. The fissure of Sylvius has two branches, and the portion of the cortex between them is called the "Operculum." Belonging to the occipital lobe, finally, there are three irregular and not always easily distinguishable occipital

convolutions, between which two occipital fissures have been described.

On the basal aspect we see those parts of the three frontal convolutions which are included in the base, of which the first (uppermost) is here called the gyrus rectus; then the tractus, with the sulcus olfactorius; next the uncinate gyrus, which belongs to the gyrus fornicatus and which will be better seen on the median aspect; further, the basal part of the third temporal convolution (gyrus temporalis inferior) and two lobules, which belong to both the temporal
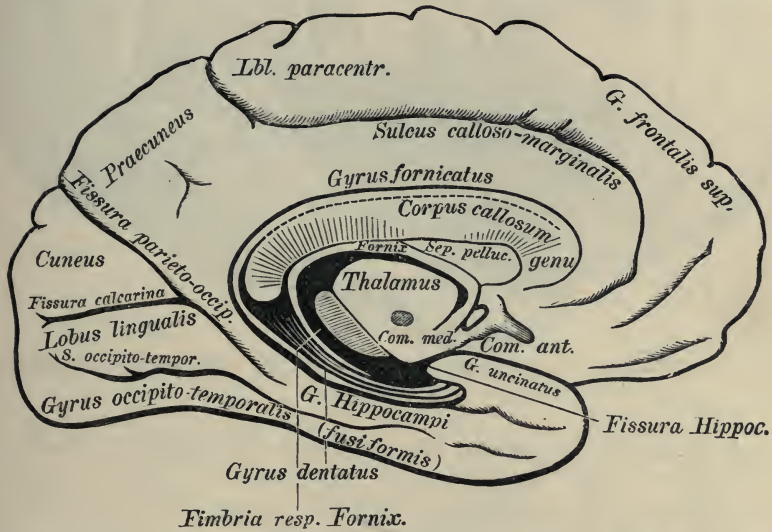
Fig. 42.—CONVOLUTIONS AND FISSURES OF THE MEDIAN ASPECT OF THE BRAIN. The posterior portions of the thalamus and the crus cerebri are cut off.

and occipital lobe, the inner (median) one called the lingual lobule, the one more external the fusiform lobule.

The median surface (Fig. 42) shows in the middle the corpus callosum (in front the genu, behind the splenium); immediately below is the "septum lucidum," immediately above the gyrus fornicatus, the temporal part of which is called the hippocampal convolution, and is continuous with the uncinate gyrus. Above the gyrus fornicatus, and separated from it by the calloso-marginal fissure, are the frontal convolutions; further back, the paracentral lobule, which meets the central convolutions. Behind this and belonging to the parietal lobe is the præcuneus, and still further back the cuneus (of the occipital lobe). The latter is bounded by two deep fissures—below by the calcarine, in front by the occipito-parietal fissure. Extending from the posterior part of the corpus callosum to the uncinate gyrus is the hippocampal fissure.

Covered by the above-mentioned operculum, in the depth of the fissure of Sylvius, is the lobus intermedius s. opertus, the so-called island of Reil, on which five to seven small convolutions are seen.

Fig. 43.—Convolutions of the Island of Reil (J. R.) made visible by removing the Operculum.

Their position is shown in Fig. 43, where the operculum has been removed.

In Fig. 44 the topographical relation between the surface of the brain and surface of the skull is illustrated.

Among the parts which most frequently may be the seat of lesions without producing any consequent sensory or motor disturbances during life is the frontal lobe. Some convolutions —viz., the first and second frontal—are certainly not to be reckoned as forming part of the motor area (cf. Figs. 38 and 39, with the lesions marked on the frontal lobe). On the other hand, of great significance and importance is the anterior central convolution, as a part of the so-called motor region, and the third (lowest) frontal convolution of the left hemisphere, as the area of speech. To the motor region belong not only the anterior, but also the posterior central convolution, and the paracentral lobule. Extensive lesions of this region, foci of softening, which cause destruction of the cortex, produce symptoms which collectively give us the picture of cerebral hemiplegia, of which we shall give a more minute description later

on. Circumscribed lesions produce affections of the individual centres of the extremities (absolute cortical areas, Exner), and give rise to symptoms either of a paralytic or of an irritative nature. To the latter belongs, among others, the so-called symptomatic or Jacksonian epilepsy.

That the cortex of the frontal lobes is the seat of the higher psychical functions is shown by the changes which are found in certain forms of idiocy, often also in dementia paralytica, where wide-spread disease on both sides has been observed—
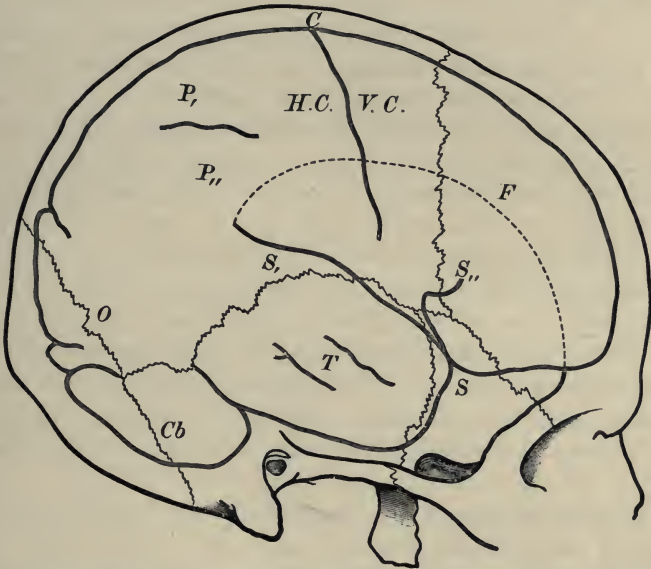
Fig. 44.—Topographical Relations between the Exterior of the Skull and the Surface of the Brain. (After Ecker.) *C*, fissure of Rolando. *V.C.*, anterior central, *H.C.*, posterior central convolution. *SS,S,,,* fissure of Sylvius. *T*, temporal lobe. *F*, frontal lobe. *P,,* upper, *P,,,* lower parietal lobe. *O*, occipital lobe. *Cb*, cerebellum.

viz., chronic inflammatory processes, atrophy of the convolutions of the right as well as of the left frontal lobe.

The functions of the cortex of the parietal lobe are entirely obscure. Lesions in the upper parietal lobe have often been observed after paralysis in the extremities; but in this connection we should never lose sight of the influence of the posterior central convolution, which belongs to the motor region. The statement that it is in the lower parietal lobule, especially in its posterior part (angular gyrus), that the centre for the movements of the levator palpebræ superioris ought to be looked

for (Landouzy) has as yet to be proved, and the relation which the parietal lobe bears to sensibility is equally obscure, notwithstanding the disturbances in muscular sense which have often been observed in diseases of this region (Bastian, cf. lit.).

In the cortex of the temporal lobe, in the first temporal convolution, most commonly on the left side, important centres of the auditory nerve are situated, and it is most probable that lesions of this region give rise to central deafness in the ear of the opposite side. Lesions of the first left temporal convolution give rise to "sensory aphasia" (Wernicke), "word deafness" (Kussmaul). Motor disturbances have never been found associated with diseases of the temporal convolutions.

The cortex of the occipital lobe finally contains the centre for the opticus, and lesions of sufficient extent in this region produce hemianopia (cf. page 34). Quite a number of postmortem examinations seem to point to the first occipital convolution and to the cuneus as the seat of the centre for vision. The interesting fact, observed by Fürstner, that certain patients, although able to see objects before their eyes, are unable to recognize them—i. e., have forgotten their significance—is also a consequence of lesions in the occipital cortex; the memory for optical pictures is lost, and we have what is called a psychical blindness (Munk). Motor disturbances (paralysis, spasms) are not caused by diseases of the occipital convolutions.

The convolutions of the island of Reil are but rarely affected by themselves, usually other parts, the third frontal convolution, the centrum ovale, etc., being also implicated; it is therefore impossible at present to state with any certainty at all in what way speech is connected with the insula of the left side, and to give the relations between pathological conditions in this part and aphasic symptoms.

While from what has been said we see that the functions of a large part of the cerebral cortex are as yet obscure—in other words, that the "area of latent lesions" is of lamentably large dimensions—we have, on the other hand, the satisfaction of knowing that the significance of some areas is understood with a fair amount of clearness. It is true even these can to a large extent only be regarded as "relative" (in the sense of Exner), for, as we shall presently see, there are only very few "absolute" areas.

In the motor region of the right side, the paracentral lobule, the anterior central convolution, and the upper part of the posterior central convolution, on the left side, the paracentral lobule, the three upper quarters of the two central convolutions, and a part of the upper parietal lobule, are properly called the absolute cortical area for the upper extremity (Exner). The absolute area for the lower extremity is formed by the paracentral lobule and the upper third of both central convolutions on the right side, on the left by the paracentral lobule, the upper half of the posterior central convolution, and the greater part of the upper parietal lobe.

The cortical area of the trigeminus has been spoken of on page 55, that of the facial on page 78, and that of the hypoglossus on page 142. Of the centres for the eye muscles, especially for the levator palpebræ, mention has been made in the third chapter of the second part (cerebral blepharoptosis, page 44).

The speech centre, which, as we have seen, is situated partly in the frontal, partly in the temporal lobe of the left hemisphere, is certainly of larger extent than is commonly supposed. It is well known that after Bouillaud in 1825 had pronounced the frontal brain, and Marc Dax in 1836 the left hemisphere, to be the seat of speech, Broca claimed that the posterior part of the third left frontal convolution, the pars opercularis, or, as it later was called, the region of Broca, contained the speech centre; and, indeed, in speech disturbances a lesion of this very region has most frequently been found at the autopsy. There are, however, other parts of the cortex, as the island of Reil, the central convolutions, and, above all, the temporal lobe, more especially its upper convolution, which are connected with speech and which are of no less importance. Thus we have, after much laborious work and after many careful observations and comparisons, come to the conclusion that a different form of speech disturbance (aphasia) is produced according as the lesion is one of the frontal or of the parietal lobe—(of the left side only). In the former case the patient knows the word which he wishes to pronounce, but can not do so because he has lost the memory for the movements necessary for speech—i. e., he no longer knows how to use his tongue and lips in the act of speaking—motor aphasia. In the latter case the patient knows exactly what he wants to say, and he has no difficulty in repeating it if it is spoken for him;

but he can not find the expression for himself, he has "forgotten" the word—sensory aphasia. That the understanding of words is situated in the temporal lobe was first stated by Wernicke, who also originated the terms motor and sensory aphasia.

In the diagram of Wernicke which is shown in Fig. 45, y represents the motor, x the sensory speech centre; the latter is the terminus of the centripetal path of the auditory nerve $a\,x$, the former is the beginning of the centrifugal path $y\,m$ going to the muscles used in speaking; $x\,y$ an assumed association path between both; y is situated in the third frontal, x in the first temporal convolution.
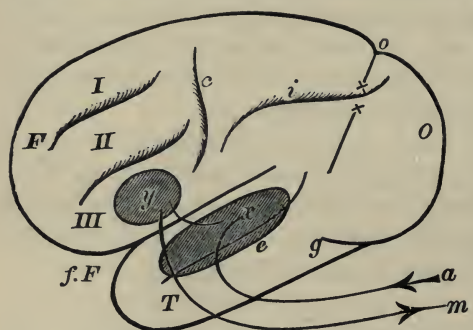
Fig. 45.—Wernicke's Schema for the Cortical Mechanism of Speech.

According as one or the other of these centres or the connection between them, or both centres were destroyed, Wernicke distinguished four cardinal types of aphasia:

1. Destruction of the centre y—motor aphasia. Mobility of the muscles used in speech is retained, but the patient can either not speak at all or only say a few words or syllables. Understanding and memory of words is intact.

2. Destruction of the centre x—sensory aphasia, " word deafness " (Kussmaul). The patient can use as many words as ever, but in speaking they are mixed up. The understanding of words is lost, although the power of hearing is not interfered with.

3. Destruction of the association path $x\,y$, situated in the insula (?)—the so-called conduction aphasia of Wernicke. The patient can use as many words as ever, but in speaking they are mixed up. The understanding of words is retained.

4. Destruction of both centres, x and y—total aphasia. Power and understanding of speech are lost.

If we then consider as proved that a certain group of motor and sensory memory pictures are localized in the brain, if we further agree that the former correspond to certain groups of muscles which serve a common purpose, the latter to the distribution of a sensory nerve—it is not difficult to conceive that

this same arrangement may exist for all the muscles and for all the sensory nerves. It is certainly easy to understand the occurrence of other motor defects in cases of aphasia. Thus there may be loss of simple movements (e. g., of the power to put out the tongue), or more complex ones (e. g., writing may become impossible—agraphia ; *aphasie de la main*, Charcot). Again, we have a patient who, in consequence of a cortical lesion in the central termination of the optic nerve, no longer recognizes his letters, and has thus lost the faculty of reading ("alexia"), or the visual memories may be lost altogether (not only those of letters), and a condition ensue which Munk calls psychical blindness.

In examining a patient affected with aphasia, with a view of determining which path has become interfered with, we may meet with considerable difficulty, and the diagnosis of the particular type of aphasia with which we are dealing is often not easy, for the cases are not so sharply defined or so well characterized as we might be led to expect from the simplicity of the schemata. On the contrary, we meet often with combinations of the different types or with transition forms of aphasia in which even the most experienced clinician will venture a differential diagnosis only with much reservation. Take, for instance, the different degrees of that form of speech disturbance known as ataxic aphasia, in which the patient is unable to pronounce a word, though it constantly is floating, as it were, before his mind. This inability may go so far that the patient can only pronounce a few words or syllables (monophasia), that he involuntarily confounds words without being in the least uncertain about their meaning ; or it may, on the other hand, only amount to a slight disturbance, shown by some misplacement or omission of some letters, as in saying *dy* instead of *dry*, *turk* instead of *truck*, and the like. In the latter case we speak of syllable-stumbling (*Silbenstolpern*). Likewise we have different degrees of the so-called amnesic aphasia, where there may be loss or only slight impairment in the memory for words (sometimes only for words of foreign languages which have been learned later in life). As the faculty of writing and reading may often be more or less altered, it is important that it should be minutely examined into. Every case of aphasia must be carefully studied by itself, and in each one is bound to find some interesting points which we can not now stay to discuss in detail.

12

In general we may be guided by the following rules :

1. If we find a patient whose sanity is established, who possesses a normal acuteness of hearing and understands what is said, but is unable to repeat sentences or to speak spontaneously, and can only utter individual words and syllables, we may assume a lesion of the third frontal convolution, possibly of the lowest part of the anterior central convolution.

2. If a patient, although able to speak without difficulty, does not understand simple questions, then the first temporal convolution is diseased (*in toto*). If the understanding of words is only impaired, then only a part is affected.

3. If the patient has lost the faculty of reading, although there is no motor aphasia to be noted, we have to deal with a lesion of the cortical centre for vision (cf. page 29).

4. A disease of the cortical speech centre does not exist if the patient gradually regains speech which he had suddenly lost ; if in such a case the hemiplegia, which has simultaneously appeared after an apoplectic stroke, persists, the white substance near the cortex is wont to be diseased (Gowers).

We should be going beyond the limits of our work if we examined into all the details of the complex symptomatology of aphasia, difficult, and indeed in some phases still uncertain, as it is, the more so as there exist quite a large number of excellent special works dealing with this subject, an acquaintance with which is indispensable for the more accurate study of these interesting affections. Let it suffice to outline the different forms of aphasia, and this, according to our experience, will be best and most easily accomplished with the aid of schemata, of which, besides the above-mentioned one of Wernicke, quite a number have been brought out. The one we have deemed most suitable and the best fitted for teaching purposes is probably that which Lichtheim demonstrated at the conference of neurologists at Baden Baden in 1884. It has been here given in Figs. 46 and 47.

The reflex arc necessary for repeating words contains the centre for auditory images of words, A, the centre for motor images, M, the centripetal path for auditory impressions, $a A$, the connecting path, $A M$, the centrifugal motor path, $M m$. B is the place where concepts are formed—voluntary speech necessitates a centrifugal path from B (brain cortex) to M. O is the centre for the visual images of letters. E is the centre for the innervation of the muscles required in writing. Now,

according to the path affected, we distinguish seven different forms of aphasia :

1. Interruption in the point *M*.   Broca's (motor) aphasia.

2. Interruption in point *A*. Wernicke's (sensory) aphasia.

3. Interruption in the path *M A*. Conduction-aphasia (Wernicke).

4. Interruption in the path *M B*. A variety of motor aphasia ; the faculty of repeating words and sentences being retained.

5. Interruption in the path *M m*. Variety of motor aphasia ; the power of expressing thoughts in writing being retained.

6. Interruption in the path *A B*. A variety of sensory aphasia ; the patient being, however, able to repeat spoken language, to read aloud, and write from dictation.

7. Interruption in the path *A a*. Inability to understand spoken language and to write from dictation, or to repeat spoken language. Numbers six and seven have not yet been observed.

As to the occurrence of aphasia, it is
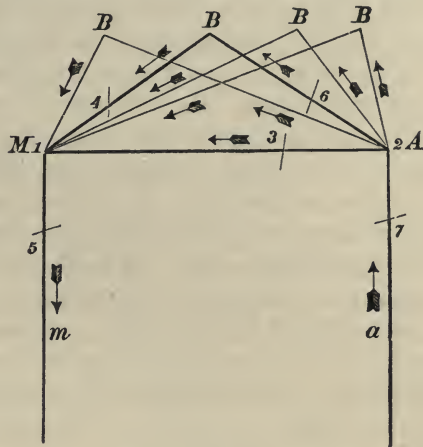
Fig. 46.
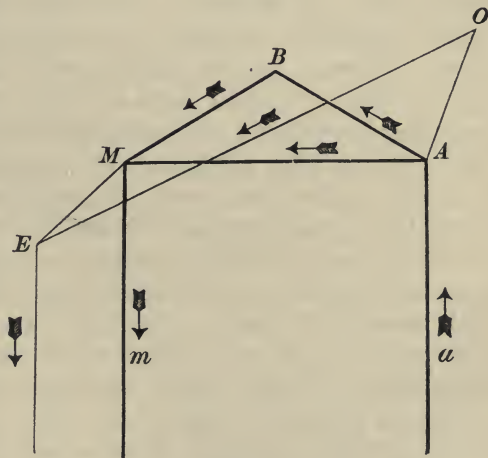
Fig. 47.

Figs. 46, 47.—LICHTHEIM'S SCHEMA ILLUSTRATING THE SEVEN DIFFERENT FORMS OF APHASIA. *a. A.*, centripetal path for auditory impressions. *A*, centre for auditory images. *M*, centre for motor images. *M, m*, centrifugal motor path. *B*, the place where concepts are formed. *O*, the centre for visual images. *E*, centre from which the organs of writing are innervated. (The seven different forms of aphasia have been indicated in Fig. 46 by the numbers 1–7.)

most frequently seen as a sequence

to an apoplectic attack, either as a direct or indirect symptom; in the latter case it is transient, and lasts, as we shall see later, a few minutes, hours, or days. In the former it persists, and may trouble the patient, though he may retain his full mental vigor, to the end of his life. The most common form is motor aphasia, which appears in widely different gradations; thus, in some cases the patient's speech may be just a little thick, while in others it may be altered so that it is no longer intelligible. After what has been said, it is easy to understand that these defects chiefly occur after hæmorrhage in the left side of the brain—that is, with a right-sided hemiplegia—but it would be a decided error to suppose that they occur only or always in those cases, for motor aphasia may indeed be found in connection with a left-sided hemiplegia, and it may be wanting in the right-sided form. Other diseases also may implicate the cortical speech centre and give rise to aphasia; such are processes of softening, chronic meningitis, tuberculous deposits, etc., and traumatism of the left hemisphere. Aphasia has also been observed in acute, especially infectious, diseases—e. g., typhoid and scarlet fever. Most instances of this latter form occur in children. Of special interest is that form of total or motor aphasia which sometimes suddenly, sometimes gradually, comes on after a fright. That after a fright, such as makes "the hair stand on end," the voice may refuse to perform its duty even Virgil seems to have known full well, as we see from the verse, "*Steteruntque comæ, vox faucibus hæsit.*" The nature of this form is uncertain, still it is by no means impossible that, just as we find that vaso-motor spasm acting on the facial vessels will produce pallor, so we may have a similar condition in those finest distributions of the middle cerebral artery which supply the region of Broca (cf. page 175). That the spasm in these vessels is usually of longer duration and produces more serious and more lasting consequences than the spasm of the cutaneous vessels may be explained by the difference in their arrangement, as well as by the difference in the function of the parts which they supply. .

It is not organic changes of the cortex which produce the symptoms in this case, the disturbances being entirely of a functional character, and this fright aphasia therefore constitutes a transition form to those instances in which, though the aphasia may have lasted for years, no changes are found at the

autopsy, either in the cortical or subcortical area for speech. No doubt there is, besides the aphasia due to actual lesions in the cortex, also a functional form which we may imagine to originate in different ways, and it is at least probable that variations in the blood supply of the centres play an important part in this connection. Not long ago Grashey (cf. lit.) has shown, in an ingenious piece of work, that we have to recognize a third form of aphasia, in which neither the centres nor the conducting paths are insufficient in their functions, but which is simply due to a diminished duration of the sensory impressions, giving rise to a disturbance in perception and association, and thus to an aphasic condition. Maybe it is this aphasia of Grashey which we find after concussion of the brain and after acute diseases, but it is difficult to diagnosticate it, and to differentiate a functional disturbance of the centres from a diminished duration of sensory impressions. A correct diagnosis is, however, of no small importance in the question of prognosis.

The outlook is absolutely unfavorable in cortical lesions where the centre is destroyed by processes of softening, tuberculous deposits, atrophy of the gray cortex, etc., but is, of course, materially better if the centres remain intact, and are only temporarily rendered unable to perform their function, for then speech returns gradually, if not wholly, partially, and it can not be denied that systematic exercise and regular instruction in speaking is capable of hastening an amelioration, nay, even a cure, especially if the patient be still young.

The by no means rare aphasia of children, which we sometimes find in consequence of intestinal worms (" reflex aphasia "), in the course of acute infantile cerebral palsy, occasionally, but very rarely also, after a cerebral hæmorrhage, is in no other way to be distinguished from the aphasia of adults except in its prognosis. Children, *ceteris paribus*, always stand a better chance of improvement or recovery from aphasia than adults, no doubt because it is easier to educate in them the well half of the brain to perform the function of the damaged one. If the disturbance is only functional, as I saw in one case which was due to an overdose of santonin, in which the disorder in speech only lasted a few hours, the outlook is still more favorable, and complete recovery may be confidently expected ; but if the function of one speech tract—that is, the left—be impaired by cortical or deep-seated lesions, even then it is in

children usually not very difficult to educate the right side to some vicarious action, especially in cases .where, before the lesion, the children have been taught to use both hands equally. The possibility of a cerebral disease should be thought of in the gymnastic cultivation and development of the body of children; the extremities of both sides should be exercised and strengthened equally, the children should be made ambidextrous; only then can, in a case of necessity, the right hemisphere fully take the place of the left.

We have repeatedly pointed out that the motor disturbances produced by cerebral diseases are either due to destructive or irritative lesions. The former consist of paralyses and pareses, the latter of involuntary movements in different groups of muscles—the so-called spasms. Those disturbances which are due to affections of the cortex (cortical motor disturbances) present much that is characteristic and interesting. They will be considered presently.

The motor centres, the motor area of the cortex, comprise, as has already been stated on page 172, the two central convolutions, the paracentral lobule and the parts lying immediately adjacent. Upper and lower extremities, neck and face, have their own special centres, which are distinctly separated from one another in the central convolutions. Thence fibres converge, as is shown in the diagram of Fig. 48, into the anterior two thirds of the posterior division of the internal capsule. One thing at once becomes apparent from this arrangement, namely, that in cortical lesions the paralysis or paresis may easily be confined to one extremity, an arm or a leg, while if the lesion affects the tract lower down in the brain—for instance, in the region of the capsule—the paralysis must extend over the whole half of the body. A paralysis of one extremity only is called a monoplegia (monoparesis) in contradistinction to a hemiplegia, which means paralysis of one side (half) of the body, and it is a perfectly warrantable conclusion, sufficiently confirmed by post-mortem evidence, that, if the patient presents a paralysis of only one arm or one leg, we are dealing with a cortical lesion. A hemiplegia is only then likely to be of cortical origin if its development indicates that the lesion beginning in one motor centre has gradually encroached upon another. It goes without saying that in diagnosticating cortical lesions we must not rely on a single symptom, but all must be considered, and especial care must be taken not to

confound a paralysis of cortical with one of peripheral origin. One great distinction between these two is to be found in the manner of onset. While a paralysis of cortical origin may develop quickly in a few hours, a peripheral one will be more gradual, and only reach its full extent after weeks or even
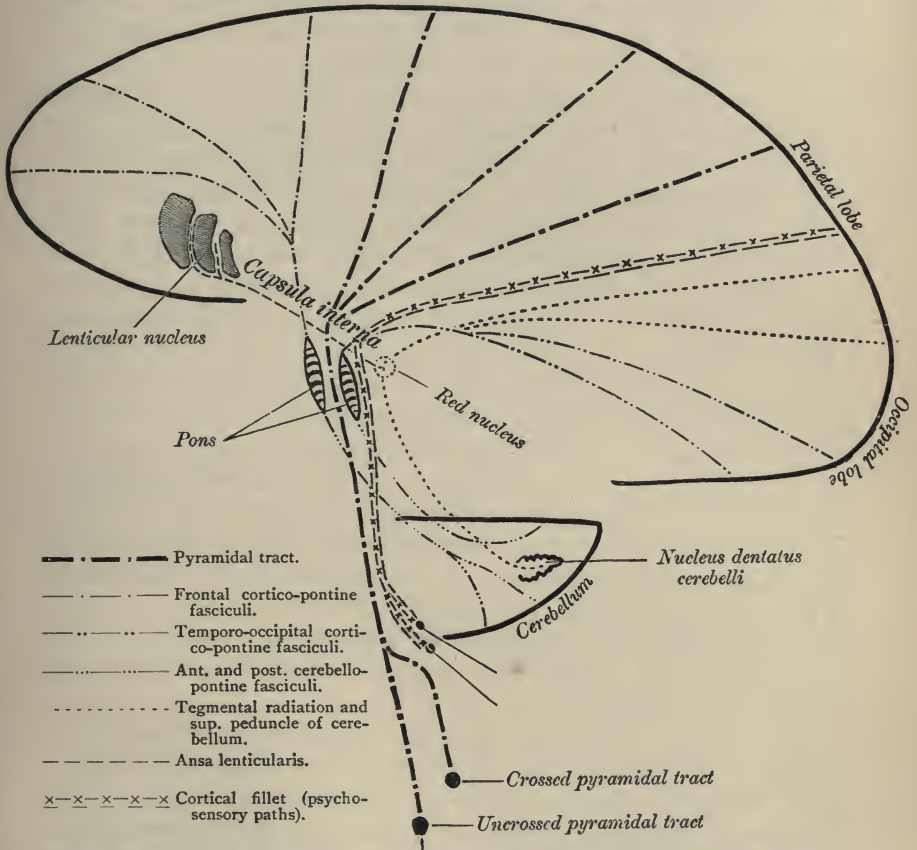
Fig. 48.—DIAGRAM SHOWING THE DIRECT SYSTEM OF FIBRES (FLECHSIG, MENDEL).

months. Moreover, the latter, the peripheral, is easily recognizable by the changes which take place in the electrical excitability—e. g., if reaction of degeneration and visible atrophy in the muscles can be demonstrated. The absence of cerebral symptoms, which are rarely entirely wanting in cortical affections, is also characteristic of peripheral disease. Great pain may be entirely absent in the central, but is commonly present to a greater or lesser degree in the peripheral variety. Re-

membering then these points, and making it a routine practice never to omit the electrical examination in doubtful cases, we are not likely to make an error in the diagnosis.

In cortical lesions the loss of motion is usually not absolute, and we find more frequently a paresis than an actual paralysis. The affected arm or leg can be moved *in toto*, though a strong effort may be required, and it is only in the fingers and toes that the loss of power is often complete.

A characteristic symptom is the inability of the patient to execute complicated movements, such as buttoning his coat, counting money, and so forth, acts which are performed awkwardly and with difficulty, owing to a loss of the motor images. This condition has been called ataxia, and in these cases we have a " cortical ataxia." The lesion has to be referred to that part of the cortex which contains the sensory area (*Fühlsphäre* of Munk) for the affected, that is, the ataxic extremity. The trouble is very distressing to a patient in a brachial as well as in a crural monoplegia, and becomes almost unbearable if the sensory disturbances, which we shall shortly describe, are superadded (cf. lit., Observations of Bernhardt).

In infective tumors, gummata, tubercles at the surface of the brain, we occasionally meet with symptoms of irritation, such as monocontractures, which depend upon an irritation in the corresponding portion of the motor path (Wernicke). They are not seldom accompanied by sharp pains. In such cases the differential diagnosis between a hysterical and a true organic cortical lesion may cause considerable difficulty (cf. chapter on hysteria).

Of the greatest practical importance are the epileptiform attacks which occur, either with or without loss of consciousness, as a consequence of direct or indirect irritation of the cortex. If they occur in the further course of the monoplegia, the onset of which was apoplectiform, the diagnosis of a cortical lesion can be made with a high degree of probability. In some cases the convulsions are not general, but only appear as localized twitchings or spasms, confined to one half of the body or one extremity ; they may be clonic (that is, an alternation in quick succession of contractions and relaxations) or tonic (that is, steady contractions lasting for some time), and may be of considerable intensity ; their occurrence later in parts already paralyzed would indicate a disease of the brain surface, though we may not always be able to say whether the irritation of the

cortex depends upon a direct or—as, for instance, in tumors, which cause an increase of the intracranial pressure—an indirect action. In the latter case, also, general or partial convulsions may ensue.

We shall later on have occasion to go more thoroughly into the study of the clinical picture as well as the pathological changes of this "cortical epilepsy."

The sensory disturbances which are produced by the affections of the brain cortex are remarkable, and by no means fully understood. As we have seen before, they do not, as a rule, cause pain, but rather manifest themselves in alterations of sensation, known as paræsthesias. Thus the patient may speak of a curious numbness, deadness—further, a feeling of ants crawling under the skin, a feeling as if the part had gone to sleep, etc. There may also be a distinct increase in pain perception, a slight "analgesia," a diminution or loss of pressure, touch, and temperature sense, and, oftener as it seems in diseases of the parietal lobes, a more or less pronounced disturbance of the muscular sense, in consequence of which the patient can with closed eyes either give no account at all or only a very imperfect one of the position of his extremities. If, as often happens, the above-described awkwardness in motion (ataxia) co-exists with these changes, we may be tempted to refer the trouble not to the cortex, but to the spinal cord ; more especially are we liable to think of tabes, although the ataxia is produced in an entirely different manner in the two diseases. However, the differential diagnosis will in most cases present no difficulties if we take into consideration all the symptoms, and examine into the condition of the patellar reflexes, the reaction of the pupils, and ascertain whether there are bladder symptoms and whether lancinating pains are present or not. These sensory changes, which we must not forget, are by no means always observed in cortical lesions, and in the cases in which they existed the white matter of the brain has often been found to be likewise the seat of disease ; they are therefore in no way to be regarded as pathognomonic, and we have to be cautious in using them for diagnosis. The same is true to a greater degree of the vaso-motor and trophic changes, the relation of which to the brain cortex is still obscure.

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Symptoms Referable to Lesions of the White Matter of the Hemispheres and Lesions of the Basal Ganglia.

Looking at the anatomy of the parts, we notice that the fibres coming from the cortex pass through the white matter of the hemisphere, which in the region of the frontal and parietal lobes is designated centrum semiovale Vieussenii.   Turning toward the brainstem, in its neighborhood they appear arranged in bundles placed side by side, completing by their convergence what has long been known as the corona radiata.   With this corona radiata begins the great nerve tract which connects the hemispheres with all parts of the brain situated lower down, and finally with the spinal cord.   That part of the medullary path through which the corona radiata is continued into the crura cerebri is called the internal capsule.   As is seen in Fig. 49, this is situated anteriorly between the caudate and the lenticular nucleus, posteriorly between the lenticular nucleus and the optic thalamus.   The point where the two segments meet is called the genu, the knee of the capsule.

From the internal capsule the fibres reach the crusta (pes pedunculi cerebri), whence they pass through the lower (anterior or ventral) portion of the pons and enter the medulla oblongata as the anterior pyramids.   At the lower end of the medulla most of them decussate and pursue a downward course in the lateral columns of the spinal cord on the opposite side.   This, the most important of all direct systems of fibres, was discovered by Deiters in 1865, and

most carefully studied by Flechsig in 1876. It is generally known as the lateral pyramidal tract, and it represents the path for the voluntary movements. A lesion of it is therefore of grave consequence for the motor functions.

In Fig. 50 the position of the centrum semiovale is represented, while Fig. 51 gives another horizontal section in a plane somewhat
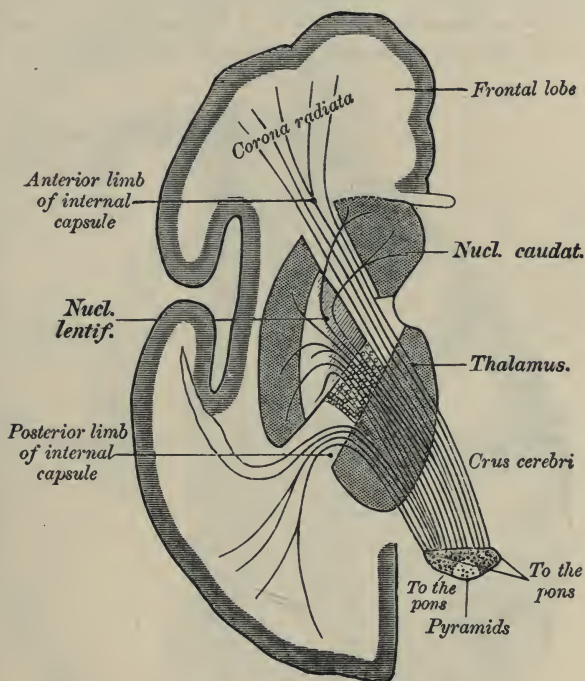
Fig. 49.—COURSE OF THE FIBRES FROM THE INTERNAL CAPSULE TO THE CRUS CEREBRI. The thalamus is represented as transparent. (Diagrammatic after WERNICKE and EDINGER.)

lower down, and shows the relative position of the caudate nucleus to the optic thalamus, the corpus callosum, the fornix, the two white commissures, the anterior and the posterior, the gray middle commissure, the pineal gland, and the corpora quadrigemina.

Fig. 52 is a third horizontal section through the cerebrum at a still lower plane. It has to be studied in conjunction with the diagram given in Fig. 48.

Fig. 53 represents the base of the brain, with the superficial origin of the twelve pairs of cranial nerves (cf. Fig. 28).

Figs. 54, 55, and 56 are three so-called frontal sections, of which the first is made through the anterior commissure, the second in

front of, the third behind the middle (gray) commissure. They also show the course of the internal and external capsule, and the situation of the so-called basal ganglia, the caudate and the lenticular nucleus (together known as the corpus striatum), and the optic thalamus.

In another frontal section, Fig. 57 (after Edinger), the direction of the fibres is illustrated diagrammatically.

Pitres has recommended a series of frontal sections in order to facilitate in our descriptions of autopsies a more accurate localiza-

Striae longitud.

Centrum semiovale

Corpus callosum
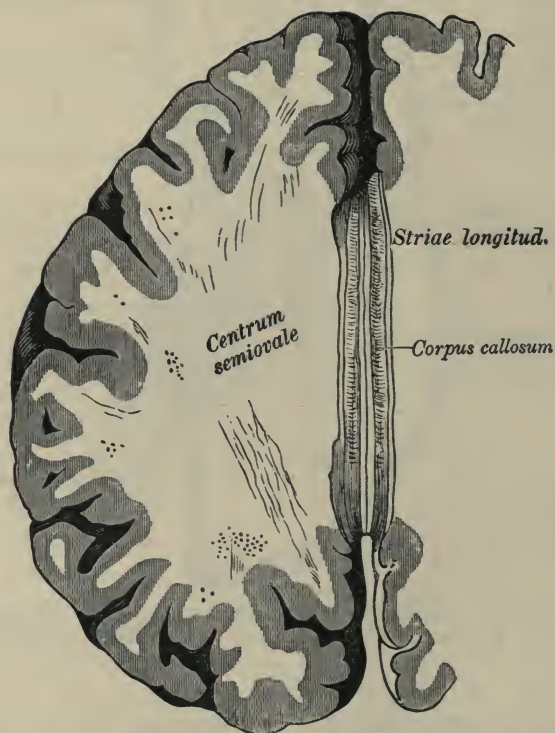
Fig. 50.—HORIZONTAL SECTION THROUGH THE HEMISPHERES.

tion of lesions and tumors within the very extensive white matter of the brain. Nothnagel has modified somewhat these sections of Pitres with regard to their position and designation. The table on page 196 contains the necessary explanation. With the help of these sections we need not content ourselves any longer with locating in the post-mortem accounts a tumor " in the anterior part of the brain," " in the temporal lobe," etc., but we give the one or more sections which correspond to the situation of the neoplasm, and so

attain an accuracy which is indispensable for the after-use of our autopsy records.

Figs. 59 to 64 represent diagrammatically Pitres's sections. From Fig. 58 we can get an idea of the points on the surface of the brain

Corp. callos.
Ventric. later.
Ventric. sept. pelluc.
Nucl. caud.
Fornix.
Commiss. anter.
Commiss. media
Tænia semicircularis
Thalamus
Commiss. poster.
Pineal gland
Pulvinar
Corpora quadrigemina
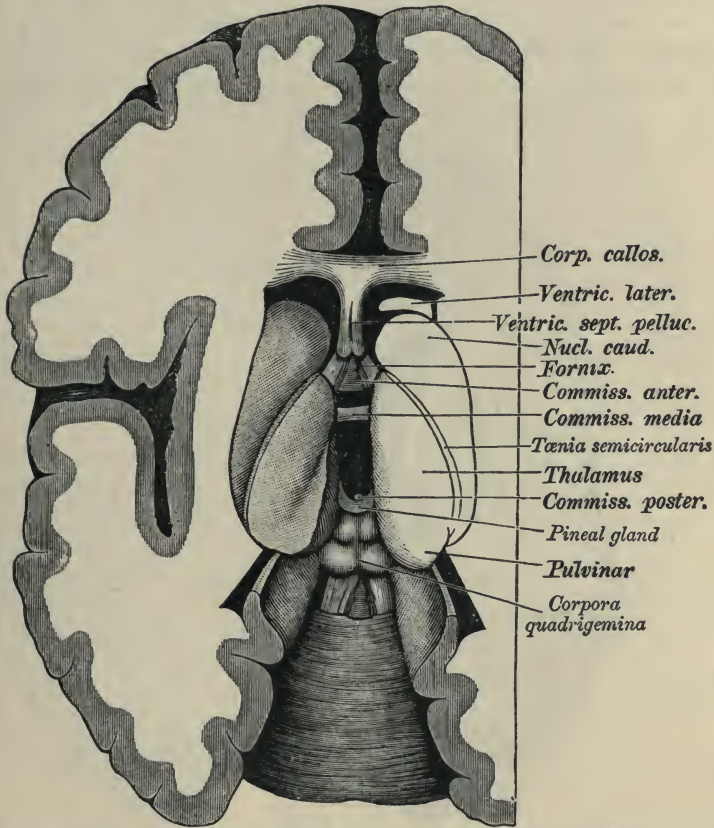
Fig. 51.—View of the Ventricles on Horizontal Section. (After Edinger.)

at which the sections are to be made (cf. Pitres, Recherches sur les lésions du centre ovale des hémisphères cérébraux étudiés au point de vue des localisations cérébrales, Paris, 1877).

We can likewise avail ourselves of the charts of the human brain published by Exner (two plates, with twelve diagrams: Wien, Braumüller, 1888). On the plates the discovered lesion can be easily marked off, and thus the extent and situation of it represented.

With reference to the lesions in the centrum ovale, it should be stated that, as a rule, the symptoms produced by them are

similar to those which we find in lesions of the corresponding
area of the cortex. Thus we shall meet with motor disturb-
ances if the fronto-parietal fasciculi of the corona radiata, which
take their origin in the motor area, are diseased; while lesions
in the præfrontal or occipital bundles may, and indeed very
often do not evoke any symptoms. If the left (inferior) pedic-
ulo-frontal bundles in addition are affected, the patient will
also be aphasic, the aphasia, however, being of long duration
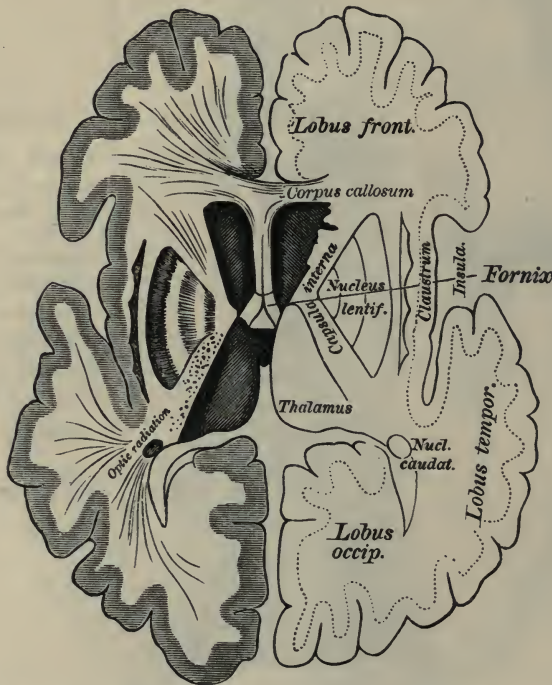
Fig. 52.—Horizontal Section through the Brain, about a Finger's Breadth
below that represented in Fig. 51. (Edinger.)

only if the lesion extends close up to the cortex. Lesions in
the white matter of the occipital lobe may produce hemianopia,
in the temporal lobe auditory disturbances. Whether, how-
ever, diseases in the parietal lobes ever produce sensory
changes—anæsthesia, for instance—and whether, as a conse-
quence of any lesion in the centrum ovale, vaso-motor-trophic
changes may be developed, is unknown.

The idea that the basal ganglia were true motor centres,
and that the common form of hemiplegia was due to lesions
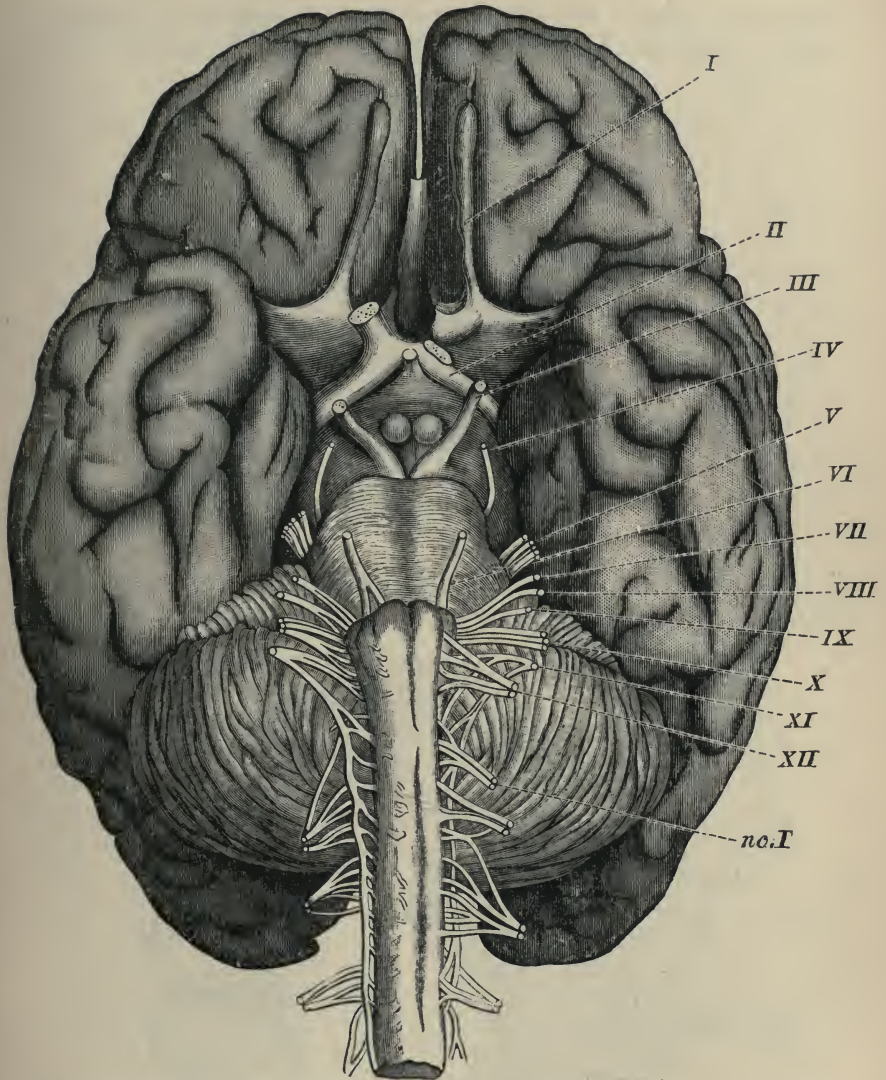
Fig. 53.—Base of the Brain (with the First Portion of the Spinal Cord). *I–XII*, the twelve cranial nerves. *nc1*, first cervical nerve.

thereof, has been given up, and we have learned from the investigations of Flechsig and Wernicke that direct connections between the motor centres of the cortex and these basal ganglia do not exist. Moreover, it has been proved by numerous thoroughly reliable observations that destruction of the lenticular

13

or of the caudate nucleus does not necessarily give rise to a
motor paralysis.   One or both lenticular nuclei have repeatedly

Corpus callosum.
Sept. pelluc.
Nucl. caudat.
Caps. int.
Nucl. lentif.
Claustrum.
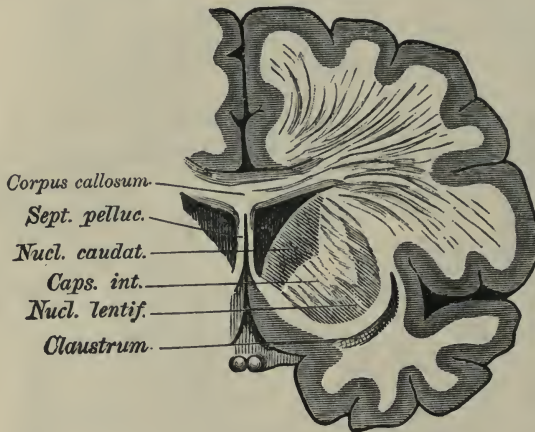
Fig. 54.

been found destroyed in cases in which there was no sign of
paralysis (Lépine, Nothnagel).   In order that this may not

Nucl. caud.

Corpus callosum
Crura forn.
Thalam.
Insula
Caps. int.
Nucl. lentiform.
Caps. ext.
Claustr.
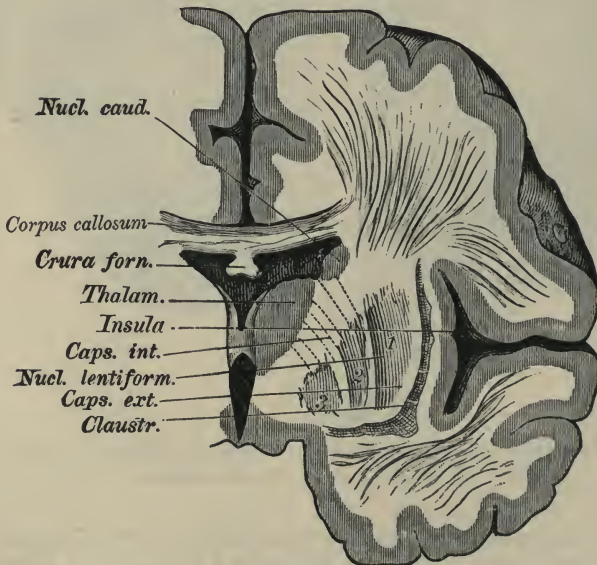
Fig. 55.

ensue, it is only necessary that the internal and perhaps also
the outer capsule remain intact.   As soon as the former (the

Nucl. caud.

Fornix.

Thalamus

Commissura mollis (media)
Nucl. tegment. (red nucleus)
Subst. nigr.
Pes pedunculi

Gyr. Hippoc.
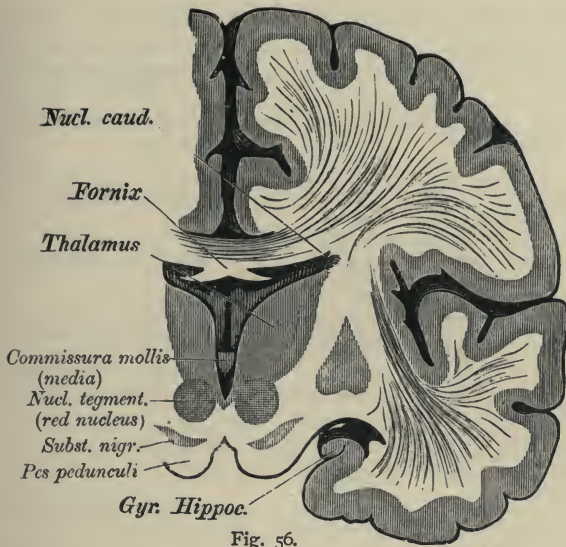
Fig. 56.

inner) capsule is either directly or indirectly implicated, we have a hemiplegia which is either transient or persistent, according to the nature of the lesion in the capsule. Whether the lenticular or the caudate nucleus alone is diseased can not be determined from the symptoms.

There is no doubt but that lesions of the thalamus, especially of its anterior and middle part, may occur without symptoms, and it is impossible to say whether motor paralysis is ever produced by lesions of the thalamus, for in all instances where this may have been the case the motor paralysis may also have been a result of damage to neighboring parts (peduncles, internal capsule).

Better founded is the idea that lesions of the pulvinar, the posterior part of the thalamus, give rise to defects in sight — crossed amblyopia or homonymous bilateral hemianopia — but the possibility that the

FIBRES OF THE CORONA RADIATA GOING TO THE OPTIC THALAMUS

TEGMENTAL RADIATION

CORP. CALLOS.

NUCL. LENTIF.

CAPS. INT.

THALAM

TO THE FILLET

COMMIS. ANT.

FORNIX

N. OPTIC

LOBUS TEMP.

ANSA LENTICULARIS.

INF. PEDICLE OF THALAMUS.
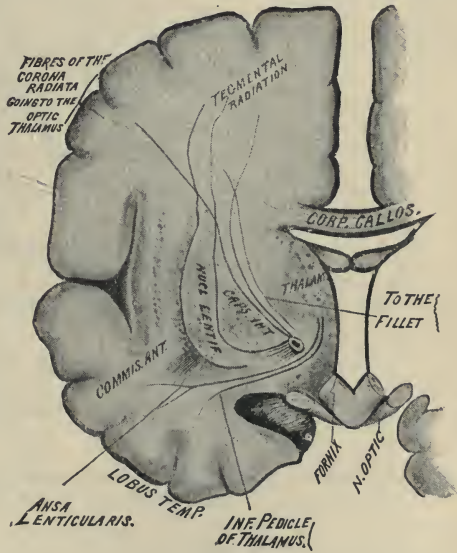
Fig. 57.

Figs. 54–57. — So-called "Frontal Sections" through the Brain. Fig. 54, through the anterior commissure. Fig. 55, in front of the middle commissure. Fig. 56, behind the middle commissure. Fig. 57, immediately behind the chiasm. The radiating fibres are shown diagrammatically in the last illustration. (After EDINGER.)

PITRES–NOTHNAGEL'S FRONTAL SECTIONS.

Designation.	Points where sections are made.	Called by Pitres—	Comprises, according to Nothnagel—
A	Immediately in front of genu of corpus callosum.	Coupe préfrontale.	Centri ovalis pars frontalis anterior.
B	Starting at the beginning of fissure of Sylvius.	Coupe pédiculo-frontale.	Pars frontalis media.
B₁	Between anterior central and frontal convolutions.		Pars frontalis posterior.
C	Through the fissure of Rolando.	Coupe frontale.	Pars centralis anterior.
D	Through ascending parietal convolution.	Coupe pariétale.	Pars centralis posterior.
E	Through parietal lobe 3 cm. posterior to the fissure of Rolando.	Coupe pédiculo-pariétale.	Pars parietalis.
F	Through occipital lobe.	Coupe occipitale.	Pars occipitalis.
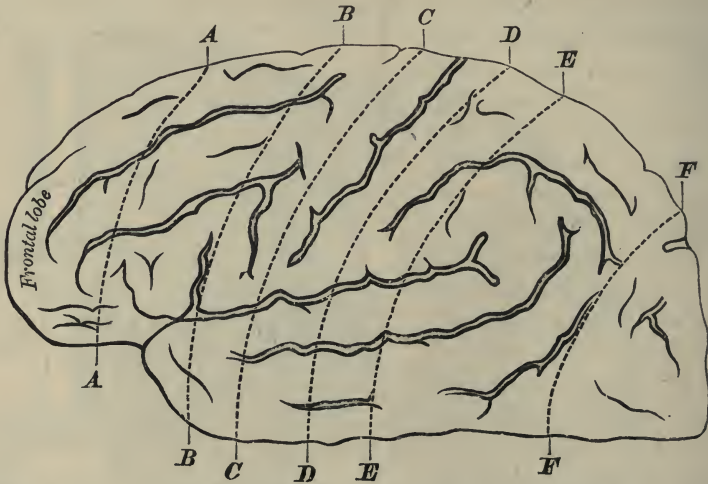
Fig. 58.—POINTS AT WHICH THE PITRES-NOTHNAGEL'S SECTIONS ARE MADE. They all run parallel with the fissure of Rolando.

posterior part of the optic tract is interrupted can even then not be excluded. The athetoid movements and symptoms of motor irritation (hemichorea, posthemiplegic tremor, athetosis) are, even if a connection actually exists between them and lesions of the thalamus (Greif, cf. lit.), certainly not characteristic of such lesions. The same holds good for the disturb-

ances in the muscular sense which have been observed in diseases of the thalamus (Meynert, Jackson). The relation finally between these latter and loss of the movements of facial expres-

Section A.
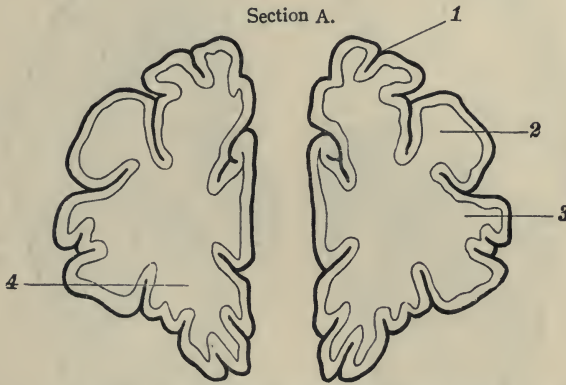
Fig. 59.—1, 2, 3, first, second, and third frontal convolutions. 4, præfrontal fasciculus of the centrum semiovale.

sion in the course of central facial paralysis (von Bechterew) has been spoken of in Part II, Chapter V.

Lesions of the internal capsule produce symptoms, varying

Section B. (B₁ Nothnagel.)
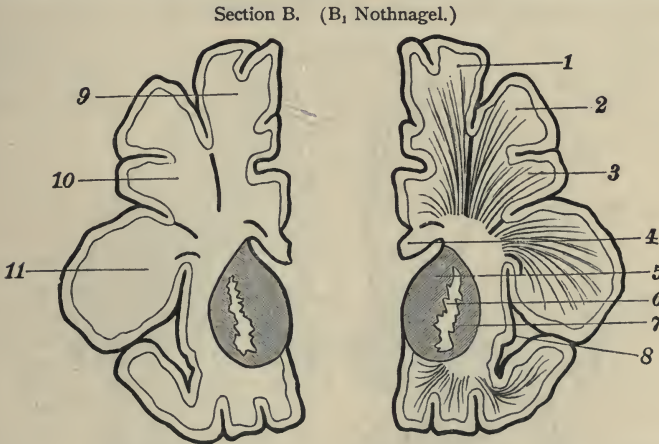
Fig. 60.—1 and 2, first and second frontal convolutions. 3, middle pediculo-frontal fasciculus. 4, corpus callosum. 5, nucleus caudatus. 6, internal capsule. 7, lenticular nucleus. 8, island of Reil. 9, 10, 11, superior, middle, and inferior frontal fasciculi.

according as the anterior or posterior limb is attacked. Pure capsular lesions—i. e., those in which the caudate as well as the lenticular nucleus remain intact—have rarely if ever occurred.

Section C.
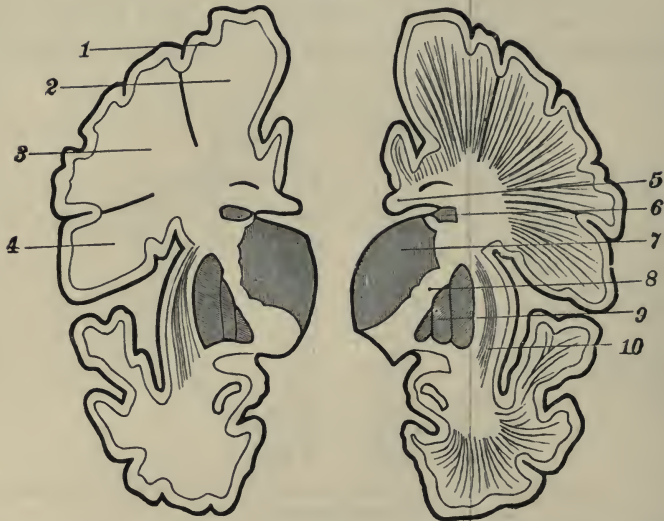
Fig. 61.—1, first frontal convolution. 2, 3, 4, superior, middle, and inferior frontal fasciculi. 5, corpus callosum. 6, nucleus caudatus. 7, optic thalamus. 8, internal capsule. 9, lenticular nucleus. 10, claustrum.

Fissures have been occasionally known to occur without having necessarily produced any motor disturbances in life (cf. Noth-

Section D.
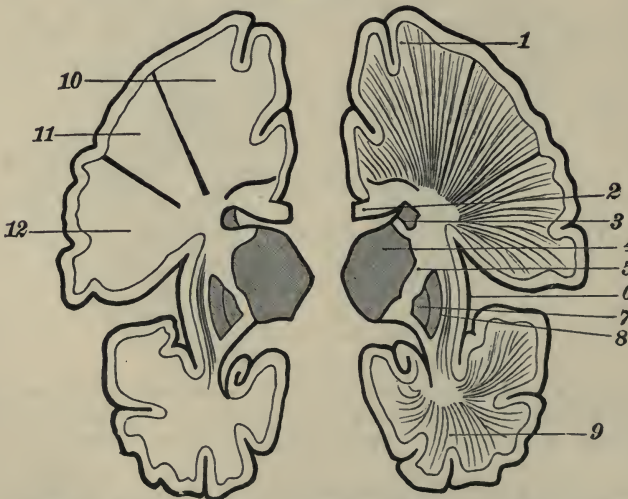
Fig. 62.—1, anterior central convolution. 2, corpus callosum. 3, caudate nucleus. 4, optic thalamus. 5, internal capsule. 6, island of Reil. 7, lenticular nucleus. 8, external capsule. 9, temporal fasciculus. 10, 11, 12, superior, middle, inferior parietal fasciculi.

nagel, *loc. cit.*, page 272). The functions of the anterior limb of the internal capsule are obscure, and lesions of this part do not produce any symptoms. With the posterior limb we are better

Section E.

Fig. 63.—1, superior parietal fasciculus. 2, corpus callosum. 3, inferior parietal fasciculus. 4 and 6, caudate nucleus. 5, optic thalamus.

acquainted, and, above all, is this one fact well established, that a lesion of the anterior two thirds of the posterior limb gives rise to the usual typical hemiplegia, with paralysis of the

Section F.

Fig. 64.—1, occipital convolutions. 2, occipital fasciculus of the centrum semiovale.
Figs. 59–64.—PITRES-NOTHNAGEL'S SECTIONS, the position of which is made clear by the table on p. 196.

lower facial branches. A very small lesion at the knee may produce an isolated facial paralysis. If the posterior portion of the anterior two thirds is the chief seat of the disease, the

paralysis is best marked in the leg.   The posterior third of the
posterior limb is occupied by the sensory fibres (*le carrefour
sensitif* of Charcot), and lesions of that region cause a loss of
sensation on the opposite side of the body (" hemianæsthesia "),
in which often the nerves of special sense are implicated, and
hearing, smell, and taste (on the anæsthetic side) are, if not lost,
at least diminished.   Often hemiplegia is accompanied by hemi-
anæsthesia, because if the one portion of the capsule is affected,
an indirect and transitory implication of the other may occur.
Usually such a hemianæsthesia soon disappears in the same
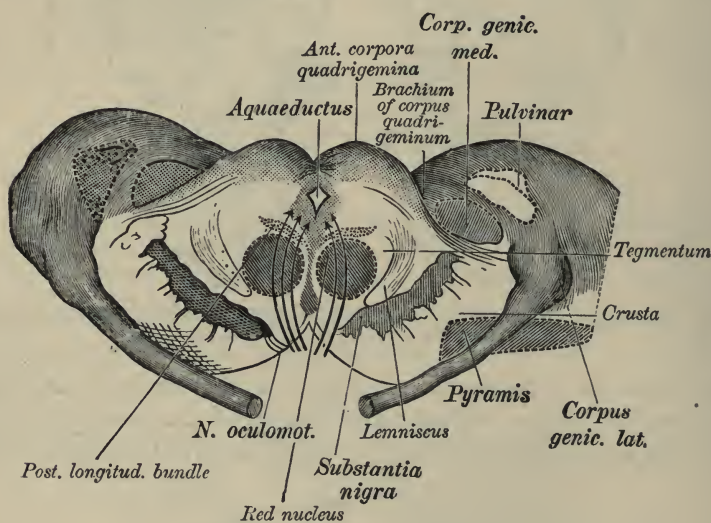
Fig. 65.—DIAGRAMMATIC CROSS-SECTION THROUGH THE ANTERIOR CORPORA QUADRI-
GEMINA.   (After EDINGER.)

way as the indirect motor disturbance often soon passes off in
cases of persistent hemianæsthesia.   Whether the symptoms of
motor irritation (the so-called post-hemiplegic chorea, for in-
stance), which are a not rare accompaniment of hemiplegia,
are due to disease of the internal capsule or to disease of the
neighboring basal ganglia is as yet undecided.

By the corpora quadrigemina we mean that peculiar eminence
which by a crucial furrow is separated into four parts (bodies), and
forms the posterior boundary of the third ventricle.   In front it is
bounded by the commissure which unites the two thalami; on it
rests the pineal gland (conarium).   The anterior pair of bodies,

which are called the nates, are larger than the posterior, the testes. The appearance and structure, which these two pairs of bodies present in the lower mammals, justifies the conclusion that they are totally different from each other. Above the corpora quadrigemina is situated the splenium of the corpus callosum; between the two is the transverse fissure of Bichat (the fissura choroidea).

The frontal section through the anterior pair of the corpora quadrigemina (Fig. 65) shows the three divisions—the crusta, tegmentum, and quadrigeminal ganglia. Toward the outer side is the
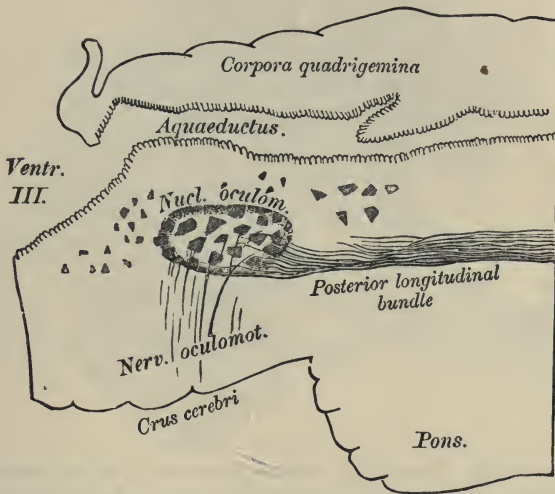
Fig. 66.—Longitudinal Section through the Region of the Corpora Quadrigemina of a Human Fœtus Twenty-eight Weeks Old. (After Edinger.) Shows that the posterior longitudinal bundle terminates in the nucleus of the oculo-motor nerve.

pulvinar, with the lateral geniculate body. Emerging below the pulvinar is the crus, which contains the pyramidal tract (cf. page 188). Between it and the tegmentum, in which is seen the red nucleus, is situated the substantia nigra. Below the aqueduct are the root fibres of the motor oculi, and in characteristic transverse section the posterior longitudinal bundle. The position of the latter is made still clearer in the longitudinal section represented in Fig. 66.

The manner in which the fibres from the red nucleus pass under the posterior pair of the corpora quadrigemina toward the middle line and then decussate with the fibres of the opposite side—the so-called " crossing of the brachia conjunctiva " (sup. peduncles of the cerebellum)—is represented in Fig. 67.

Isolated lesions of the corpora quadrigemina are almost as rare as similar lesions of the capsule; nearly always neighboring structures are implicated. The data which we possess in this connection seem to indicate that lesions of the anterior pair produce visual disturbances, amblyopia, amaurosis, and loss of pupillary reaction. Physiologically important is the fact that a root going to the optic tract is given off
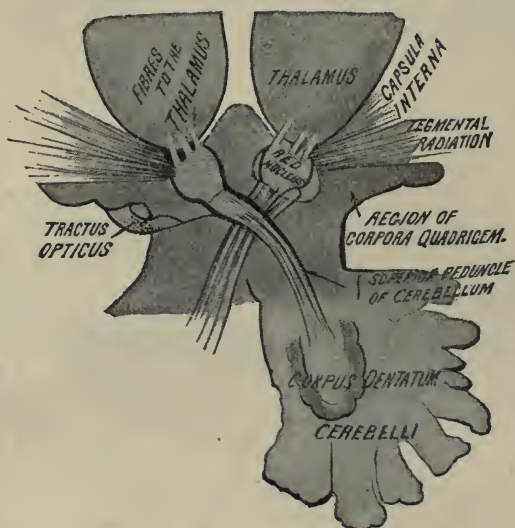
Fig. 67.—Diagrammatic Horizontal Section through the Decussation of the Superior Peduncles of the Cerebellum. (After Edinger.)

from this anterior pair, and that radiating fibres pass to the nucleus of the third nerve, so that a connection exists between stimulation of the optic nerve and stimulation of the oculo-motor (pupillary reflex) (Mendel). Authors seem to differ, however, about the extent to which this reflex is influenced by disease of the anterior pair of the corpora quadrigemina. Impairment of certain movements of the eyes, especially the upward motion of the ball, has been repeatedly noted by competent observers (Gowers). Nothnagel assumes that a lesion of the same oculo-motor branches on both sides, without the existence of an alternating paralysis of the extremities, speaks for a lesion of the corpora quadrigemina (cf. *loc. cit.*, p. 220). As to the function of the posterior pair of the corpora quadrigemina, all explanations are uncertain and hypo-

thetical. Baginsky assumed them to have a similar signifi-
cance for the ear as the anterior pair for the eye, an idea in
support of which further evidence is needed, and the disturb-
ance of equilibrium which has been ascribed to disease of
these bodies may well be produced by pressure upon the
neighboring vermiform process of the cerebellum. On this
point nothing positive is known.

The crura cerebri emerge from the pons Varolii as two thick
cylindrical white bundles of fibres; on leaving it they diverge,
having between them the posterior perforated space and the
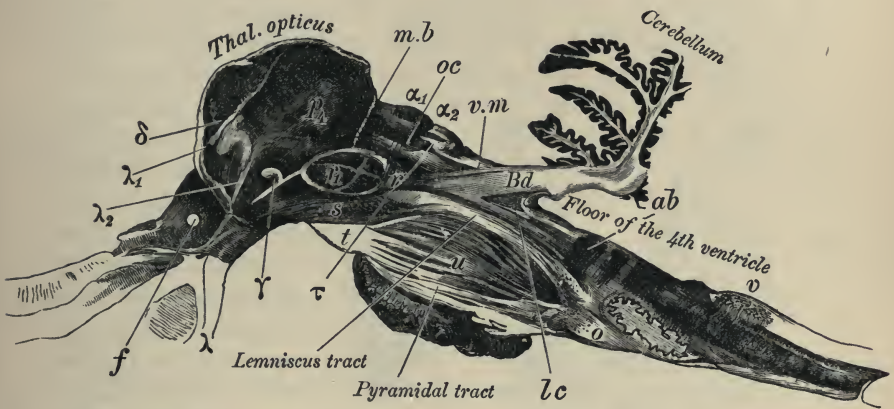
Fig. 68.—SAGITTAL SECTION THROUGH PONS AND MEDULLA OBLONGATA. (After MEN-
DEL.) *f*, anterior commissure. *p₄*, pulvinar. *s*, substantia nigra. *r*, tegmentum of
crus cerebri. *r₁*, red nucleus. *t*, pes pedunculi. *u*, pons. *v*, hypoglossus nucleus with
fibres emerging from it. *a₁*, corpus quadrigeminum anterius. *a₂*, corpus quadrigemi-
num posterius. *γ*, ansa lenticularis. *δ*, Vicq-d'Azyr's bundle. *λ*, optic tract. *λ₁*,
external thalamus-root of optic tract. *λ₂*, internal thalamus-root of optic tract. *v*,
olivary body. *o*, anterior pyramid. *τ*, posterior longitudinal bundle. *lc*, locus cæru-
leus. *v. m*, valve of Vieussens. *m. b*, Meynert's bundle. *ab*, abducens nucleus with
emerging fibres. *Bd*, superior peduncle of cerebellum. *oc*, oculo-motor nucleus with
emerging fibres.

corpora albicantia (mammillaria s. candicantia). The situation
of the crusta and the tegmentum (Fig. 69), and the masses of
fibres contained in them, is once more shown in Fig. 68, which
represents a longitudinal, sagittal section made almost in the
middle line (*r*, tegmentum; *s*, substantia nigra; *t*, crusta). That
the crusta forms the path for the voluntary, the tegmentum the
path for the reflex movements, and that the latter also contains
the sensory paths, as Meynert assumes, has not yet been proved
by physiology. That the crusta, however, contains the motor

path—namely, the pyramidal tracts—is a fact established beyond doubt; hence its lesions will for the present be of more practical interest. Only a small number of instances of lesions in the tegmentum have been reported. A case of Buss (cf. lit.) presented ataxia of all four extremities, anæsthesias, disturbances of the muscular sense, and an affection of the right hypoglossus. At the autopsy a focal lesion was found in the tegmentum of the crus and the pons.

Considering the relation which the third nerve bears to the median part of the crus cerebri, as is shown in Fig. 69, we can well understand that in lesions of the latter the oculo-motor is not rarely implicated, and autopsies have frequently demonstrated that, wherever an oculomotor paralysis has been associated with paralysis of the extremities on the opposite side, the lesion is situated in the crus cerebri. For example, in a patient with oculo-motor paralysis of the right side and hemiplegia of the left side (if both come on at the same time!), we
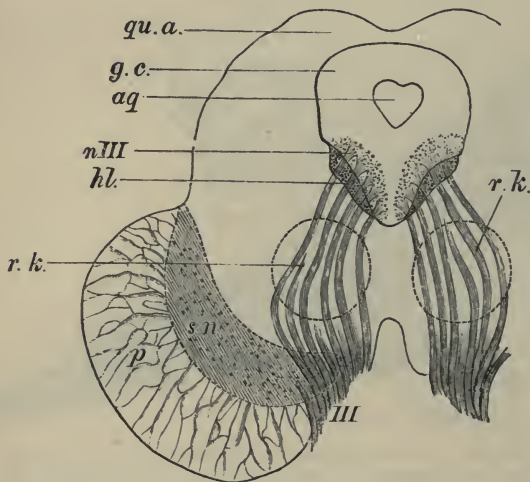
Fig. 69.—Cross Section through the Region of the Ant. Corpora Quadrigemina.

qu.a. anterior corpora quadrigemina ; *g.c.* gray matter around the aqueduct of Sylvius ; *aq*, aqueduct of Sylvius ; *nIII* nucleus of the third nerve ; *hl.* posterior longitudinal bundle ; *r. k.* red nucleus (tegmentum) ; *sn*, substantia nigra (locus niger) ; *p*, cerebral peduncle.

may without hesitation diagnosticate a focal lesion in the right crus cerebri; if, in addition, anæsthesia exists on the paralyzed side, an implication of the tegmentum must be suspected.

The pons Varolii, which connects the two hemispheres of the cerebellum, contains, as we said above (cf. page 78), the nuclei for several nerves and the fibres passing from them to the brain. The nuclei, which are situated in the upper segment, are those of the fifth, the facial, and the abducens. Since the pons naturally also contains the motor fibres, situated, as

we said above, in the lower or ventral segment, while in its dorsal part one meets the sensory bundles, pontine lesions may produce a complication of symptoms as characteristic as those produced by lesions in the crus. As we have attempted to make clear in Fig. 70, the fibres of the facial nerve decussate higher up than the motor fibres of the pyramidal tracts. Keeping this fact in mind, we can easily understand that a lesion of the lower part of the pons concerns the facial fibres after their decussation, the fibres going to the extremities, however, before they cross, and consequently gives rise to a facial paralysis on the side of the lesion, but a paralysis of the extremities on the opposite side (hemiplegia alternans) (Gubler, 1859). A lesion of the upper part of the pons concerns both of these paths before their decussation, and produces, therefore, hemiplegia, with a facial paralysis of the same side, which, however, is distinguished from the typical hemiplegia in that the facial paralysis in our case resembles somewhat the peripheral type, as it takes in all three branches of the facial, and as, though but rarely, reaction of degeneration may be present (cf. page 84).

If, then, we meet with a paralysis which affects the facial on one, the extremities on the opposite side (alternating paralysis), simultaneously, we are justified in assuming the lesion to be situated in the pons, and more especially in its lower part. If paralysis first occurs in the face alone, and does not develop in the extremities until later, and if the whole process is gradual, it may arise from a tumor at the base of the brain. If, besides the symptoms described, the patient complains of pain in the face, the trigeminus is included in the lesion. A paralysis of the external rectus points to the implication of the abducens nerve, in which case a paresis of the internal rectus of the other side not rarely co-exists, so that a conjugate deviation
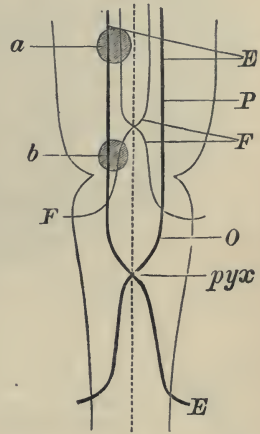
Fig. 70.—Diagram showing the Decussation of the Fibres going to the Extremities, and of those going to the Face, in the Pons and Medulla Oblongata. *F*, facial fibres. *E*, fibres going to the extremities. *P*, pons. *O*, medulla oblongata. *pyx*, decussation of the pyramidal tracts. *a*, a focus in the upper, *b*, a focus in the lower part of the pons (the latter is situated below the decussation of the facial fibres).

of the eyeballs toward the paralyzed side—that is, away from the focus—may occur.

Bilateral lesions of the pons must be thought of in combined paralyses of the extremities and cranial nerves, or in cases of bilateral facial paralysis or bilateral paralysis of the extremities (either of both legs or of all four extremities). The diagnosis, however, as a rule, can not be made with certainty.

Convulsions will be observed if by acute lesions the spasm centre, as Nothnagel calls it, becomes excited. Tonic spasms
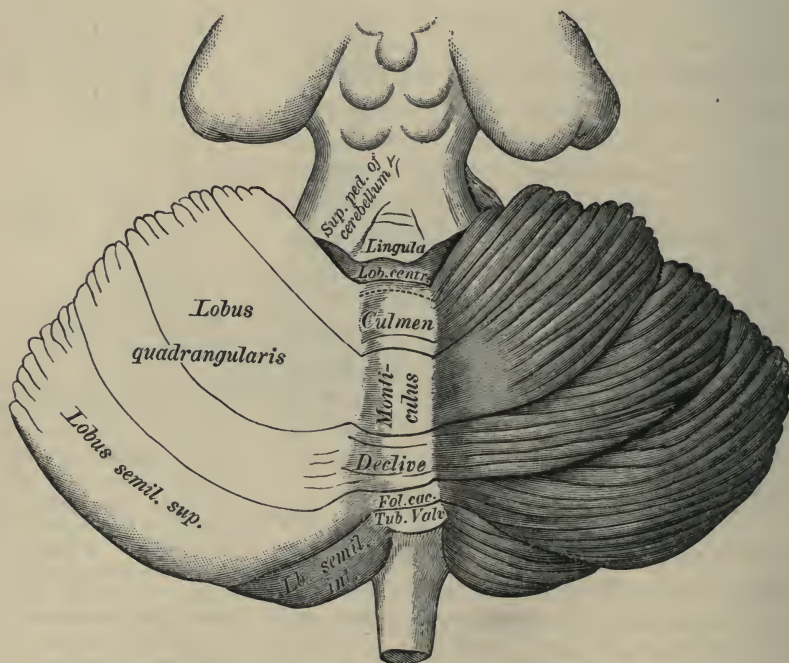
Fig. 71.—THE CEREBELLUM SEEN FROM BEHIND (dorsal aspect).

in the paralyzed limbs are not uncommon. Psychical changes, which occur in connection with lesions in the pons, are very irregular in their occurrence, and assume the most diversified forms. They deserve a more careful study than has as yet been devoted to them. Their entire absence has been repeatedly noted. Anæsthesias in the distribution of the trigeminus, as well as in the extremities, are comparatively frequent, but we are not at present able to utilize them for the purpose of topical diagnosis.

The small brain—cerebellum—is made up of a middle part, the so-called vermiform process, and two lateral parts, the hemispheres.
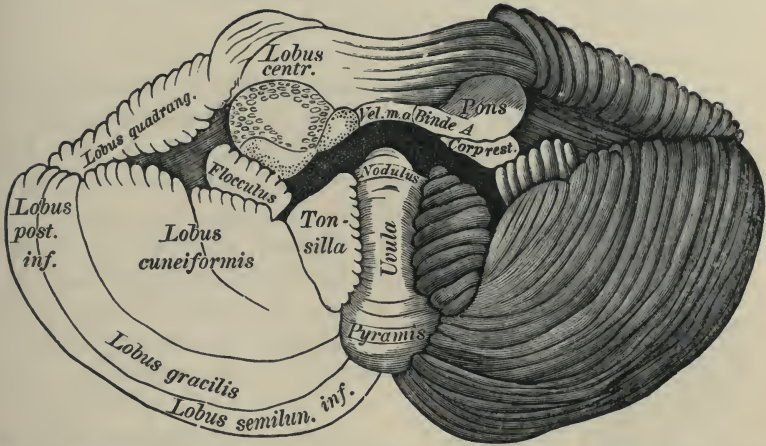
Fig. 72.—CEREBELLUM SEEN FROM IN FRONT (ventral aspect). *Vel. m. a.*, valve of Vieussens. *Binde A*, superior peduncle of cerebellum.

Fig. 71 represents the superior (the upper, posterior, dorsal aspect), Fig. 72 the inferior vermiform process (the lower, anterior, ventral
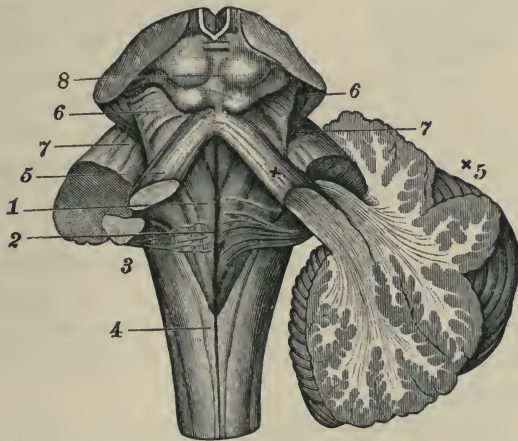
Fig. 73.—THE CONNECTIONS OF THE CEREBELLUM—with *a*, the midbrain (superior peduncles), 5; *b*, the pons (middle peduncles), 7; *c*, the medulla oblongata (inferior peduncles or restiform bodies), 3. 1, fourth ventricle. 2, striæ acusticæ. 4, funiculi graciles. 6, lemniscus. 8, corpora quadrigemina.

aspect), with the various portions of the hemispheres. The cerebellum is connected with the corpora quadrigemina by the superior

peduncles of the cerebellum, with the pons by the middle peduncles, the processus e cerebello ad pontem, and with the medulla oblongata by the inferior peduncles or restiform bodies. The situation of these arms or brachia is shown in Figs. 73 and 75.

To enable us to point with certainty to the cerebellum as the seat of disease, the implication of the vermiform process is necessary, since, as Nothnagel has pointed out, we may have extensive disease in the hemispheres without the manifestation of a single symptom during life. In the cases, however, in which the vermiform process is affected, marked disturbances of co-ordination and equilibrium ensue ; the patient staggers and complains of severe vertigo on walking and standing. This is almost a pathognomonic symptom, especially if it be associated with occasional spells of more or less serious vomiting, when the name " cerebellar " is not improperly applied to it. Of course we must also keep in mind, if such symptoms are remarked, that something similar occurs in other diseases, especially in Ménière's disease, and must therefore look for additional points before excluding these. This is, however, not always easy, and often

Fig. 74.—The Anterior (Lower) Surface of the Medulla Oblongata. With (1) the anterior pyramids ; (2) the decussation of the pyramids ; (4) the anterior, (3) the posterior nerve-roots (the anterior median fissure has been widened by drawing the pyramids apart).

enough—for instance, if vomiting occurs without vertigo— the diagnosis must remain open. If, on the other hand, the change in the walk of the person is so pronounced that it can be easily confounded with that of a drunken man, the question is easier to solve.

Acute lesions of the middle peduncles of the cerebellum produce highly characteristic symptoms, so that a diagnosis can be made with a fair amount of certainty. The body is involuntarily gyrated around its longitudinal axis (" forced movement "). This symptom, however, can only be observed as a consequence of irritation of the peduncles, but is absent if the latter are wholly destroyed—*e. g.*, by hæmorrhage. Sometimes

the patient has an irresistible inclination to lie on one side, and this is, if the remaining symptoms point in the same direction, also to be estimated as a forced movement, or rather a "forced position." It is not uncommonly accompanied by a corresponding twist of the head and eyeballs. This phenomenon, however, is not a pathognomonic symptom for lesions of the middle peduncles. The direction in which the body is turned is sometimes toward the diseased side, sometimes away from it, a fact for which no explanation has as yet been found.

For lesions of the other peduncles of the cerebellum (the superior and inferior) no diagnostic points are known.

The lowest part of the encephalon is called the medulla oblongata. It is continuous below with the spinal cord on a level with the lower margin of the foramen magnum. On its anterior (lower, ventral) aspect we observe (Fig. 74) the pyramids with their decussation, the olives with the nucleus dentatus (Fig. 19), and to the outer side of these are to be found the restiform bodies, the inferior peduncles of the cere-

Fig. 75.—THE POSTERIOR (DORSAL) ASPECT OF THE MEDULLA OBLONGATA. 1, posterior commissure. 2, peduncle of pineal gland. 3, corpora quadrigemina. 4, superior peduncle. 5, middle peduncle. 6, inferior peduncle of cerebellum. 7, striæ acusticæ. 8, funic. teres. 9, obex. 10, funiculi graciles.

bellum. The latter contain the so-called direct cerebellar tracts, which, coming from the outermost portion of the lateral columns in the cord, pass, through the anterior commissure of the vermiform process, to the cortex of the cerebellum. We shall later on have to refer to these tracts again. That a relation exists between the olives and the cerebellum is apparent from the fact that wherever we have a congenital atrophy of the cerebellum these bodies are also atrophic (Flechsig).

On the posterior (dorsal, upper) aspect is the floor of the fourth ventricle, the fovea rhomboidalis (Fig. 75), which is bounded below by the diverging restiform bodies, above by the diverging superior

peduncles of the cerebellum. The median columns are called the posterior pyramids (funiculi graciles). They are the continuations of Goll's columns of the spinal cord. To the tracts situated to the outer side of these the name funiculi cuneati, or Burdach's columns, has been given.

To diagnosticate the medulla oblongata as the seat of a lesion is only possible if the nuclei in the floor of the fourth ventricle are diseased, in which case we get the clinical picture of the bulbar paralysis described on page 154. Other characteristic symptoms do not exist, and more especially it must not be forgotten that foci in the medulla may give rise to a paralysis only in the extremities, which presents nothing characteristic during life. If, however, the nerve nuclei of the medulla are implicated, a characteristic picture is presented which can hardly be mistaken. Another point of which, in making our topical diagnosis, we must not lose sight, is the fact that certain brain lesions may give rise to a similar combination of symptoms constituting the clinical picture of the disease which we have described above as pseudo-bulbar paralysis. Other diseases of the medulla—traumatism, acute and gradual compression, hæmorrhage, and embolism—are of no practical significance, since they cause death so quickly that a certain diagnosis is impossible. Hence we will pass them over without further remark. Of the tumors observed in the medulla oblongata we shall speak later.

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II. The Study of Cerebral Lesions with Reference to their Pathological Nature.

Pathological Diagnosis.—We have before pointed out that the question as to the nature of a brain disease is not only of interest to the physician, but of the greatest importance to the patient, as on this the prognosis as well as the mode of treatment turns. An error in the topical diagnosis may deserve the censure of scientific criticism, but does not necessarily entail damage to the patient. If, on the other hand, we mistake the nature of the lesion in a given case—if, for instance, a disease of the vessels is taken for a new growth, if the tuberculous or syphilitic nature of the affection is overlooked, or, again, a severe alcoholic intoxication is diagnosticated where in reality an apoplexy exists—when such errors have influenced the treatment, not only opportunities may be lost for the patient which may never present themselves again, but an unfavorable event of the disease may actually be brought about or at least precipitated. On these grounds we ought to be particularly careful and conscientious in forming this part of our diagnosis, and no symptom, however small it may seem, should be overlooked, as we never know but that it may later perhaps become of diagnostic value.

In looking over the several pathological processes which here concern us, we find that their number is comparatively limited. First of all, we shall devote our attention to diseases of the blood-vessels, which so frequently are the cause of cerebral lesions. We shall have to determine the nature of these diseases and carefully distinguish the affections of the blood-vessels from the secondary changes produced by them. The clinical symptoms, the complaints of the patient, and the objective signs are a direct consequence of the latter only, and it is therefore not the disease of the blood-vessels which we
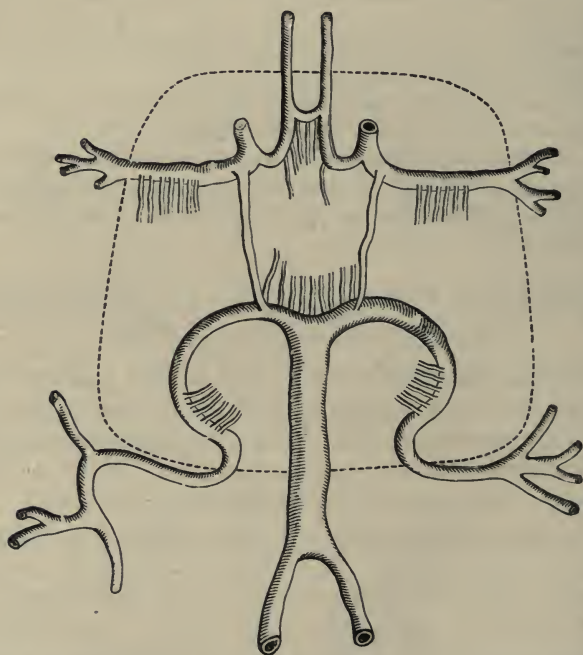
Fig. 76.—DIAGRAM SHOWING THE CIRCLE OF WILLIS. The carotids with the anterior and middle cerebral arteries and the basilar with the posterior cerebrals are connected by communicating branches.

have practically to deal with, but the changes in the brain substance which they entail. The clinical manifestations vary according to the seat of the diseased vessel and the portion of the brain supplied by it. The symptoms we shall describe in detail later; but first let us speak of the pathological nature of the diseases of the cerebral vessels.

AFFECTIONS OF THE BRAIN DUE TO DISEASE OF THE
BLOOD-VESSELS.

A. **Diseases of the Cerebral Vessels and their Conse-
quences.**—The arteries of the brain are derived from the internal
carotids and the basilar, which is formed by the two vertebrals.
The internal carotid gives off two terminal branches, the anterior
cerebral (arter. corpor. callos.) and the middle cerebral (arter. foss.
Sylv.). The basilar divides into the two posterior cerebrals (arter.
profund. cerebri). These receive on each side a communicating
branch from the internal carotid, the so-called posterior communi-
cating artery, and the two anterior cerebrals are connected by an
anterior communicating branch, so that a closed circle (or rather a

Fig. 77.—THE CORTICAL DISTRIBUTION OF THE MIDDLE CEREBRAL ARTERY. (After
CHARCOT.) From left to right the five branches are named as follows : The inferior
frontal branch to Broca's convolution, the ascending frontal branch, the ascending parie-
tal branch, the parieto-sphenoidal, and the sphenoidal branches.

heptagon, according to Hyrtl) of arteries is formed, known as the
circle of Willis, an arrangement which is of the last importance for
the distribution of the blood in the brain (cf. Fig. 76).

The fact that the left carotid comes off from the aorta nearly in
a straight line with the blood-current in the arch, while the innomi-
nate, which gives off the right carotid, leaves the aorta almost at
right angles, easily explains the greater frequency of embolism on
the left side. A somewhat similar condition exists in the vertebrals,
where the left, often the larger one, arises from the subclavian at its
highest point. This is, however, of less moment for cerebral lesions,

as the blood has first to pass the basilar before entering the brain substance.

Of the three before mentioned arteries—the anterior, middle, and posterior cerebrals—each one supplies two sets of vessels totally distinct from each other—namely, first, the so-called cortical arteries; secondly, the arteries of the basal ganglia. The important difference between these two systems consists in the fact that the former, as Heubner and Duret have shown, possess anastomoses, while the latter are, as they have been called by Cohnheim, terminal arteries—that is. they do not communicate with each other, but pass directly into the capillaries. The significance of such an arrangement is ap-

Fig. 78.—Frontal Section through the Cerebral Hemispheres, One Centimetre behind the Chiasm. Shows the distribution of the middle cerebral artery in the lenticular nucleus.

parent, and we shall not be surprised to find that occlusion of an artery of the second set almost always produces death of the parts supplied by it.

Of the three cerebral arteries, the middle, the Sylvian artery, has by far the widest distribution and is the most important; for while the anterior supplies the corpos callosum, the gyrus rectus, the paracentral lobule, and the præcuneus; the posterior, the crus, the temporal, and the occipital lobe, and the cuneus, sending also a few branches to the optic thalamus (arter. optic. posterior.), it remains for the middle cerebral to supply the whole lenticular and the caudate nucleus, and, above all, the internal capsule. Moreover, the central and cortical motor region, the cortical areas concerned in the process of speech (on the left side), the cortical centre for hearing, probably also for vision, depend on this artery for their nutrition.

Its cortical distribution, its subdivision into the frontal, parie-

tal, parieto-sphenoidal, and sphenoidal arteries, is made clear by Fig. 77.

Its distribution to the lenticular nucleus is illustrated in Fig. 78. The internal artery of the corpus striatum, also called the lenticular artery, goes to the first and second segment of the lenticular nucleus, while the external branches are the so-called lenticulo-striate and lenticulo-optic arteries. Among the former, the one which supplies the third segment of the lenticular nucleus, the upper portion of the internal capsule, and the caudate nucleus deserves special mention. It is so frequently the seat of hæmorrhage that Charcot has called it "*l'artère de l'hémorrahagie cérébrale.*"

The 'tween-brain and the mid-brain are mostly supplied by the posterior communicating and its branches, the cerebellum by several so-called cerebellar branches (arter. cerebell. super. et infer.) coming from the vertebrals; the pons and medulla oblongata also by branches of the vertebrals, which are the so-called rami ad pontem and rami ad medullam oblongatam. For details the reader is referred to Duret.

The internal carotid and the basilar measure 4 mm. in diameter; the vertebrals, 3.5 mm. (Luschka). The blood pressure in the carotid is generally taken to correspond to from 140 to 160 mm. Hg. How guarded, however, we ought to be in accepting such statements has been shown by Loewenfeld, who drew attention to the variations in the development of the cerebral arteries; and it seems at least possible that this is of considerable ætiological significance for different cerebral affections (Loewenfeld, Arch. f. Psych. und Nervenkrankh., Bd. xviii, 3, page 819, 1887).

1. *Cerebral Hæmorrhage, Hæmorrhagia Cerebri* (*Periarteriitis Cerebralis, Miliary Aneurisms of the Cerebral Arteries*).

Pathological Anatomy and Ætiology.

Of all cerebral affections, hæmorrhage, the result of the rupture of a vessel, is by far the most important and the most frequent. As we should expect, hæmorrhages of various kinds may be produced by traumatism (injury to the skull, with or without fracture). They may occur between the inner side of the skull and the loosened dura mater, or in the sac of the dura or that of the pia (submeningeal hæmorrhage); but, disregarding these, there is one affection especially which gives rise to cerebral hæmorrhage—namely, a diffuse periarteriitis—which was first described by Charcot and Bouchard in 1868. In this process a thickening of the lymph-sheaths, and consequent changes in

the muscularis take place, by which the formation of miliary aneurisms is favored (cf. Figs. 79 and 80). Rupture of these aneurisms then gives rise to hæmorrhages, and so frequently is this the case that the authors before mentioned found this
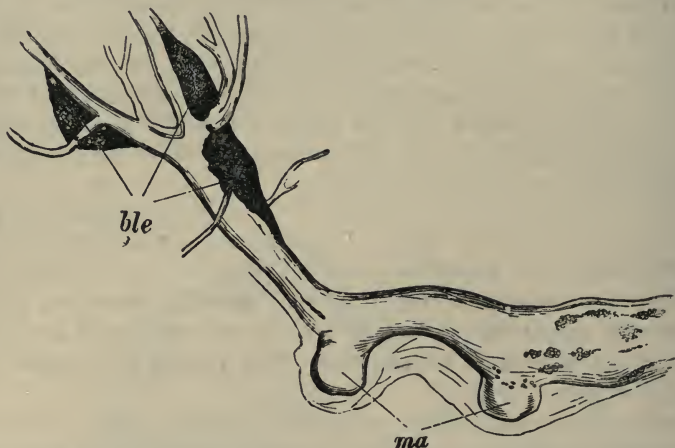
Fig. 79.—Cerebral Artery from an Apoplectic Focus. *ma*, miliary aneurism. *ble*, extravasations of blood into the adventitial lymph space. (After Cornil and Ranvier.)

condition in every one of seventy-seven consecutive cases which came under their observation.

For the rupture of these aneurisms it is by no means always necessary to have any extraordinary exciting cause, such as an elevation of the blood pressure, which may temporarily be produced by bodily exertion, sneezing, coughing, vomiting, and the like, or which may permanently exist where the heart is hypertrophied, as in valvular disease or in cases of contracted kidney. In many of the instances nothing of the kind can be demonstrated.

Fig. 80.—Miliary Aneurism of a Small Artery of the Lenticular Nucleus. (After Marchand.)

The size of the aneurism varies from 0.2 to 1 mm.; their color and consistence often differ greatly. Their favorite seat is in the thalamus, the corpus striatum, the convolutions, and the pons, while they are less frequently met with in the centrum ovale, the crura, and the medulla oblongata. Sometimes only two or three, at other times as many as several hundred,

have been detected in one brain. On being squeezed they are found to contain white corpuscles, fat droplets, and amorphous granular masses.

The hæmorrhage which is produced by their rupture consists when fresh of a dark-colored loose coagulum. The wall of the "focus" is red and spotted with punctiform hæmorrhages (capillary apoplexies), and presents a ragged and torn appearance. Gradually the dark color becomes lighter, the neighboring parts become infiltrated, yellowish, and very soft (lemon-colored œdema). As a rule, the focus becomes later encapsuled by a layer of neuroglia, the fibrin masses become mixed with the *débris* of the nerve elements, and we get a smooth-walled cavity with liquid contents, the so-called apoplectic cyst, occupying a smaller space than the original hæmorrhage. If the walls approach each other before the coagulum is transformed, a great increase of fibrillated connective tissue takes place and we get a so-called apoplectic scar.

The effect of the hæmorrhage varies according to its position, according to the calibre of the ruptured vessel, upon which depends its amount, and according to the rapidity or slowness with which the blood escapes. The favorite seat for hæmorrhages is in the large ganglia (Charcot, Andral, Rochoux) ; with decreasing frequency they are found (Nothnagel) in the remaining portions of the cerebral hemispheres, much more rarely in the pons and the cerebellum. The frequency is directly influenced by the size of the different cerebral vessels and by the blood pressure. The diameter of the vessels of the brain stem is considerably larger than that of those going to the cortex. The above-mentioned "artery of cerebral hæmorrhage" is of an especially large calibre ($1\frac{1}{2}$ to 2 mm.), and causes therefore when it bursts a particularly large and extensive hæmorrhage, because the bleeding is prolonged. "The traumatic effect of the hæmorrhage," as Wernicke calls it, is equal to the product of the mass of effused blood into the square of the rapidity with which it is poured out, which latter depends directly upon the blood pressure in the vessels. Hence it follows that, as regards the effect of a hæmorrhage, the blood pressure is of more importance than the calibre of the vessel.

Ætiology.—In examining into the ætiological factors concerned in a cerebral hæmorrhage, we must distinguish those

which produce the disease of the vessels from those which directly cause the hæmorrhage ; in other words, the predisposing from the exciting causes.

About the former not much is known ; nevertheless, considerable influence in the causation of arterial disease must be ascribed to age, as we can not deny that it is decidedly less frequently to be observed in the young than in older persons, and that the smallest percentage of apoplexies is found between the fifth and thirtieth years of life. Still, to lay so very much stress upon the significance of age is not warranted by experience. The fact that cerebral hæmorrhage is by no means rare in people from twenty to thirty years old clearly shows that miliary aneurisms may occur even at a comparatively early period of life, nor are these cases by any means always those of persons laboring under hereditary disadvantages, since even members of perfectly healthy families who are yet young may fall victims to a stroke of apoplexy. The influence of heredity as well as that of age has undoubtedly been overrated in this connection. It is true there are families in which apoplexy seems to be a natural occurrence, but such instances are exceptional, while on the other hand the arterial disease develops in an infinitely larger proportion of cases apparently without special hereditary cause. Sometimes the development of the disease seems to be favored by a peculiar " habitus." So, corpulent individuals of medium height, with short necks, broad thoraces, who on the least exertion or excitement become purple in the face, have usually been looked upon as particularly predisposed to apoplexy, and in many cases with justice, yet those who have in an extensive practice seen how often tall, spare individuals with narrow chests die from cerebral hæmorrhage, will readily give up the idea that an apoplectic habitus is a *conditio sine quâ non.*

The *rôle* which sex plays can not be denied. The disease is much more frequently observed in males than in females, while with embolism, as we shall see, the reverse is true. To explain this predisposition in males, other factors—namely, the mode of life—must, I think, be taken into account, and here it is, in the first place, the occupation, and, secondly, the abuse of alcohol, which must be considered. Notwithstanding the fact that we know very little about the influence of occupation on the formation of miliary aneurisms, our statistics of fatal cases of cerebral hæmorrhage in the different trades being somewhat unre-

liable, still we have some sure grounds, the correctness of which can scarcely be called in question. That, for instance, the working in certain poisons, especially in lead, predisposes to arterial disease, and consequently to apoplexy, is indisputable. In his thesis on encephalopathia and arthralgia saturnina, prepared under my auspices, Schulz (Breslau, 1885) points out the frequency of the so-called hemiplegia saturnina, and calls attention to the fact that Berger has made similar observations. Later on, in our consideration of lead poisoning, we shall have to refer again to this subject, and discuss the probable action of the metal on the nervous system. In the second place, those who are exposed to radiating heat—workers at furnaces, puddlers—are in danger, especially if their work is connected with much bodily exertion, and this can hardly surprise us if we remember how much circulatory disturbances are favored by such circumstances. The same may be said of occupations which necessitate uncomfortable positions of the body, as, for instance, is the case in agate polishers, who constantly have to lie on their abdomens, or in coal miners, who have to remain in a stooping position all the time.

In regard to the abuse of alcohol we refer not only to the confirmed drunkards, but much rather to that class of individuals who habitually consume more alcohol, especially beer, than is good for them. Such men rarely, if ever, get drunk, but they drink several times a day one or two glasses of beer, do not take enough exercise, and become fat and predisposed to fatty heart and arterial disease, especially arterio-sclerosis, which affection, we may say finally, is the real cause of the greater frequency with which apoplexy is met with in men than in women. The fatty heart may be present even without any marked obesity.

The important influence of syphilis in the origin of cerebral hæmorrhage is proved by many irrefutable observations, and, considering the part played by it in disease of the cerebral vessels, this can easily be explained. We shall mention it again in this chapter, and later dwell more particularly on the symptoms peculiar to the syphilitic hemiplegia. Exceptionally, hemiplegia occurs after diphtheria, sometimes in conjunction with a paralysis of the palate, sometimes independently. In a girl aged fifteen under my care, hemiplegia developed fourteen days after diphtheria without any simultaneous disturbance of consciousness, and only slight improvement was noticed after

several years.   With equal rarity is this complicating sequela
found after other acute diseases—for instance, scarlatina.

Sometimes no exciting cause can be demonstrated, but if
such be observed, they are always associated with a sudden
more or less marked increase of the blood pressure.   People
with diseased cerebral vessels are not rarely suddenly attacked
by an apoplectic stroke on a strong emotion, hard bodily exer-
tion, during violent attacks of coughing, sometimes also in a
cold bath and after a full meal.

How it comes about that the coldest months of the year
yield the largest percentage of victims to apoplexy, and why it
is that in the twenty-four hours there are two periods with a
maximum and a minimum death-rate, if such be actually the
case, can not be explained.   Such has, however, been claimed
by Sormani, who based his statements upon an extensive study
of statistics (Riv. clin., ser. 2, i, 12 Dicembre, 1871).   The same
author also is inclined to attribute to the position of the ba-
rometer some influence on the mortality, as in his opinion sud-
den changes in the weather materially increase the mortality
from apoplexy.

Symptoms and Course.—The rupture of a fair-sized cere-
bral vessel is always, no matter what part of the brain is affected
by it, attended with more or less violent symptoms.

Only in exceptional cases is it preceded by premonitory in-
dications.   Occasionally there are temporary sensory disturb-
ances in the extremities of one side, formication, numbness, a
feeling of heaviness in the limbs, pain in the soles of the feet,
certain choreiform movements in the face and arms (hemicho-
rea præhemiplegica, Raymond), symptoms which indicate that
things are not going in their usual order.   The patient may
also complain of headache and a feeling of fullness in the head,
which makes itself manifest on the least provocation, on the
slightest emotion, or after a small amount of wine has been
taken.   But rarely enough are such premonitions sufficiently
appreciated by the patient, and only too often are they incor-
rectly interpreted by the physician.   Usually they are over-
looked, and are first remembered when the catastrophe is
either imminent or has already taken place.

When the attack does come on, the patient gradually or
suddenly loses consciousness, and remains in this condition for
a few minutes, hours, or even for a day or two, according to
the severity of the "stroke."   The higher the blood-pressure,

and the greater the rapidity with which the blood escapes, the more pronounced and severe are the general symptoms, which collectively are called "apoplectic stroke" (the "insult" of the Germans). The way in which the disturbance of consciousness comes on varies very widely in different cases. Thus one patient may for some hours before the actual attack present a peculiar excitement, he is restless and bewildered, may even have forgotten the ins and outs of his own house, his speech is agitated, etc. Another patient may complain of headache and vertigo; a third, of a feeling of heat in his head and of general prostration ("different forms of delayed stroke"). All these premonitory symptoms which we have described may, however, be absent, and a person apparently enjoying the best of health may suddenly, as if "struck by lightning," sink to the ground and lie there unconscious (*apoplexie foudroyante*).

If we are called to such a case, the following conditions will present themselves to us on our first examination: The patient lies on his bed as if asleep, his respiration is either quiet and deep or loud and stertorous, he can not be aroused in any way, not even by strong irritation of the skin (pricking, tickling), his eyes are closed and the pupils, usually of medium size, neither much dilated nor much contracted, have lost their power to react. With every expiration the cheeks are slightly puffed out, and it is often soon apparent that one corner of the mouth is lower than the other. The extremities are relaxed, and when raised drop loosely. The tendon reflexes are absent in severe cases and neither the cremasteric nor the plantar reflex can be obtained. The pulse is full, somewhat slow; the temperature normal, perhaps slightly subnormal; the urine presents no changes, or may contain a trace of albumen, rarely of sugar.

This condition may, as we have said before, last several minutes, several hours, or even one or two days. It is modified gradually according as the hæmorrhage sooner or later comes to a stop or continues without interruption until a fatal result ensues. In the former case the patient gradually begins to react to strong stimuli, and may open his eyes for a short while, when called loudly or when water is thrown over him; he may give a loud yawn and show some voluntary motion of the extremities. Gradually consciousness returns, and the patient attempts to make himself understood by gestures and words, and in the most favorable instances, which are, however, unfortunately very rare, the physician can feel assured

that everything has cleared up, that the patient is again in possession of perfect consciousness, of the power of speech, and of motion. In such cases we speak of a "stroke without focal symptoms."

But the bleeding may continue, although only under low pressure, and only cease very gradually; then the symptoms abate but slowly and the recovery is only partial; the patient lies for days in a state of somnolence, and repeated examinations show that one corner of the mouth is distinctly lower than the other, and that the saliva dribbles from it involuntarily. If we can, by strong stimuli, evoke spontaneous movements, it becomes evident that only one side is moved, that only one arm or one leg is raised, while the other side remains perfectly motionless, and after consciousness is fully restored the certainty is forced upon us that one side of the body is deprived of its power, or, as we say, is paralyzed. This we call a "stroke with focal symptoms."

Again, the hæmorrhage may not cease at all, but continue with increased blood-pressure; then the patient remains unconscious, the breathing becomes irregular and more rapid, and assumes the so-called Cheyne-Stokes' type, the pulse becomes more rapid and small in volume, the face grows pale and haggard, the saliva getting into the trachea produces the well-known tracheal rattling, the temperature rises gradually but noticeably, and the patient dies without having come to himself, and after a period of unconsciousness which may have lasted many hours or even several days and nights.

If, in the course of a "delayed stroke," the breathing, until now quiet and regular, suddenly gives place to a rapid, irregular, stertorous respiration, and if at the same time the partial unconsciousness deepens into a profound coma, the reflexes become lost, and tetanic convulsions of the whole body and hemicontracture of the paralyzed side make their appearance, then we can assume that the hæmorrhage has burst through into a ventricle, and give an absolutely unfavorable prognosis, because in a few hours, more rarely in one or two days, death almost invariably follows. The hæmorrhage itself in such cases is, as can be demonstrated at the autopsy, generally by no means copious, but the fact that it is also found, even if the ependyma of the ventricles be thickened and hardened, speaks most clearly for the high arterial pressure under which the blood escapes (Wernicke). The bursting of the blood into the

fourth ventricle is the most rapidly fatal, and it is in these cases that we sometimes observe nystagmus.

The disturbance of consciousness in its many gradations, from the light vertigo to the deep coma, is the most character- istic, or at least the most important, symptom of an apoplectic attack produced by hæmorrhage, and it ought not to be under- rated, even if it does not become fully developed, but only amounts to a transient slight speech disturbance, accompanied by a feeling of faintness and weakness. There are patients in whom such slight disturbances occur several times before the onset of a real attack. Such patients complain of transient vertigô, slight weakness, and heaviness in one or the other hand or foot; they can at times not find the right word, the correct expression, or lose speech entirely for a short while. All these indications are premonitions, not direct forerunners of the attack, but symptoms which warn us, indicating to us that the brain is subject to alterations in the blood-pressure, a condition which may lead to serious consequences if the arte- rial walls are diseased ("apoplectic equivalents").

Complete absence of all disturbance of consciousness is a rare exception, and can only be found when the blood escapes quite slowly, so that the increased pressure rises only very gradually, and to no great degree. The patient then is seized with a sudden weakness, purely physical; he sinks into a chair, and after a few moments, during which time there is not the slightest disturbance of consciousness, he becomes aware of a sort of difficulty in moving the extremities of one side, which, in the most unfavorable instances, in a short time passes into a genuine paralysis of that side (focal symptoms without stroke). Here may also be mentioned the cases observed by Romberg, Graves, Andral, Senator, and others, in which after a hemi- plegia no trace of hæmorrhage was found at the autopsy, but only a diffuse hyperæmia of the brain could be demonstrated— "pseudo-apoplexy."

On the other hand, it is not a very unusual occurrence that a patient awakening in the morning after a quiet night's rest finds himself paralyzed on one side; in such cases we are, of course, not able to decide how much his consciousness would have been impaired had he been awake.

In every hemiplegia that occurs in the course and as a con- sequence of cerebral hæmorrhage there is a possibility of re-

generation to a greater or less extent, but whether this regeneration will take place, and when, and, moreover, whether it will be complete or not, are questions that can not at once be decided. They all depend on the condition of the cortico-muscular tract, as we have pointed out before—upon whether this be actually interrupted, whether its fibres in places, for instance at the internal capsule, be completely destroyed, or whether their function be only temporarily impaired in consequence of the increased blood pressure, so that after the cessation of the hæmorrhage a *restitutio in integrum* of the nerve tissue can follow. In the latter case, the paralysis disappears after a few hours or days, while after an actual interruption of the cortico-muscular tract the hemiplegia is incurable, and the patient is deprived of the free use of the affected limbs, and, even though he may regain after a long time some power of motion, his movements will always remain awkward and restricted.

Sometimes, and this is not very rare, a patient may have an apoplectic stroke after which the paralysis disappears quickly and entirely, but which is in a few days, on some slight provocation, followed by a second attack, accompanied by a severe permanent hemiplegia, which under certain circumstances can cause death. Such a possibility should always be thought of, and we would here say that the prognosis, no matter how slight and favorable the apoplexy may seem, should always be very guarded.

Among the "concomitant symptoms" which only exceptionally persist for any length of time, and ought therefore to be regarded as indirect focal symptoms, may be mentioned a peculiar deviation of the eyes and the head—the "*déviation conjuguée*" of Prévost—generally toward the side of the lesion, so that the eyes "look toward the disease-focus." This has been thought to be associated with a lesion in the upper parietal lobule. Prévost and Landouzy gave this rule: "Le malade regarde son hémisphère altéré s'il y a paralysie—il regarde ses membres convulsés s'il y a excitation" (the patient looks toward the damaged hemisphere if he have a paralysis, if there be irritation he looks toward the convulsed limbs). This is seen, for instance, in the so-called cortical epilepsy, which we have spoken of on page 185. The head seems forcibly turned to one side, and the eyes are turned so far over to the canthus that we are scarcely able to test the condition of the pupil; along with this symptom there is found almost always a more

or less marked dullness of the sensorium. Why this condition is generally transient is explained by the fact that the muscles of the eyes and neck can be innervated from both hemispheres, so that even if one side becomes incapable of working, the other can act vicariously for it. Only in bilateral hæmorrhages which produce a permanent paralysis of the eye muscles is conjugate deviation found to persist (cf. page 48).

Unilateral oculo-motor paralysis on the side of the hemiplegia is very rare ; it is supposed to be associated with lesions of the lower parietal lobule.

After a severe attack there may be a transient polyuria lasting for one or two days; the specific gravity of the urine, which is then faintly acid, may be 1.003 or 1.002; at times, but not always, albumen or a trace of sugar can be demonstrated. This some authors, among them Ollivier, were inclined to attribute to an action on the centres situated in the floor of the fourth ventricle, the existence of which Claude Bernard had already demonstrated. This polyuria after an apoplexy does not persist, while this may be the case in tumors of the posterior fossa, in focal lesions of the pons or the medulla oblongata, where it has to be looked upon as a focal symptom (Kahler, Zeitschr. f. Heilk., vii, 2, 3, 1886).

In proceeding to the examination of a fresh hemiplegia— that is to say, one of a few days' or a week's duration—the following points must be borne in mind :

The facial and the hypoglossal nerve deserve the most attention. The former is injured in its central course, and shows a paralysis or only a paresis in its lower branches, while the upper ones are intact ; the patient is unable to inflate the paralyzed cheek, and can not whistle, while wrinkling of the forehead on the paralyzed side presents no difficulty. Careful examination shows distinctly that the disturbance on the paralyzed side of the face is much more marked on attempting voluntary movements of one side alone, whereas those of expression—for instance, laughing, crying—are at least passably executed. This, again, may be explained by the fact that muscles used involuntarily are innervated from both hemispheres. The duration of the facial paralysis varies ; sometimes the difference between the two sides of the face disappears almost completely in a few days, while in other instances it may be noticeable for weeks or, in rare exceptions, even during the whole life. In this point it resembles the speech disturbance caused by a

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lesion of the hypoglossus, a disturbance which essentially consists in faulty articulation, which is noticed by the patient more than those who converse with him. It may disappear in a few hours, but may persist for months, even years, when improvement in the affected side has gone on for a long while, and gratifying progress has already been made. A paralysis of the same nerve, or rather of the genioglossus muscle supplied by it, is also responsible if the patient is unable to protrude the tongue straight ; it is deviated to the paralyzed side because the well genioglossus is stronger than the diseased one, and consequently pushes the tongue over toward the side of the latter.

The condition of the soft palate is not the same in all cases. The velum may be considerably lower on the paralyzed than on the well side, but it may also occupy its normal position. The uvula is at times deviated to the well, at times to the paralyzed side, and again at other times its position may be unchanged. These changes do not give rise to any noticeable disturbance of function.

Examination of sensibility in the first few days reveals decided alterations. Sensibility to pain in most cases is dulled and sensibility to touch and pressure is decreased, though to a less marked degree. The patient feels a pin prick either not at all on the affected side or, at any rate, with less acuteness.

Of the nerves of special sense, it is especially the optic which takes part in the disturbance. The apoplectic attack may be followed under certain circumstances by hemianopia of the corresponding side (Gowers), often, too, by amblyopia.

Smell and taste, as a rule, do not suffer to any great extent ; but there is a decrease in hearing power, so that the patient is no longer able to understand words spoken in an ordinary tone at a distance of fifteen or twenty feet. Such a decrease is not rare, yet an absolute (unilateral) deafness never seems to follow as a result of an apoplectic attack.

With regard to mobility, examination shows that either the extremities of one side of the body are completely paralyzed (hemiplegia) or that they are impaired in the power of movement (hemiparesis). In the latter case the arm is usually more affected than the leg and the hand more than the arm. Indeed, the movements in the shoulders and elbow joint may be as good as normal, while those of the fingers are very awkward ; in such cases the leg can generally be moved quite well. The

muscles of mastication and those of respiration are, for the reasons above mentioned, almost intact, the muscles of the trunk are only slightly implicated, and, if at all, the change is only apparent in the trapezius, so that the shoulder of the affected side is raised less energetically than its fellow.

The tendon and skin reflexes are, in the first few days after the attack, decreased or even lost on the affected side, a condition which, as we shall see shortly, soon becomes materially changed.

The sensorium usually clears up in from one to four days, especially in light cases. The patient again becomes conscious of his surroundings and recollects quite well all incidents which happened nearly up to the time of the attack. Thence on, there is, of course, a blank in his mind. On awakening, at first he has no idea of what has happened to him. His frame of mind varies according to the degree of his bodily helplessness, but, as a rule, is better than we might expect, considering the damage which has been done. Sleep is for weeks much interfered with. The patients are extremely restless; they throw themselves about in bed, and are unable to remain in one position for any length of time.

The further course depends upon whether the hemiplegia proves to be an indirect or a direct focal symptom.

The slighter cases of indirect hemiplegia, when they have not completely passed off after several weeks, are at any rate generally improved. The one-sidedness of the face, seen at the beginning, has disappeared; the tongue now is protruded straight, speech is again normal, the leg can be moved almost as freely as ever, and the only thing which is left as a reminder of the dangers through which the patient has passed is a certain awkwardness in the movements of the affected hand.

The graver cases of indirect hemiplegia need from two to three months for complete recovery. For weeks after the attack the patient presents marked disturbances in motion as well as sensation, and only painfully and with the help of a stick can he hobble about his room, while the arm and hand are almost useless. Yet a constant progressive improvement of the paralyzed limbs enables us to recognize the favorable tendency of the case and to predict with certainty a complete recovery.

In cases of direct hemiplegia also the course of the disease may assume many varieties. All are characterized by the per-

sistence of the focal symptoms. The attack, too, we should keep in mind, need not be particularly severe nor need the initial general symptoms have been especially grave ; only the conjugate deviation of the eyes and head is a symptom which preferably occurs in grave hemiplegia. Its presence, therefore, permits *a priori* of an unfavorable prognosis with regard to complete recovery.

In the first three or four weeks, things remain apparently about the same ; the paralyzed side is flaccid and about five ninths to one degree centigrade warmer than its fellow, the slightest motion is impossible, speech remains impaired, and the face is one-sided. It is not until from three to six months have passed that we are able to notice a slight improvement in the power of motion, so that the patient (who is still confined to bed) is able to move with ease some of his toes, perhaps also the lower leg, while in the thigh motion is still incomplete, and in the arm and hand quite impossible. In such cases all the improvement that can be expected is but small and the damage which the stroke leaves very apparent. After from six to twelve months the patient again begins to be able to use the paralyzed leg, which in the mean time, in consequence of the flaccid condition of the ankle, has become longer. The walk is then very characteristic. Flexion in the hip being insufficient, the leg is brought forward by the aid of the pelvis, so that the affected leg trailing along the ground describes a half-circle around the sound one. The centre of gravity of the body then is transferred to the paretic leg, the knee-joint passively extended, and the leg thus used as a stilt (Wernicke). If improvement goes on, the movement of circumduction gradually disappears and the paretic leg is simply dragged behind. The gait is so characteristic that the diagnosis, especially when simultaneously there is a paretic condition of the upper extremity, can be made at a glance.

The upper arm is slightly abducted, the forearm flexed, the hand hangs down, the fingers, which are fixed in a somewhat flexed position, are completely useless, and the patient is unable to grasp large or small objects. The arm can hardly be raised at all, and the movements of the forearm on the upper arm are very limited. In the lower leg extensor are more frequently developed than flexor contractures, and it is remarkable that in the morning, when the patient awakens after a long sleep, how slight they are and how little they trouble

him, whereas in the course of the day they are materially increased.

Contractures, which are in old hemiplegias hardly ever absent, are most likely to be attributed to a shortening of the muscles produced by disuse. This idea is supported by the fact that by systematic passive exercise, begun as soon as possible, we are able to prevent contractures; and, if they exist, a proper galvanic treatment, which takes the place of passive motion, perceptibly diminishes them. It is true it remains unexplained why contractures are not found in all cases, and why in some the paralyzed extremities remain for life flaccid. That anatomical changes, too, especially, as Charcot assumes, the secondary degeneration of the pyramidal tract, are not without influence, and that, at any rate, the contractures are more marked the farther this secondary degeneration advances, can not be denied.

A symptom which accompanies contractures, but which often occurs much earlier, is an increase in the tendon reflexes on the paralyzed side. Tapping of the triceps and biceps tendon of the arm, of the patellar tendon, and the tendo Achillis evokes lively muscular contractions. From the last named—the tendo Achillis—we can also obtain the so-called ankle clonus, of which phenomenon we shall speak later. Even tapping of bones is attended by jerkings, which are best seen in the leg when the tibia is struck ("periosteal reflex"). Here again we must leave the question open whether this increase in the reflexes is due to the degeneration in the pyramidal tracts or merely connected with the suspension of certain reflex-inhibiting influences in the brain. In favor of the latter hypothesis speaks the fact that this increase in the reflexes is occasionally observed as early as a few days after the stroke, at a time when there can be no question of degeneration in the spinal cord.

With the skin reflexes it is just the reverse; they are usually entirely lost on the paralyzed side or are at least decidedly diminished. This is especially the case for the abdominal and cremasteric reflexes, which can only in exceptional cases be obtained on the affected side.

Sensation either returns soon after the initial disturbance or is lost forever. In the latter case—i. e., where besides the hemiplegia there exists also a hemianæsthesia—the lesion is to be located in the posterior portion of the posterior limb of the internal capsule. The hemianæsthesia takes in, in pronounced

cases, the whole half of the body, including the mucous membranes, and extends as far as the median line. Face and trunk are equally affected; occasionally we may find that the trigeminus remains exempt.

In slight cases the disturbance is confined to the extremities and concerns more the sensibility to touch than the sensibility to pain. The patient feels the prick of a pin, but is unable to direct his fingers properly if the eyes are closed; he makes mistakes in recognizing objects which are given him to feel; he is unable to fasten small buttons, etc. Changes in the muscular sense also may exist for a considerable time, the patient being unable with his eyes closed to give any information about the position into which his hand has been brought.

In examining sensation in hemiplegics, Oppenheim (cf. lit.) has noticed that at times bilateral impressions are appreciated only on one side; that, for instance, if a patient is pricked simultaneously in the right and left thigh, he only perceives one prick—namely, that on the well side, although he is perfectly conscious of a prick on the paralyzed side alone.

One of the rarest of sensory disturbances is the persistent hyperæsthesia of the paralyzed side, described by M. H. Fischer (Arch. de phys. norm. et path., February 15, 1887, ix, page 185).

The psychical condition is not always the same. In certain cases the patients seem to have regained all their former faculties satisfactorily, so that a careful examination brings to light nothing more than a slight loss of will power and of the capacity for grasping ideas; but in other instances the patient becomes mentally weaker and at the same time irritable. He is easily made to cry and is liable to sudden changes of temper. Such patients are, however, notwithstanding their apparent obstinacy, very manageable and easily guided. Again there are cases in which the psychical weakness becomes very apparent. The patient forgets the commonest things, the number and the names of his children, confuses things and places, does not know what day of the week and what season of the year it is, etc.; at the same time he may have different delusions and hallucinations. Some cases finally go on to complete dementia, which takes a course not unlike that of general paralysis. Legrand du Saulle has published an interesting study of such disturbances among the apoplectics of the Salpêtrière (Gaz. des hôp., 68–71, 1881).

In the further course of severe hemiplegias where regeneration is impossible to any great extent, motor disturbances which we have designated as posthemiplegic (cf. lit. under treatise of Greidenberg) may follow. One of these is the so-called hemichorea, consisting of involuntary irregular movements in the paralyzed limbs, which become aggravated by every mental emotion and voluntary movement and which entirely cease during sleep. These movements, which are best studied on the upper extremity, occur more frequently after cerebral infantile hemiplegia than in any other affection. The "hemiataxia" described by Grasset (cf. lit.) is closely related to hemichorea, and ought to be regarded as a variety of it. According to Charcot, the seat of the lesion in these cases is in the posterior portion of the internal capsule, the posterior part of the optic thalamus, and in the foot of the corona radiata. The so-called hemiathetosis will be considered in the chapter on the cerebral palsies of children.

A second class of motor disorders is made up of those peculiar involuntary movements which have been described as "associated movements." They are observed in the paralyzed extremity when the patient moves the corresponding, unaffected, one; thus, for instance, if a patient uses his right, well, arm, the paralyzed arm makes similar movements, of course being restricted to a lesser or greater extent by any contractures which may be present. These movements have nothing in common with the so-called reflex movements which are found to occur in the paralyzed limb on stimulation of the sound one by the prick of a pin, the faradic current, etc. A peculiar instance of "associated movements" in an old hemiplegia I had the opportunity of observing for months. It was as follows: Every time the patient yawned the left arm was raised involuntarily at the shoulder-joint, and was kept up while the yawning continued; as soon as it ceased the arm dropped down helplessly. Sometimes one sees the sound limbs make involuntary movements if the patient attempts to use the affected ones, and again and again I have seen patients, straining to bend the paralyzed leg, become greatly astonished at the flexion which took place in the well leg without any such intention on their part. That in intended movements of certain muscle groups the antagonists begin to make involuntary movements—that, for instance, if an extension of the flexed fingers be attempted, the flexion at first becomes more forcible

before extension begins (Hitzig)—is, according to our experience, very exceptional.

There are other associated movements which occur in the paralyzed half of the face when the sound side is moved; so, for instance, in laughing, the muscles of the paralyzed side are seen to contract equally, or even more strongly, than those of the well side. The origin of this phenomenon, and of the movements before mentioned, must be regarded as being perfectly different.

Various theories have been proposed to explain associated movements (Westphal, Benedikt, Broadbent, and Ross), but none of them can be taken as entirely explaining the facts. It is by no means impossible that all such motor disturbances are reflex in nature (Charcot and Brissaud).

As a third posthemiplegic phenomenon we have the tremor. It is not rare, and that form especially which occurs on voluntary movements of the affected side is rather frequently met with; on the other hand, we shall very rarely have the opportunity of observing this tremor while the extremities are at perfect rest. Relatively, the largest number of cases who presented tremor, in my experience, showed sensory changes, which consisted of paroxysms of pain in the affected extremities. On a cursory examination this tremor may be mistaken for unilateral paralysis agitans (hemiparalysis agitans), especially as the number of oscillations is about the same in both affections, $4\frac{3}{4}$ to $5\frac{1}{4}$ in a second. Pronounced intentional tremor, which we look upon as a pathognomonic symptom in multiple sclerosis, I never have observed in hemiplegia. Probably the cause of this posthemiplegic tremor has to be sought in the general increase of reflex activity, which, as we may remark here by the way, is observed besides only in a very few cases of tremor of a different nature. Here it seems to play the most important *rôle*.

Of great interest, as well as, at times, of no small practical importance, is the fact that in cases of incurable hemiplegia the non-paralyzed side, that is, the apparently well extremities, undergo certain changes which we are compelled to regard as pathological. Thus, Pitres (cf. lit.) has found that the well arm loses somewhat in strength, and that this is often more marked in the beginning of the hemiplegia than later on. On an average the loss amounted to about 38 or 40 per cent, while no increase in the tendon reflexes could be demonstrated at

the same time. The well leg becomes weaker, and indeed in a more marked degree than the arm, the strength being reduced in some cases even by one half. The patient, though able to move that leg with perfect ease while in bed, finds it almost useless to attempt to stand or walk. Pitres was also the first to notice that the patellar reflex of the sound side, as well, is abnormally active, an observation which is daily confirmed. The presence of the foot phenomenon (ankle clonus) is noted by Westphal and Déjerine. All authors, however (Hallopeau, Brissaud, Féré), agree that it is extremely unusual to find the later contractures on the non-paralyzed side. On the whole, these changes, which occur on the so-called unaffected side, are more marked and of greater significance to the patient than we should be led to suppose from a superficial examination.

Trophic vaso-motor changes are not uncommon in the paralyzed limbs. While in the beginning of a hemiplegia the skin of the affected side is warmer and redder than that of the well side, it becomes cooler as the disease progresses, and frequently assumes a somewhat cyanotic color. The œdema often seen in the affected extremities is due to the absence of muscular movement and the consequent slowing of the blood and lymph current. In a patient who, two years before, had a pretty severe apoplectic attack with persistent speech disturbance, I have repeatedly observed slight repetitions of the hæmorrhage, during which the speech, which had considerably improved, again became entirely unintelligible. Simultaneously there was developed on each such occasion over the whole body, and not merely over the paralyzed right side, an urticarial rash which persisted as long as the cerebral symptoms lasted. No doubt this was due to a disturbance in the vaso-motor innervation of the vessels of the skin, which reappeared with the transient increase in the intracranial pressure. Charcot describes an acute malignant bed-sore which appears two or three days after the onset of the hemiplegia in the gluteal region, beginning as a red spot and developing in a few days into a brown, dry eschar six to seven centimetres broad. It always ends fatally, and is, according to Charcot, a purely trophic disturbance, an alteration in the tissue, which we can only attribute to nervous influences.

The nutrition of the muscles which for years have been paralyzed usually suffers but little. We can easily understand

that a slight degree of atrophy, due to inactivity, occasionally manifests itself, yet the excitability to both electrical currents remains normal. Only in exceptional cases is there pronounced muscular atrophy in the affected limbs when these, although their motion is impaired, can still be used to a certain extent. In such cases the atrophy can not be referred to inactivity, but we must rather assume a lesion in the trophic centres of the cortex, the seat of which is, however, still unknown.

The simultaneous appearance of a hæmorrhage in each hemisphere is exceptional. It needs hardly to be stated that such an accident must necessarily give rise to the gravest symptoms: bilateral hemiplegia—that is, paralysis of all four extremities—bilateral facial and hypoglossal paralysis, amaurosis, and total anæsthesia.

Diagnosis.—The diagnosis of cerebral hæmorrhage may give rise to considerable difficulties. It is only then easy if a suddenly or gradually developing unconsciousness is followed by a paralysis or paresis of one side in a patient not suffering from any valvular disease of the heart. Under such circumstances the case is absolutely clear, and even the most cautious diagnostician, if he can exclude hysteria, may safely assume a cerebral hæmorrhage with consequent hemiplegia.

It is a different matter where we have to make a diagnosis at a time when we are unable to ascertain the presence or extent of the paralysis, but where we are restricted to an interpretation of the unconsciousness of the patient. Under these circumstances we have to be familiar with the conditions which, besides cerebral hæmorrhage, are capable of giving rise to unconsciousness, and be acquainted with the characteristic manifestations which each offers.

In the first place we may have to deal with a simple fainting fit. The concomitant symptoms—the wax-like pallor of the face, the small, frequent pulse, the cold sweat which covers face and body—are not likely to allow us to mistake the condition for one of apoplexy, especially as the gravest symptom —the loss of consciousness—as a rule, is not of long duration, but vanishes rapidly if the patient is laid down with the head low, the face sprinkled with cold water, or if ammonia or eau de Cologne, etc., be held to the nose. The success or non-success of these measures will help us to settle the differential diagnosis in a few minutes.

Secondly, we may have before us an epileptiform attack without convulsions or the coma which so often follows epileptic fits. Here the loss of consciousness is also complete, and the diagnosis can only be made if we can obtain a history of previous epileptic convulsions, or if we are able to assume this from scars on the tongue. In the absence of such evidence the color of the face may sometimes be of value to us; in some epileptics this is very pale, in cerebral hæmorrhage of a purplish color, yet this rule by no means always holds good, and should therefore be accepted *cum grano salis.*

The unconsciousness so often occurring in the course of a meningitis may be recognized from the temperature and the pulse, the peculiar drawing in of the abdomen (scaphoid abdomen), the jactitations, the rigidity of the neck, and possibly from the existence of a papillitis.

In the beginning and in the course of the progressive paralysis of the insane (dementia paralytica) apoplectiform attacks occur which resemble those produced by cerebral hæmorrhage very closely indeed, and which only can be recognized as belonging to the former disease from the previous history of the patient (and later from the results of the autopsy). If we can get no information about the history the differential diagnosis is impossible.

Intoxication with chloroform and alcohol may be attended by complete loss of consciousness. An individual in the unconsciousness of alcoholic intoxication is just as hard to arouse as one in apoplectic or epileptic coma, and the diagnosis may present some difficulties under certain circumstances—when, for instance, nothing can be learned about the cause, or what has immediately preceded the loss of consciousness. Usually, however, it is easy enough. Sometimes the smell of the ingested substance puts us on the right track, sometimes prompt reaction to energetic stimuli applied to the skin may make our diagnosis clear. As long as we are not sure of our ground, we ought to abstain from all therapeutic measures. Of opium or of morphine poisoning we need only think when the pupils of the patient are conspicuously small. A degree of myosis as high as we find in opium poisoning has only its parallel, and then but rarely, in hæmorrhages into the pons, which are rapidly fatal.

Uræmic coma can easily be excluded, if we are able to examine the urine, and can detect neither albumen nor tube casts;

besides this, with the history, the examination of the heart for a possible hypertrophy should not be forgotten.

Diabetic coma, finally, is characterized by a peculiar fruity odor which comes from the mouth of the patient. It, of course, only enters into the question if sugar can be demonstrated (or has previously been repeatedly detected) in the urine.

The anatomical nature of the hemiplegia may remain entirely obscure, and only in certain cases are we able to give a decided opinion about it.

Whether hemiplegia following a stroke is due to hæmorrhage or embolism can only be determined by accompanying circumstances. The existence of valvular lesions and of atheroma speaks for embolism; nephritis, heart hypertrophy, albuminuria for hæmorrhage; yet this rule has many exceptions, and we may assume that in about half the cases a correct diagnosis is impossible.

The meningitic hemiplegia has these points in common with the hæmorrhagic—namely, the paralysis on one side and the "conjugate deviation"; but, as we have before pointed out, in meningitis we generally have the characteristic rigidity of the neck and the scaphoid abdomen; where these latter symptoms are not even suggested, a differential diagnosis, or rather the recognition of a hemiplegia as of meningitic origin, is impossible.

The hysterical hemiplegia, finally, if it persist for a long time, and if other hysterical symptoms, as anæsthesias or contractures, are wanting, can never with any certainty be differentiated from that depending upon cerebral hæmorrhage. Both may present the same peculiarities, and a decision as to which condition we are dealing with may be beyond the powers even of the practiced diagnostician. We are indebted to Charcot for a new symptom, to which he has drawn attention, and which is said to be characteristic of hysterical hemiplegia—namely, a paroxysmal spasm of the muscles of the cheek of one side, associated with an excessive deviation of the tongue to the same side. This "glosso-labial hemispasm" never exists in organic lesions of the pyramidal tract, and is therefore pathognomonic for hysterical hemiplegia (Brissaud and Marie, cf. lit.).

If the question of the anatomical seat of the hæmorrhage is to be considered in our diagnosis, we must in the first place not forget that the mere existence of a hemiplegia is not suf-

ficient to give us an answer, for as long as we do not know whether to regard it as a direct or an indirect symptom, we can say nothing positive. If we further add that even an indirect hemiplegia may persist for years, we can easily see with what difficulties we meet in attempting a topical diagnosis. It may be quite true that in a great many cases where an apoplectic attack is followed by hemiplegia, the lesion is situated in the internal capsule, and we have become accustomed to associate in our minds a certain typical clinical picture—that is, hemiplegia with more or less marked sensory changes—with a lesion in the internal capsule. We must, however, in making a diagnosis of that kind always keep in mind that an indirect hemiplegia may be produced by lesions in any part of the brain, by lesions in the frontal, in the parietal, the occipital lobe, of the thalamus, of the lenticular nucleus, of the external capsule, and that, as we have also said, the duration of such indirect hemiplegias is by no means always restricted to a period either of a few days or a few weeks, Hence a certain reservation must ever be observed by a prudent diagnostician, and he should only then speak with some certainty if he has some other direct focal symptom to guide him. Among these, we have, for instance, sensory aphasia for the (left) temporal lobe; for the occipital lobe, hemianopia; for the optic thalamus (with a high degree of probability), posthemiplegic chorea; for the crura, alternating oculo-motor paralysis; for the pons, alternating facial paralysis.

Prognosis.—After all that has been said, we hardly need to add anything about the prognosis. Any cerebral hæmorrhage is a grave event, which puts the life of the patient in danger, or rather it is a symptom which denotes that a grave arterial disease, without which a hæmorrhage never occurs, has reached a state dangerous to life. If once a hæmorrhage has occurred we are not sure but that it may be repeated at any moment, since the condition which favored it, the brittleness of the arterial walls, means a lasting incurable predisposition to a fresh hæmorrhage.

In the presence of a recent apoplectic attack, it is impossible for us to give a certain prognosis, or to predict what will follow. The severity of the disturbance of consciousness is in a way indicative, and we may say that the severer this is found to be—in other words, the greater the traumatic effect of the hæmorrhage—the less favorable is, *cæterus paribus*, the out-

look with regard to life, as well as with regard to recovery. Yet exceptions occur, and even a very severe coma which has persisted for hours does not only not always produce death, but need not necessarily leave behind it focal symptoms, as hemiplegia or the like, and such patients may then be well for years afterwards. Unfortunately, so favorable a result is rarely met with. As a rule, a hæmorrhage of any considerable size is either fatal or is followed by a hemiplegia.

As to the difference in the prognosis for the individual, indirect as well as direct, focal symptoms, most that deserves mention has already been spoken of. The indirect symptoms, as a rule, disappear after a certain time, and a *restitutio in integrum* is not impossible; the direct ones are only curable when vicarious innervation takes place from the unaffected hemisphere which assumes the function of the damaged one. This can be the case, for instance, in unilateral facial and hypoglossal paralysis, and in the lateral deviation of the eyes (lesions of the lower parietal lobule, cf. page 173); it may also occur in motor aphasia if the patient is still capable of learning to speak with his right hemisphere (lesion of the region of Broca, page 175). On the other hand, it does not occur in cases of direct hemiplegia due to a lesion of the internal capsule; then the paralysis is incurable, and the improvement which may take place is always very imperfect, although a properly conducted treatment may effect some amelioration, and thus conduce much to the well-being of the patient.

Treatment.—The primary affection, the disease of the arteries to which cerebral hæmorrhage is due, is beyond the reach of therapeutics. We possess no remedy which can cause the miliary aneurisms to disappear, and our efforts are confined to combating those symptoms which accompany, and those which follow the hæmorrhage. Thus we have to deal with (1) the apoplectic attack itself; (2) the anatomical changes which are produced in the brain by the hæmorrhage; (3) the focal symptoms, the paralysis (or paresis) of one side; and, in general, all motor and sensory disturbances referable to the attack.

(*a*) The treatment of the attack itself varies according as we have to deal with a suddenly or gradually developing apoplexy. In the former case we may assume that the hæmorrhage has already stopped when we first see the patient, whereas in the second case the presumption that the bleeding

is still going on is justifiable, and hence all measures which tend to arrest the hæmorrhage are strenuously indicated at once. One of these is venesection, which produces a fall in the blood pressure, and should always be resorted to in cases where, after (or during) a hæmorrhage, the carotids are found throbbing, the action of the heart is tumultuous, and the face red and congested. The success is sometimes surprising. The patient, who just before was comatose and motionless, with stertorous breathing, immediately after a free bleeding begins to breathe more quietly, and evidently with greater ease. He stirs, opens his eyes, and becomes conscious. In such a case venesection was the only measure indicated; it could not have been replaced by anything else—in short, it has saved the patient's life. If the pulse is small, the face pale, and the heart sounds are weak, no one will ever think of taking away blood. Then the administration of stimulants will be found useful; of course, they have to be given with great caution, and be selected carefully. Vinegar enemata, sinapisms, and ether injections may be tried. Changes in the blood pressure of the brain ought to be avoided most carefully; they may be produced by turning the patient in bed, by shouting at him frequently, and by other attempts to wake him from his coma. The physician will have to warn the friends against doing this, and do his best to have the patient left quiet and undisturbed. If the face is congested, he will order his head to be placed high and have him kept in one position. Local bleeding from the head is, if not directly harmful, absolutely useless. If bleeding is indicated at all, we shall choose venesection—cupping and leeching are matters of so much detail and are so slow in their action that they can not be recommended.

Immediately after the attack has passed off and the patient has regained consciousness the chief task of the physician is to see that he has absolute rest. Even more than any other sudden illness, apoplexy produces the greatest excitement and consternation in a family, and it can hardly be wondered at that this gives place to the greatest joy when the patient, who has already been given up, is seen to return to life and that each member of the family is anxious to express his feeling of satisfaction. All such outbursts may be very harmful to the patient, and these demonstrations must be crushed by the physician with iron firmness in order to avoid any emotion on the part of the patient; besides, he should give directions as to a

proper bed, which will answer all the therapeutic and hygienic requirements of the case, and, above all, from the very first due precautions against bed-sores ought to be taken. Proper arrangements should be made for the reception of the stools and the urine. The head ought to be covered with thin compresses, cooled with ice water or with a light ice-bag that will exert no pressure. Any simple medicine which contains acids or cream of tartar or tartar. boraxat. and the like is sufficient for the first few days, during which the patient ought to be fed upon a light, nourishing, but unstimulating diet.

(*b*) The treatment of the focal lesion in the brain—that is, of the place where the hæmorrhage has occurred—should not be begun until a considerable time has elapsed after the general symptoms have abated. This will usually be from about four to six weeks after the attack. Whether it is actually necessary to wait so long we do not know, but, as a matter of fact, we are afraid to undertake any active measures at an earlier moment, and certainly if a physician should go contrary to tradition, and if accidentally another hæmorrhage should occur, he would lay himself open to severe censure on the part of the family.

On the other hand, it seems more than doubtful whether we are able to influence the disease-focus in any way by treatment or succeed in hastening the absorption which we desire. It is, however, supposed that this can be accomplished in two ways—namely, by internal and external remedies, by potassium iodide and mercury, and by galvanic treatment respectively. The iodide treatment is based on the supposed absorbent properties of the drug. Whether it possesses such a power to any great degree is doubtful, and the fact that it so frequently fails to give good results seems to speak very much against it. On the other hand, there is no question but that iodide, if used for any long period of time, acts deleteriously on the stomach and spoils the appetite and may lead to symptoms of intoxication. An unprejudiced practitioner who does not administer medicines in a routine way will therefore always first ask himself which of the two is the lesser evil—whether he should renounce such help as is supposed to be derived from the remedy in the process of absorption and keep the patient's appetite in a good condition, or whether he should depend upon the more than doubtful action of the drug and at the same time ruin the patient's digestion. But if we have once decided to administer

iodide of potassium, let it be done boldly, and let 2.0, 3.0, 5.0 (30, 45 to 75 grains) a day in one or two doses be given in hot milk. Given in this way the administration of the drug is less likely to be followed by unpleasant effects than if we order a tablespoonful three times a day of a solution of iodide, 4.0–8.0 ; water, 200 (ℨ j – ℨ ij to ℥ vj). The mercurial inunctions to the portion of the skull corresponding to the focus are not harmful if any symptoms of intoxication are watched for and salivation is at once treated energetically ; but their success is in no way greater than that obtained with potassium iodide.

With regard to the galvanic treatment, it must, first of all, be absolutely admitted that it is possible to act upon the brain with the galvanic current. The peculiar phenomena which occur during galvanization of the head—vertigo, seeing of sparks, etc., the cerebral nature of which can not be doubted—speak strongly in favor of such a possibility, and the experiments of Loewenfeld on animals seem to indicate that these are due to an influence on the circulation in the brain. Whether, however, the galvanic current possesses, besides this undoubted action on the vaso-motor nerves, definite catalytic properties, and, if so, in a measure sufficient to enable us with its help to influence the disease-focus, nobody knows. We will suppose this and hope that it is so, because it is the only weapon upon which we have to depend. The best and most reliable electrotherapeutists, Erb at their head, with his unusually wide experience, admit the scarcity of positive results from such a treatment, and acknowledge that in by far the greater number of cases they are negative (Erb, Handbuch der Electrotherapie, page 320, Leipzig, 1882). Yet cases may occur where the physician is forced to resort to galvanization of the head— "the electrical treatment of the brain." He should therefore be familiar with the mode of application. Only large electrodes ought to be used. Fig. 81 represents the head electrode of Erb. The anode being placed on the forehead, the cathode on the neck, weak currents without make or break should be allowed to pass through the head of the patient for from one minute and a half to two minutes. Transverse and oblique currents may also be tried. (Details are to be found in Erb's text-book on electro-therapeutics, page 325).

(*c*) The treatment of the hemiplegia and the posthemiplegic motor disturbances demands, if any success is to be expected, much perseverance on the part of the patient as well as of the

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physician. If we can not familiarize ourselves with the idea
that for weeks and months the same procedures and manipula-
tions have to be gone through in exactly the same way, we
should not begin the treatment at all nor order it to be under-
taken. We shall then at least spare ourselves the disappoint-
ment of a failure ; yet with patience, and where the necessary
means are not wanting, it should be undertaken. The cases,
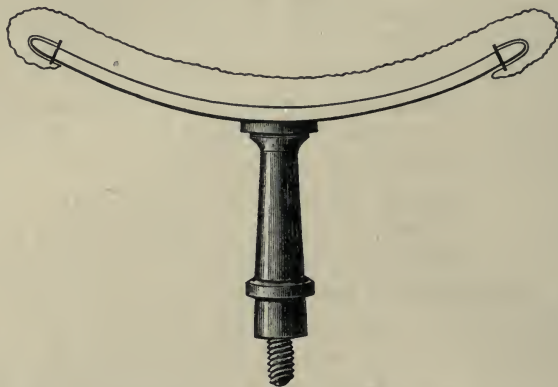
Fig. 81.—THE LARGE HEAD ELECTRODE (COVERED WITH SPONGE) OF ERB.

where a systematic treatment for a long time has benefited the
patient very materially, are numerous enough, and they would
undoubtedly be met with more frequently if a fair trial were
given it more often than is unfortunately the case.

From internal medicines absolutely no effect on the hemi-
plegia is to be expected, and even the most sanguine thera-
peutists, whose faith in drugs is unbounded, abstain here from
fruitless attempts. The same may be said about the posthemi-
plegic motor disturbances, and if we here make mention of the
use of veratrine (0.002 to 0.003 [gr. $\frac{1}{30}$ to $\frac{1}{20}$] a day in pills)
against the posthemiplegic tremor, it is only to declare the utter
futility of this medicament. We have here also to resort chiefly
to electrical treatment, but with this difference, that the good
results observed here are much more frequent and much more
marked than in treating the disease-focus. Definite rules for
the electrical treatment of the focal lesions as well as the hemi-
plegic and the posthemiplegic phenomena can not be given.
Every experienced electrotherapeutist follows certain rules
and principles which he has found out for himself in the course
of years from personal observation. Thus one claims only to

accomplish his end with quite weak, while the other has seen better results from the use of strong currents. The one uses galvanism, the other by preference the faradic current. Every one adduces reasons for his own method, which, as a rule, are strongly combated by other writers who claim to possess infinitely more experience.

Above all, the wishes of the patient should guide us in deciding which mode of electrization should be chosen. One man will have a genuine idiosyncrasy against the faradic current, and more especially against the faradic brush. Another can not stand strong galvanic currents; they excite him, make him nervous, and disturb his sleep. In a very general way we may lay down the rule that in paralytic conditions most is accomplished by the galvanic current, with frequent makes and breaks, so as to produce contractions of the muscles. In conditions of irritation, especially contractures, on the other hand, most is accomplished by local faradization. We hardly need to insist that the greatest attention must be given to the groups of muscles most severely affected—for instance, in the upper extremities, to the extensors. The faradic treatment may, especially if contractures are threatening, be begun earlier than is allowed by our rule given above, even twelve to fourteen days after the cessation of the general symptoms, without any danger to the patient.

Patients in good circumstances expect their physician to send them to a watering-place every year, as a stay there is a pleasant change from the monotonous electrical treatment, and we can not blame anybody for putting great faith in it. Unfortunately, these hopes are not by any means justified, and by a course of treatment at Oeynhausen, Wildbad, Gastein, and Ragatz, where, by the way, the temperature of the baths ought not to exceed 93° F., painfully little is accomplished, certainly a great deal less than by electrization or this alternated with massage. The latter ought only be carried out by well-trained masseurs, and only with the greatest care. From the cold-water treatment we also have seen little success on the whole, although it is decidedly to be preferred to the simple hot baths and the like. This also must be administered carefully and must be adapted to the idiosyncrasies of the patient, a rule which is unfortunately not always observed. Hydrotherapeutics can not be learned in the clinics, where only an occasional remark is made about it, but deserves and demands

a practical study in establishments where this treatment is intelligently and carefully conducted. The reason why it is not estimated everywhere as highly as it ought to be is because it is frequently not understood. Those who wish to acquire the theory of this treatment thoroughly I would refer, among other works, to the excellent text-book of Winternitz.

While we have seen, then, how helpless therapeutics is against cerebral hæmorrhage and its consequences, we have, on the other hand, the satisfaction of knowing that so much success is promised by a timely and appropriate prophylaxis, that we must recommend it most earnestly to all individuals of a so-called apoplectic habit, all who are inclined to cerebral congestion, all patients with a heart hypertrophy, and, finally, all those with hereditary tendencies. They should try to avoid putting on too much flesh and shun everything which would conduce to the production of an undue increase in the blood pressure. Among the most important rules upon which we must insist are moderation in eating, regulation of the bowels, frequent exercise in the open air, systematic gymnastics indoors—for instance, on the "ergostat" of Dr. Gärtner, of Vienna, a small apparatus which can easily be kept in the room and on which a large amount of work, measured by kilogramme-metres, can be done (the work can be prescribed in kilogramme-metres). This apparatus I can highly recommend, as I have very often seen good results from its use. To avoid increase in the blood pressure, the use of alcohol, coffee, and other stimulants, finally, all excitement, be it sexual or of any other kind, should be interdicted. Unfortunately, these warnings of the physician are not listened to until it is already too late, and men who will protect themselves in time and give up some pet habit—the customary nap after dinner or the like—in order to avoid a danger that only threatens, are few and far between.

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2. *Embolism and Thrombosis of the Cerebral Arteries. Encephalomalacia.*

Pathological Anatomy.—We have already adverted to the fact that the arteries of the cortex anastomose among themselves, while those of the basal ganglia are what we call terminal arteries ; from this it is evident that the embolus has quite a different significance where it plugs up an artery of the former type to that which it possesses when the obstructed vessel is a terminal artery, and no collateral circulation is possible. In the first case the collateral circulation compensates for the damage, while in the second case we are bound to have a necrosis in the areas supplied by the obstructed artery, a "focus of softening." It is unnecessary to dwell much upon the important bearing of this fact; suffice it to say that the arteries usually concerned are the main branches and, above all, the middle cerebral. The reason why embolic processes are more frequent on the left than on

the right side has already been explained (p. 213). Brain emboli originate in the same manner as emboli in other organs; among the causes are diseases of the left heart—chronic endocarditis, mitral disease, and weak heart—aortic aneurisms, more rarely diseases in the pulmonary circulation. Thus in certain cases purulent particles may pass from the lungs into the pulmonary vein (in ulcerative bronchitis, gangrene of the lungs, etc.), and be carried into the systemic circulation. Pölchen (cf. lit.) has also shown that certain poisons, especially carbon monoxide, as it seems, may produce softening of the brain substance. According to him, the CO while circulating in the blood acts injuriously on the nutrition of the vessels, and brings about fatty degeneration and calcification in them. Hence there finally results a necrosis of the tissue. It is possible that phosphorus acts in a similar way. Age plays a still less important *rôle* in the ætiology of embolism than in that of hæmorrhage, whereas the influence of sex can not be denied, as it is well known that by far more women are attacked by cerebral embolism than men; it is possible that this is the case owing to the greater frequency with which we find articular rheumatism with its accompanying heart lesions in the female sex, especially in its younger members. The puerperal state may also have something to do with it.

Thrombosis of the cerebral arteries is either produced by an atheromatous process which narrows the lumen of the vessel, and by slowing the blood current gives rise to coagulation, or by an abnormal proneness of the blood to coagulate. The first happens frequently in old people, and we can fairly say that atheroma is just as often the cause of senile softening as miliary aneurisms are the cause of cerebral hæmorrhage. The abnormal tendency to coagulate (hyperinosis), which the blood presents in the puerperal state, in pneumonia, etc., is rarely or never the only cause of coagulation. It can not be said to do more than favor it, and hence we need not go further into the question. Considerable general increase in the intracranial pressure may give rise to thrombosis (compression thrombosis), as also the pressure exerted on the vessels which occurs sometimes in basilar meningitis. If, in addition, the arterial walls are diseased—for instance, by tuberculosis or syphilis—the conditions are still more favorable for the formation of thrombosis. According to Gerhardt, the hemiplegias which occur in the

course of basilar meningitis are due to thrombosis with secondary softening.

Finally, we should remember that traumatism—a fall or a blow upon the head—may produce a disease in the arteries which long after may give rise to thrombosis.

The necrosis of the brain tissue which follows the cutting off of the arterial blood supply is called softening, encephalomalacia, and we speak, according to the special ætiological factor, of a traumatic, an embolic or thrombotic, and an atheromatous (senile) softening. The process is as follows (Wernicke): The vessels in the area from which the blood supply is cut off collapse, the lymph spaces dilate and through aspiration become filled with cerebro-spinal fluid, so that the whole tissue appears soaked, and the recent focus of softening shows a decided increase in volume; the nerve fibres and nerve cells then become macerated in the fluid, and soon undergo destruction. With the microscope we detect varicosities of the nerve fibres, myelin drops, and the neuroglia and the connective tissue appear œdematous. If many red corpuscles are present, the coloring matter coming from them gives to the whole focus a yellowish tint; such a discoloration is especially seen in the cortex (*plaques jaunes,* Charcot); the white matter which lies beneath is usually of a lighter tint. If then no sufficient collateral blood supply is established, which, as seems not impossible in a recent focus, might produce complete regeneration, there commences to develop in from thirty-six to forty-eight hours a fatty retrograde metamorphosis of the necrotic tissue. Polynuclear leucocytes emigrate from the dilated blood-vessels and invade the necrotic tissue; they take up the fatty particles, and some reach the blood current again through the lymph channels as compound granular corpuscles. The latter, which are invariably present in foci of softening more than two days old, on account of their infiltration with fat granules, are larger than the normal leucocytes. A part of them seem to undergo fatty degeneration, others seem to be transformed into myelin drops, especially in old foci. A quite gradual absorption of the dead and disintegrated brain tissue takes place, and a so-called cyst is formed, which can not be distinguished from that following a brain hæmorrhage; more rarely we find a cicatrix of connective tissue, which becomes as hard as cartilage, and grates under the knife. Softenings, which from the onset take a chronic course, have frequently been found to

form sclerotic cicatrices, so that the softening can eventually become a sclerosis (Wernicke).

In softening of the cortex quite considerable areas may become deficient, which are partly replaced by serous fluid, partly by thickened pia. The convolutions, which sometimes remain, present a yellowish discoloration, appear atrophic, and are of a firm sclerotic consistence.

Symptoms and Diagnosis.—Just as in hæmorrhage, we may in embolism have symptoms which have to be regarded as premonitory of the regular attack. They resemble very closely those above described, and chiefly consist of vertigo, headache, an occasional feeling as of pins and needles in the limbs, etc. The headache may be especially prominent ; it may persist for weeks with undiminished intensity, and then disappear, or be followed by a distinct deficiency in memory or beginning mental decline.

The attack proper, which occurs at the moment the lumen of the vessel is completely obstructed by the embolus, may simulate the apoplectic attack so closely that it may be absolutely impossible to distinguish the one from the other. All the above described differences in the nature and degree of disturbance of consciousness may be met with here also, and though it is true that at times the attack sets in with more violent epileptiform convulsions, that the face is less congested and respiration less disturbed, these points are by no means sufficient for a differential diagnosis. It is supposed that complete loss of consciousness speaks more against embolism and for hæmorrhage, and the early disappearance of the paralytic symptoms present point rather to embolism. Gerhardt considers (Berl. klin. Wochenschr., May 2 and 9, 1887) a well-pronounced aphasia to be in favor of embolism in doubtful cases.

In embolism the attack is not evoked by an increase in the blood pressure, as in apoplexy, but by a "negative pressure." "Since the vessels lying to the peripheral side of the embolus suddenly collapse, and the blood contained in the capillaries flows into the veins owing to the *vis a tergo* exerted by the contraction of the vessels, a vacuum is suddenly formed in the tissue, and hence a negative blood pressure is produced" (Wernicke, *loc. cit.*, p. 133). In its efforts to fill up the empty space, the brain parenchyma is bound to be subjected to a more or less considerable traction from all sides, which may

sometimes lead to disintegration. If only a very small area is affected by the embolus, a regular attack may not take place and consciousness not be lost; if it is very large, various indirect symptoms may appear, and indeed even the non-affected hemisphere be implicated. But even after a severe stroke and after consciousness has been lost for quite a long time, a favorable event is by no means impossible, because the tissue does not necessarily disintegrate, as in hæmorrhage, but an equalization of the blood pressure can take place, which will cause the disappearance of all the symptoms.

Cerebral thrombosis rarely gives rise to a stroke, owing to the slowness with which the process takes place, and when an apoplectiform attack actually does occur, it must be due to the previous obstruction of, other, neighboring vessels. We had a considerable area dependent for its blood supply on a single vessel which before remained open, but has now gradually become so narrow that the pressure in it becomes too low to keep up the function (Wernicke).

The necrosis (softening, encephalomalacia) to which the obstruction of an artery, if lasting sufficiently long, is bound to give rise, manifests itself by certain focal symptoms, which may, just as in hæmorrhage, be divided into direct and indirect. Among the indirect the hemiplegia, often attended with hemianæsthesia, which closely resembles that described above, is the most important. Monoplegias also and hemianopia may set in without a definite stroke, and may be produced indirectly from the focus of softening, which lies in close proximity to the part the functions of which are interfered with. If an embolus obstruct an artery which can communicate by anastomoses with those of neighboring areas, and thus the damage can be compensated, we shall meet with transient focal symptoms (Wernicke), which at the most require eight days for complete recovery.

To determine the exact seat of the focus of softening, we must go to work with the same caution as in making a topical diagnosis of a cerebral hæmorrhage. Here as there we have to look for direct focal symptoms, and it is to these that most attention should be given in our examination; on the other hand, we must not forget that a focus of softening, even if it be of considerable extent, may pass through all its phases without a single symptom. No one region of the brain seems to be more exposed to softening than another. We found that the

number of hæmorrhages at the base largely preponderated over those in the cortex ; in embolism this is not the case. It is only because the surface covered by the cortex is much larger than that of the brain stem that we find in the latter numerically less cases of softening than in the cortex (Wernicke). The thalamus and pons are only rarely the seat of isolated soften- ing, while hæmorrhages are found there much more frequently, whereas the medulla oblongata is more commonly the seat of softening. To diagnosticate hæmorrhage in the medulla ob- longata during life is practically impossible, as in these cases death is almost instantaneous.

Prognosis.—The prognosis in embolism is, *cæteris paribus*, in general better than that of hæmorrhage. Not only is the outlook for complete recovery more favorable even if the attack has been severe and has lasted for a considerable time, but in most cases the danger to life is far less than in apoplexy.

Indirect action upon the medulla oblongata, in consequence of which the urine may contain albumen or sugar, is a rare occurrence. Even a softening of considerable extent may ex- ist for a relatively long time without the manifestation of any grave general symptoms. Yet an unfavorable turn is not im- possible, and this should always be feared if a sudden and marked elevation of temperature takes place.

Treatment.—The treatment is very limited ; indeed, embol- ism as such, and the necrosis produced by it, are entirely out of its reach. It can only be directed against the attack or consist of the prophylactic measures by which we may hope to prevent the occurrence or repetition of the accident. The latter un- doubtedly is the more important, and much can be accomplished by repeated local bleeding from the head (Laborde), a proced- ure which is also indicated in the treatment of the attack itself, as the cerebral circulation is possibly favorably influenced by it. That absolute rest is strongly indicated in cases where heart disease exists, needs hardly to be mentioned. Where there is a reasonable suspicion of syphilis, potassium iodide, 2.0–5.0 (grs. xxx–lxxv) *pro die*, ought to be exhibited (cf. p. 241).

Where there are multiple foci of softening the symptoms naturally depend on their seat. At the autopsy a number of such foci may be found which could not be diagnosticated during life because they were too small and were situated in so-called indifferent places. If several portions of the brain are

affected, each of which gives rise to a focal symptom, there may be a complication of the most varied clinical manifestations.

Of great practical interest is the observation to which of late years attention has repeatedly been called, namely, that foci of softening may occur in that cerebral portion of the cortico-muscular tract which contains the fibres destined to supply the muscles used in speaking and swallowing. These fibres pass from the lower third of the central convolutions, where the supposed centres for the hypoglossus and facial are situated, and end in the nuclear region of the medulla oblongata (cf. Fig. 75, page 209). Such foci have again and again been found. Sometimes they were bilateral and situated in the basal ganglia, especially the lenticular nucleus, sometimes on one side only—e. g., in the right corpus striatum —and it has been observed that they sometimes give rise to a complication of symptoms which simulate most closely those of Duchenne's bulbar paralysis. The fact, however, should be especially emphasized that the occurrence of such a focus on one side is sufficient by itself to produce all these symptoms (Lépine and Kirchhoff, cf. lit.).

The disturbances which go to make up the clinical picture are at times exclusively, always chiefly, referable to speech and deglutition. They resemble at first sight so much those of bulbar paralysis that the name pseudo-bulbar paralysis, or paralysis glosso-labio-pharyngea cerebralis, seems justifiable. Still, there are some points which should help us to avoid mistakes. Thus, while the beginning of the true bulbar paralysis is slow and gradual, the cerebral form often sets in quite suddenly with apoplectiform symptoms; in the pseudo-bulbar paralysis there is a manifestation of other cerebral disturbances which do not occur in Duchenne's disease. Again, the latter runs an uninterrupted progressive course, while in the cerebral paralysis long remissions are frequently met with. A certain asymmetry of the paralysis, which is especially noticeable in the orbicularis oris (Berger), favors the diagnosis of the cerebral as opposed to the bulbar affection. Far more important than all these points is the condition of the tongue, which in the pseudo-bulbar paralysis does not become atrophied, and hence does not assume the appearance so eminently characteristic of the true bulbar form. Consequently there are no changes to be made out in the electrical excitability, whereas in Duchenne's disease reaction of degeneration is the rule. If,

finally, we add that in the cerebral form the laryngeal muscles seem either to be not at all or only a little affected, we have sufficient data to solve the question of differential diagnosis in most cases satisfactorily.

The prognosis with regard to life is just as unfavorable in the one as the other form, only this should be borne in mind, that in the pseudo-bulbar paralysis remissions may occur; that we therefore can with a clear conscience give the patient good hopes of improvement. The duration of the disease may be much longer than is ever the case in the genuine bulbar paralysis.

The treatment is not so hopeless, as in Duchenne's disease. The galvanic current intelligently applied, and careful galvanization of the brain and peripheral faradization of the paretic muscles, frequent excitation of the muscles of deglutition, as was described on page 151, all may be tried with the justifiable expectation of effecting at least a transient, sometimes, indeed, a quite gratifying improvement.

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3. *Endarteriitis (Syphilitica).*

This process, first accurately described by Heubner in 1874, affects more especially the vessels at the base of the brain. The walls become opaque, show grayish translucent or whitish thickenings, and the vessels finally may be converted into firm,

grayish-white cords. The new tissue which encroaches upon the lumen of the vessel either originates in the intima by an increase of the endothelial cells, which become transformed into connective tissue (Heubner) or is derived from the nutrient vessels of the media and adventitia, and consists therefore of emigrated cells (Baumgarten). On account of this tendency to thickening and consequent obliteration of the vessels, C. Friedländer has proposed for the process the name endarteriitis obliterans. While not denying that Heubner, who has studied the question most carefully, has arrived at important results, we must at the same time affirm that the arterial disease, which he describes as specific in nature, is not peculiar to syphilis, but that we find the same changes wherever we have a chronic inflammatory process with the formation of granulation tissue, as, for instance, as a consequence of alcoholism (C. Friedländer). This one fact remains of the greatest practical importance, that in the course of syphilis the cerebral arteries are very frequently diseased, and that as the outcome of this diseased state the most diverse cerebral symptoms may arise. That under certain circumstances a hemianopia can be the result of such disease is proved by the interesting case reported by Treitel and Baumgarten (Virch. Arch., Bd. cxi, Heft 2, 1888), where, as a consequence of gummatous arteriitis obliterans of the arteria corporis callosi dextra, although the optic nerves were intact, a unilateral temporal hemianopia had developed. Furthermore, it is to be remembered that often enough an autochthonous thrombosis due to this arterial disease gives rise to an attack which can not be distinguished from the above-described true apoplectic stroke with consequent hemiplegia. If recovery takes place in these cases the same thing may be repeated several times, and it is especially in syphilitic diseases of the arteries that this seems relatively frequent. The patient suffers from intense paroxysmal headaches, occasionally loses his consciousness, and presents a transient hemiplegia, but again recovers fairly well, until finally he succumbs to a graver stroke. This, then, is the usual course which the disease takes. It can, of course, only be diagnosticated where the history of syphilis is clear.

The recognition may sometimes be difficult if other cerebral symptoms are present, such as speech disturbances, intention tremor, decrease in memory, and the like, when we are liable to think of multiple sclerosis, or progressive paralysis of the in-

sane, and it may only be the amenability of the disease to spe-
cific treatment which will clear up all doubts. This consists in
the use of bold doses of potassium iodide, 4.0–6.0 (ℨ j– ℨ jss.) a
day in hot milk until sixteen ounces are taken, and an ener-
getic course of inunctions—thirty to fifty inunctions of 2.0–2.5
(gr. xxx–xl) ung. hydrarg. It should be begun as soon as pos-
sible, as the patient is in no way injured by this procedure,
while the benefit may be most conspicuous.

4. *Dilatation of the Arteries of the Brain.*

Aneurisms of the cerebral arteries may be of traumatic
origin or, what is more common, may depend upon endarteri-
tis, and in this latter case syphilis again deserves special men-
tion, as among fifty cases of brain syphilis there were found
six instances with aneurisms (Heubner). Further, there is
the embolic origin of aneurisms, which must not be forgotten
(Ponfick).

Dilatations have been noted in the basilar artery, in the
middle cerebral, and, though but rarely, in the vertebrals.
Three cases of basilar aneurism have been reported by Noth-
nagel (Topische Diagnostik, p. 526); the symptoms presented
nothing characteristic, but varied much, and even symptoms
referable to the pons were not in all cases present. Vertebral
aneurisms, as described by Cruveilhier, Lebert, and others,
have occasionally been found to be attended with occipital
neuralgia. Dilatation of the vertebrals produced by atherom-
atous degeneration may affect the surrounding parts, and,
as a consequence of structural changes produced in the neigh-
borhood of the vagus, lead to grave respiratory disturb-
ances (Oppenheim, Berliner klinische Wochenschrift, 34,
1887).

Aneurisms of the ophthalmic or internal carotid in the
cavernous sinus may give rise to a pulsating exophthalmus,
which can by appropriate manipulation be temporarily pressed
back into the orbit. The pulsation of the eyeball, which may
be propagated to the forehead and temple, is a source of great
annoyance to the patient. In connection with multiple aneu-
risms, such as have been observed by Paulicki, for instance,
existing simultaneously in the basilar, the anterior communi-
cating, and the middle cerebral artery, epileptiform convul-
sions and psychoses have been noted. Definite pathognomon-
ic signs do not, however, exist, and the diagnosis *intra vitam*

is only exceptionally made with certainty. According to Gerhardt, there can at times be heard between the mastoid process and the thick cords of the muscles of the neck a murmur referable to the cerebral arteries; it is systolic or continuous, and is heard on one or both sides if the patient refrains from breathing or swallowing. Nevertheless, it is rather exceptional that a (small) aneurism of the cerebral arteries is diagnosticated correctly during life. In larger aneurisms, which produce characteristic focal symptoms, this will at times be easier, especially when ætiological data—e. g., traumatism—are present.

5. *The Neuroses of the Arteries of the Brain (Anæmia and Hyperæmia of the Brain).*

The vaso-motor nerves of the cerebral and meningeal arteries arise partly from the cervical sympathetic (Donders and Callenfels), partly from certain cranial nerves (Nothnagel). They may be excited or paralyzed idiopathically, or reflexly, especially from the stomach, and the resulting conditions, although as yet only imperfectly understood, are of great practical importance. Both stimulation and paralysis are, of course, usually only temporary, while in the intervals and in the normal state the vaso-motor nerves as well as their centres are in a state of moderate tonus. If the stimulation should from any cause be more than is necessary to maintain this normal tonus, a spasmodic contraction of the smaller arteries takes place, the absolute amount of blood in the brain becomes diminished, the patient gets pale, complains of dizziness, and loses consciousness—in other words, " faints " (acute nervous cerebral anæmia). At the same time the heart's action is weakened, the pulse is small, the face and body are covered with cold perspiration, and if this irritation is frequently repeated a certain predisposition to slight changes in the blood-pressure becomes gradually established, a condition of things which is favored by the mobility of the cerebro-spinal fluid. The attacks now occur on the slightest provocation, and in the intervals between them the patients complain of dull headache, vertigo, etc., the face at the same time usually being of a pale, wax-like color. Certain general diseases, especially chlorosis and pernicious anæmia, greatly predispose to these paroxysmal vascular spasms; in fact, cerebral anæmia is not infrequently one of the symptoms of general anæmia, as it is ob-

served, for instance, after frequent and profuse bleeding from hæmorrhoids.

Among the ætiological factors, certain occupations play an important *rôle.* Working in lead especially may give rise to a chronic vascular spasm, and thus to cerebral anæmia, which is associated with almost constant headache (encephalopathia saturnina).

Tanquerel des Planches, the best modern authority on saturnine affections, has described this condition, and it has again and again been made the subject of the most careful inquiries. It would be beyond the scope of our present work to speak of these in detail; those interested in the subject will find references at the end of the chapter; suffice it only to say here that this saturnine anæmia, if the obnoxious action of the metal is continued and the disease is once established, may produce in the workers severe cerebral attacks, epileptiform convulsions, and the like.

The treatment of acute cerebral anæmia consists primarily in placing the patient in an appropriate position—that is, with the head low or at about the same level as the feet, so as to aid the blood flow to the brain; the use of stimulants (wine, brandy, coffee), occasionally a subcutaneous injection of ether, may be indicated.

For chronic cerebral anæmia galvanization of the brain or of the cerebral sympathetic may be tried. As a matter of course attention must also be paid to a possible primary cause, and every pernicious ætiological factor removed (change of occupation, etc.).

The opposite condition, a paralysis of the vaso-motor nerves, produces a dilatation of the cerebral vessels, and thus an immediate overfilling of the same. This can be demonstrated by ophthalmoscopic examination. Often, but not always, the vessels of the face share in the disturbance; the countenance of the patient assumes a purplish-red color, he complains of throbbing in his temporals and carotids, of headache, of buzzing in the ears (acute nervous hyperæmia)—in general, of about the same symptoms as we have described in the vascular spasm, the only difference lying in the color of the face. It is observed in certain individuals regularly after the use of quite moderate quantities of alcoholic beverages (wine, beer), or, just as the anæmia, after emotions, strong bodily or mental exertion,

too much study, etc.; the abuse of tobacco may also give rise to it.

On account of the very varied manifestations of the affection different forms of cerebral hyperæmia have been distinguished (Andral, Eichhorst). Thus, a cephalalgic, a psychical, a convulsive, and an apoplectic form have been described, according as either headache or psychical excitement, with insomnia or epileptiform attacks or periods of unconsciousness (which latter are not rarely followed by cerebral hæmorrhage), are the most prominent symptoms. The transition between these " forms " is, however, so gradual, and so seldom are they sharply defined, that, for practical purposes, it does not seem worth while to make the distinction. We have repeatedly observed marked contraction of the pupils, while in anæmia they are more frequently dilated and react sluggishly. As we have pointed out above (page 223), simple cerebral hyperæmia may produce hemiplegia, which can easily be confounded with the apoplectic form (pseudo-apoplexy).

The treatment is rather unsatisfactory; it is true we may in acute attacks of cerebral hyperæmia give early relief to a patient by placing him in an appropriate—that is, nearly sitting—posture, by applying ice-bags to his head, or, finally, by free venesection; but these attacks are so frequently repeated in individuals predisposed to them that the question of such treatment is not of so much importance as of the adoption for months and years of a careful dietetic *régime*. Besides keeping the bowels well open—a thing which should never be omitted —the patient must be advised to take enough exercise, even practice gymnastics; he should be cautioned against indulgence in heavy, indigestible foods, and, above all, in alcoholic beverages. A yearly visit to places like Marienbad, followed by a stay in a pure mountain air, moderate but daily excursions on foot, the occasional use of Carlsbad water under the direction of the physician—all these may be prescribed with advantage. Much care should, however, be taken with the so-called cold-water treatment, which, like sea-baths, may only increase the hyperæmia. This applies equally to the massage treatment, which, unless carried out in accordance with certain indications and fixed rules, and under the supervision of a competent medical man, often is productive of more harm than good in this disease.

17

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B. Diseases of the Cerebral Veins and Sinuses.—The blood from the brain and meninges is carried back toward the heart by the internal jugular vein.   This vessel emerges from the jugular foramen and after its junction with the external jugular becomes the common jugular, which, after it has in turn received the subclavian, is called the innominate vein.   The two innominates together form the superior vena cava.

Between the two layers of the dura mater there exist spaces which convey venous blood but are without valves.   These are called sinuses.   The veins of the cortex empty themselves into the longitudinal sinus (sin. falcif. maj.), which terminates behind in the torcular Herophili (confluens sinuum).   The mode in which the veins empty into the sinus—namely, in the direction opposite to that of the blood current in the latter—produces a slowing of the circulation, and thus explains the frequent occurrence of coagula in the veins of the cortex and the sinus.   The deep cerebral veins are collected into two trunks, which are known as the veins of Galen. These again unite into one, the vena magna Galeni.   They convey the blood from the ventricles to the sinus rectus sive perpendicularis, which in its turn empties itself into the torcular Herophili. The blood from the inner ear goes into the cavernous sinus which is situated at the side of the sella turcica ; that from the mastoid cells into the lateral sinus, which at the jugular foramen passes into the so-called bulb of the internal jugular vein.   The veins themselves

anastomose but little with each other, while the sinuses do so freely. It is important to note the communications between the intracranial and the extracranial veins—for instance, of the nasal with the anterior end of the longitudinal sinus, the ophthalmics with the sinus cavernosus and the facial veins, etc.—and the communications made by the venæ diploëticæ, for only then can we understand how pathological processes can extend from the outside of the skull to the inside, and how occasionally we find an external swelling in affections of the sinuses.

Here it is more especially thrombosis with which we have to deal, which may occur in the veins as well as in the sinuses. The distinction is not always easy in life nor even after death, because after death the venous thrombosis may extend into the sinus and be taken for a sinus thrombosis.

If only one vein is affected the mischief may be but slight. Usually, however, it takes in one or two of the larger veins, which become obstructed during the course of exhausting, acute, especially infectious diseases or after an injury, for instance, a blow on the head. The preponderating number of the patients are children, and at times, especially during the hot season, quite young children, in which cases a special ætiological datum can not be found. The symptoms are the following : Hemiplegia, ushered in by convulsions and lasting only a few weeks, is followed by a permanent weakness, not infrequently by occasional spasms in the arm. The development of the child is then usually faulty, for apart from the occasional atrophy in one arm or in one leg or of the whole side, epileptiform convulsions may persist for years, which not rarely have an injurious influence on the mental development of the patient. In such cases at the autopsy often thrombosis of the longitudinal sinus and of the veins emptying into it is demonstrable. In adults, such a thing as a thrombosis of the cortical veins is extremely rare.

Sinus thrombosis may have one of two causes. Either we have a general disease which favors the coagulation of the blood—as in children profuse diarrhœa, acute infectious diseases, in old people, tuberculous and carcinomatous processes— or neighboring parts, as, for instance, the skull bones or the skin of the scalp are diseased (erysipelas), an extension of the process becoming possible on account of the communications between the extracranial and intracranial vessels above described. We distinguish the true inflammatory thrombosis,

which affects the lateral, the petrosal, and the cavernous sinus, from the so-called marantic thrombosis, which often occurs in the superior longitudinal sinus. In both cases the secondary symptoms of engorgement, which are especially marked in thrombosis of the longitudinal sinus and which manifest themselves in so-called meningeal hæmorrhages, are of the greatest importance. Such meningeal hæmorrhages are found in children (post mortem) as thick coagula distributed over the cortical motor centres, where they have in life given rise to a curious combination of paralysis and spasm, the power of spontaneous movements, however, being retained (Gowers). Choreic movements complete the picture which congenital chorea, bilateral athetosis, and double spastic hemiplegia present, cases which are difficult to interpret and still more difficult to diagnosticate. In these patients, too, the mental development remains imperfect, and their irregular movements and contractures (often most marked in the calf muscles) give them the appearance of helpless cripples.

The diagnosis of sinus thrombosis can only be made with any certainty if to the general symptoms (headache, somnolence, paralyses in the distributions of the cranial nerves) signs are added which point to circulatory disturbances peculiar to sinus thrombosis. Thus, for instance, symptoms of engorgement in the ophthalmic veins, manifesting itself by prominence of the eyeball, œdema of the lids, congestion of the retina, etc., point to obstruction of the cavernous sinus; œdematous swellings behind the ear to affections of the lateral sinus, and finally symptoms of passive hyperæmia in the nose—epistaxis, marked fullness in the veins of the temporal region, in small children fullness of the anterior facial veins situated between the large fontanelle and the temples (Gerhardt)—to implication of the longitudinal sinus. Pain and swelling of the corresponding side of the neck may be significant of a jugular thrombosis, etc. All these conditions are, however, but rarely met with, and they are more easily found in the books than demonstrable in the patient. The duration of a sinus thrombosis varies between several days and three to at most four weeks. The prognosis is usually unfavorable and the treatment unsatisfactory and purely symptomatic.

INFLAMMATORY PROCESSES IN THE BRAIN SUBSTANCE.

1. Purulent Encephalitis—Brain Abscess.

Pathological Anatomy.—Circumscribed pus formations in the substance of the brain, which anatomically differ in no way from pus formations in other organs, are called brain abscesses, and we speak of them as encapsulated and non-encapsulated, according as to whether or not they are definitely separated from the surrounding tissues by sclerotic thickening. In the former, a membrane of connective tissue incloses the abscess, which contains a thick pus; in the latter, disintegrated nerve tissue and crystals of cholesterin are found in conjunction with the frequently very fœtid pus, and the abscess walls are formed by a soft layer of brain tissue infiltrated with pus, and surrounded by areas of yellowish softening and œdema. In the softened areas compound granular corpuscles are found in great numbers. The size of the abscess may vary from that of a pea to that of an apple, and it may even take in nearly the whole hemisphere. The larger the abscess the more marked are the signs of increased intracranial pressure, the more flattened and indistinct the convolutions on the surface of the brain, and the drier and more adherent becomes the pia mater. Should the abscess break through into one of the ventricles pus may eventually be found in all of them, and the ependyma then appear œdematous. If it reaches the surface of the brain it may give rise to a diffuse purulent meningitis (Wernicke).

Ætiology.—Ætiologically, injury is of the greatest importance, though it need not necessarily have affected the skull itself, but may produce an abscess just as well if confined to the soft parts; in such a case, the inflammation extends through the bone, and the infectious material penetrates into the brain from the flesh wound. If we have no open wound, no break of continuity in the soft parts, then even extensive destructions of the brain substance often do not lead to an abscess formation, just as in the fractures of the skull healing occurs without suppuration if only the external air is excluded from the injured parts of the brain.

Besides traumatism, suppuration occurring in the neighborhood of the brain may cause a brain abscess; thus, in rare instances, it is a purulent parotitis or suppuration in the nasal cavity or, more frequently, caries of the petrous portion

of the temporal bone or suppuration in the middle ear which becomes the starting point. For years an otitis media may persist and be attended with a purulent discharge from the external ear without any brain symptoms, but suddenly this running may stop, the pus is retained, and probably gives rise to the caries of the bone, on account of which the petrous portion may become so soft that it can be cut with the knife; a brain abscess then develops either in the temporal lobe or in one of the hemispheres of the cerebellum.

Suppuration in the bronchi, putrid bronchitis, bronchiectasis (Biermer), furthermore, ulcerative endocarditis and pyæmia may also give rise to brain abscesses, which are then designated as "metastatic" abscesses. Idiopathic abscesses—that is, those in which no ætiological factor could be discovered—have been observed by Strümpell in some cases of epidemic cerebro-spinal meningitis.

Symptoms.—The symptoms of a brain abscess are divided into general and focal. There may, however—and this is of much practical importance—be no sign of brain mischief at all. A man may not complain of anything worth mentioning, save, perhaps, of an occasional headache, and at the autopsy a brain abscess be discovered. Quite a number of these cases are well authenticated, and there can be no doubt as to their existence; to be sure, we ought not to forget to add that the place in which such an abscess is developed must be in a so-called indifferent region.

Among the general symptoms the one most constant and the most distressing to the patient is headache; it can by no means always be localized, but more frequently affects the whole head, and may last with greater or less severity for weeks or even months. Occasionally the torture is such that the patient, incapable of doing anything, is forced to remain quietly in bed, although no other symptoms may be present. Very often, it is true, disturbances of the sensorium may appear after the headache has lasted for a long time; a strange apathy takes possession of the patient, his sleep is disturbed, and his general condition is aggravated if, as is common, febrile movements set in, which may be attended with convulsions, which are mostly unilateral. Attacks of vertigo, sometimes severe enough to cause great anxiety, and sometimes only transient, occur, and not rarely there are spells of vomiting, sometimes lasting for days, and acting very deleteriously

on the patient. The ophthalmoscopic examination, as a rule, does not reveal any fundus changes; choked disks are only exceptionally found, certainly much more rarely than in brain tumors. The focal symptoms of cerebral abscess are almost exclusively direct. This is a fact which is easily understood if we consider their mode of origin; they are produced either by a direct destruction of the brain substance or by the preceding œdema and the attendant "preparatory softening" (Wernicke), both of which processes are strictly local. At the same time we must not lose sight of the fact that the part affected by this "preparatory softening" is still capable of regeneration. Indirect focal symptoms have only been observed in cerebellar abscesses; in such, paralyses of the abducens and other nerves have been noted (Wernicke).

How different focal symptoms show themselves, and which are characteristic of lesions of the different parts of the brain, has been discussed above (page 192); suffice it here to add that abscesses of the so-called motor region produce hemiplegias, which appear in a very characteristic manner—namely, step by step. In abscesses of the occipital lobe hemianopia is the direct focal symptom which, if properly used, may settle the diagnosis. The direct focal symptom of the temporal lobe —the crossed deafness—can only rarely be accurately determined, as the suppuration of the middle ear, which we have shown often to be ætiologically connected with brain abscess, is mostly bilateral, and as testing of the hearing in patients, whose mental activity is somewhat dulled, is very difficult, since they are usually unable to appreciate any decrease in hearing on one side. In general, we must confess that too little attention has been paid to the testing of the hearing, and that the examinations have not been made with sufficient care.

In no one of the few reported cases of abscess of the pons, the medulla oblongata, and the cerebellum have direct focal symptoms been observed, or at least noted with any certainty; the general symptoms, which are mentioned in connection with the abscesses of the cerebellum, must be attributed to pressure produced by the growing abscess.

Course.—The disease may pursue its course in one of three different ways:

(1) It assumes from the onset a tumultuous character, whether it originate from a traumatism or disease of the middle ear. Violent pains—at first local, later spreading over the

whole head, and lasting from two to four days—together with marked elevation of temperature and paroxysms of convulsions, are followed by grave disturbances of consciousness. These may last for three, four, even eight days, when the patient, without regaining consciousness, dies in a restless delirium, presenting the picture of one suffering from severe organic disease.

(2) These paroxysmal symptoms lose, after a few weeks, their acute character, and become less and less marked; the patient seems to feel better, and he may, indeed, be free from all trouble for several months. Even the headache seems—at least at certain times—to have vanished. This state of absolute (or relative) latency may be of variable duration, and may, by the inexperienced diagnostician, be mistaken for complete re-covery, but it is doubtful whether this latter ever occurs. It certainly happens much more frequently that after this period of latency the initial symptoms again make their appearance, this time to continue without intermission until death. The duration of the whole disease comprises then three to six months or more; it is extremely rare that the period of latency lasts for years.

(3) The onset of the disease is insidious and chronic. The patient, who presents slight fever and general symptoms, grad-ually becomes emaciated. He complains of headache and dis-turbed sleep, and from time to time, apparently without reason, is taken with chills; he begins to have a cachectic appearance and bears on his face the imprint of a grave disease. In such, withal very rare, cases our patient is suffering from phthisis and the brain abscess is of a tubercular nature. The duration of this form, as a rule, does not exceed three or four months.

Diagnosis.—In the diagnosis we may have to differentiate between brain abscess, purulent meningitis, meningeal hæmor-rhage, and brain tumor. If the course of the abscess is very acute, as has been described above (eight to ten days), then it is often impossible to distinguish it from an acute purulent meningitis, an error which is the more excusable when all direct focal symptoms which often accompany an abscess are wanting. Remissions point rather to the existence of a brain abscess.

From meningeal hæmorrhage, which just as abscess may be the consequence of traumatism, it is also distinguished by its course. Traumatic meningeal hæmorrhages usually give rise to epileptiform attacks, which are to be referred to the

effect of the entrance of the blood between the dura and the skull on the motor centres. They are immediately followed by a coma, which lasts until death. In abscesses the insensibility usually lasts only a few hours, and only after a marked improvement has again taken place do alarming symptoms make their appearance.

A brain tumor can be differentiated from an abscess by the fact that in the former febrile symptoms are absent, while, on the other hand, in the latter, choked disks, which are a frequent sign in brain tumor, are only exceptionally noted. The course—more especially as regards the remissions, which are well marked and often of long duration—is characteristic of abscess; a tumor usually is steadily progressive. Finally, we are justified in diagnosticating an abscess if after a protracted and varying course the disease suddenly terminates with certain severe symptoms of collapse and death. When this occurs it is probable that an abscess existed which has perforated either into the ventricles or to the surface. In cases of traumatism or in cases in which the cerebral symptoms were preceded by an otitis media we should always think first of brain abscess.

The seat of the abscess can only then be determined with any certainty if characteristic focal symptoms—for instance, hemianopia or sensory aphasia—are present. In cases of hemiplegia we can, from the order in which the component monoplegias occur, draw a conclusion as to the point of origin of the abscess. Thus, if at first a paralysis of the leg, together with marked sensory disturbances, are the prevailing symptoms, and only later the arm and facio-lingual region become affected, we may conclude that the abscess is proceeding from behind forward, while if the symptoms occur in the reverse order, then the frontal lobe may have been the starting point and the abscess be extending backward. In cases of traumatism the abscess is to be located in very close proximity to the injury. Where there is a history of otitis media it usually establishes itself in the temporal lobe or the cerebellum. The white matter is, in the cerebrum as well as in the cerebellum, by far the most common seat. In the brain stem it occurs only very rarely, while in this situation, as we have seen, hæmorrhage and softening are more common.

Prognosis.—The prognosis is absolutely bad with regard to recovery and doubtful with regard to life. We can see from what has been said that spontaneous cures, most probably never,

therapeutic cures quite rarely, take place. It is well to be very guarded in giving an opinion as to the duration of life, and we should never forget that even during a seemingly excellent state of health suddenly grave symptoms may develop which lead to a rapid termination.

Treatment.—Of an effectual treatment we can only speak in those cases in which an operation is feasible. Since this—trephining of the skull, splitting of the dura, opening of the abscess with the knife—must always, however, even if conducted with the strictest antiseptic precautions, be regarded as a grave undertaking, we should only resort to it when the location of the abscess has been established with some certainty. If this has been done, operative measures are at once indicated, and should be carried out without delay, provided, of course, that the abscess be in a part accessible to the knife, which, we need not say, is hardly the case in the basal ganglia, the pons, the medulla oblongata, and the cerebellum.

But, unfortunately, an operation is in the greater number of cases not feasible on account of the uncertainty in the topical diagnosis. Then our treatment can only be symptomatic, and we are confined to local bleeding, hypnotics, bromides, etc., which effect but little. For that matter the results of a so-called successful operation are not always lasting either, and repeatedly one, two, or four weeks after the pus has been evacuated an unfavorable outcome has taken place—e. g., in the case of Wernicke-Hahn (cf. lit.).

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2. Non-suppurative Encephalitis and its Consequences ("Athetosis").

A. IN ADULTS.

There is no question but that inflammatory processes, acute as well as chronic, occur in the brain which show no tendency to suppuration, although our knowledge of their pathogenesis and their symptomatology is very imperfect. These processes take place preferably in quite early childhood, or even during intra-uterine life; only exceptionally may they occur in adults, as a consequence of the abuse of alcohol. They are then circumscribed inflammatory processes, occurring partly in the

Fig. 82.—PORENCEPHALY.

cortex, partly in the white matter which admit of regeneration. If larger areas are affected, the tissue becomes shrunken and of a distinctly firmer consistence, so that it cuts almost like leather. Just how these changes are brought about, in what way the nerve fibres of the white matter waste and the connective tissue increases, which of the two processes is the primary and which the secondary, can not as yet be determined with any certainty. Peculiar disturbances in nutrition in certain areas of arterial distribution may give rise to defects which cause a distinct sinking in of the surface of the brain (Kundrat), "porencephaly" (Fig. 82). At times we find a true cicatricial tissue, which characterizes the terminal process of the diffuse cerebral sclerosis. The macroscopic appearance of the brain is similar to that in the "induration cartilagineuse" of

Cruveilhier; microscopically, the same histological elements as are seen in all degenerative processes of the gray and white matter of the brain, spider cells, and compound granular corpuscles, are noted (Kast). Marie and Jendrassik (cf. lit.) see in perivascular changes the chief factor which under certain circumstances brings about a lobar atrophy. At times we have to deal undoubtedly with the consequences of a uniform arrest of development which especially takes in one hemisphere and the anatomical cause for which is not understood. The circumscribed inflammatory foci may also be found in both hemispheres, in which case we speak of a double lobar sclerosis.

The clinical course of the disease is practically unknown. Probably there does not exist any well-defined constant clinical picture, but the symptoms vary according to the anatomical seat of the process. They are symptoms of paralysis or of irritation, and are partly " cortical symptoms " and partly to be referred to disease of the cerebral vessels. In the few cases in which a diagnosis could be made during life, apoplectiform attacks, rhythmical choreic movements, longer or shorter spells of unconsciousness, were observed. The difficulty of grouping and correctly interpreting the symptoms is chiefly owing to the impossibility of an early diagnosis. Hence it will be the chief task of future observers to direct their attention to the initial stage, for only after we have once become familiar with the development and the anatomical changes in this first stage can we hope to elaborate an efficient mode of treatment, which, we need not say, at present is absolutely wanting. The irrational trials with potassium iodide we can certainly not regard as such.

B. IN CHILDREN.

Cerebral Palsy of Children—Hemiplegia Infantilis Spastica (Benedict)—Polio-encephalitis (Strümpell).

Pathological Anatomy.—In view of the comparatively frequent occurrence of cerebral palsies in children, it is rather to be wondered at that so extremely little is known about their pathogenesis and their initial stage, more especially with reference to the anatomical changes that occur. This may, perhaps, be accounted for by the difficulty, and sometimes even impossibility, of making an early diagnosis. At a time when we are able to recognize the disease we usually have to

deal with a process which has already passed through all, or almost all, of its different stages. It is the same with the lesions which we find: they in no wise explain the exact nature of the disease, but only give us an idea of the many various ways in which the brain with its meninges may be altered in early childhood as a consequence of the disease, which was most probably intra-uterine. We do not, however, know of what nature this process is, whether it is a sinus thrombosis, as Gowers claims, or an inflammation leading to atrophy, as in meningo-myelitis chronica. Neither can we tell whether the increase in the connective tissue which has been noted by many authors is a primary one, and what part the disease of the vessels, the thickening of their walls (Hayem, and others), plays in the process; but one thing is certain, that the disease is not confined to the gray cortex alone (as Strümpell has assumed, and for which reason he has proposed the name polio-encephalitis, analogous to polio-myelitis, cf. lit.), but that the white matter as well may be implicated. This is shown by the case published by Kast (cf. lit.), and also by the following observation, which was made in my wards, and which I propose to relate here in brief, as autopsies in cases of this class are rare:

Magdalena St., twenty-one years old, coming from a healthy family, was taken sick in her second year with violent fever. According to her mother's account, she had convulsions for four days and four nights. When she wanted to get out of bed after this her left side was found to be paralyzed. Inside of three months her condition was so far improved that she could walk, although with a limp. Gradually the left lower leg became smaller and somewhat curved, and she complained of pain in the whole limb. The upper extremity did not at first take part in the atrophy; it was, however, almost completely powerless. For two years the convulsions did not reappear; but for the last four years the patient has had, on an average, about one epileptiform attack every three weeks, in which she bites her tongue and passes her urine involuntarily. The following is an extract of the note made on October 25, 1885:

Head: Right parietal region painful to percussion; in the region of the left glabella and the hairy part of the scalp, on the same side, there are several areas of anæsthesia. Pupillary reactions and movements of the eye muscles normal. Nothing abnormal in the distribution of the facial and hypoglossal nerve. On the right side hearing is much below normal, on the left there is complete deafness. On

the anterior third of the left half of the tongue taste is lost. Uvula straight and movements of soft palate normal.

Trunk: On the left half of the chest touch and the prick of a pin are not perceived; temperature sense seems decidedly subnormal.

Upper Extremities: The whole left upper extremity, including the hand, is shorter and smaller than the right; motion of the wrist, especially extension, is impaired. The hand is flexed on the forearm, and only with force can the flexion be overcome. The thumb is drawn into the hollow of the hand, the rest of the fingers are slightly flexed. Motion in the shoulder joint normal; in the elbow joint extension is slightly impaired. There is a general decrease in the sensibility. Electrical reactions are found to be normal for both currents, on direct as well as on indirect stimulation. The right upper extremity does not show abnormity with regard to development, size, mobility, or sensibility.

The left lower extremity is considerably smaller and shorter than the right; sensibility is the same as in the corresponding upper extremity; the same holds for the electrical condition. Tendon and skin reflexes are retained on both sides.

The epileptic attacks continued, and occurred about every sixth or eighth day; the intelligence became more and more impaired. A tuberculous process in the left lung was superadded to the already existing trouble, and, in consequence of general failure of strength, the patient died on March 22, 1886.

Autopsy: Eighteen hours after death. Extract from the postmortem record: After opening the skull the pia is seen to be considerably thickened at different places, especially over the right hemisphere. In volume the right hemisphere is not much smaller than the left; the anterior and posterior central convolutions on the right side, especially in their lower half, are markedly atrophic, the gyri are shrunken to about a third of their natural size; the inframarginal lobule and the angular gyrus present the same atrophic condition. The upper right parietal lobule is less atrophic, nevertheless the gyri are here also remarkably narrow. The portions of the first and second frontal convolutions bordering on the central convolution appear also atrophic. On section the gray matter is seen to be considerably diminished.

The ventricles appear markedly enlarged. On frontal sections, after Pitres' method, no important changes, with the exception of the shrinking, either in the centrum semiovale or in the basal ganglia, can be observed macroscopically; on microscopical examination spider-cells and fat-granules are found in considerable numbers not only in the gray cortex, but also in the white matter.

This observation determines us in maintaining with Kast the old designation, "cerebral palsy of children," a name by which no definite pathological change is implied, and in abandoning the term polio-encephalitis, to which the pathological changes do certainly not always correspond, especially as the latter name has already been proposed by Wernicke for the disease of the gray matter around the third and fourth ventricles.

Symptoms.—While the anatomical changes at the onset of the disease are obscure, the clinical symptoms are quite distinct and striking. The disease usually sets in brusquely, the symptoms are violent and can not be overlooked. The child is seized with a high fever; soon, sometimes only a few hours later, twitchings—at first only in one extremity, later in the whole side—appear; at times the whole body may be convulsed; this may last, with but slight interruptions, for from one to three, or even four days, and be accompanied by persistently high temperature. The symptoms now abate, the convulsions become less frequent, but after their disappearance the child is found to have lost the use of the limbs of one side —hemiplegia infantilis. If an early and careful examination be made, a moderate facial paralysis is noted, the condition of the extremities being very nearly the same as has been described on page 228. Here, as in the common cerebral hemiplegia, the arm is pressed against the thorax, the forearm flexed at right angles with the upper arm, the hand flexed and adducted, the fingers bent. The leg is slightly flexed at the knee-joint, the foot extended; not uncommonly the big toe is in marked dorsal flexion. The sensibility is, as a rule, not much altered. After several weeks the little patient regains enough power to perform the coarser movements with the leg, while for a considerably longer time the arm does not take part in the improvement. If the child was able to walk before the onset of the disease it will generally regain this faculty after a time, but its gait will always be halting.

The further course of the disease is not the same in all cases, and it has been our experience that it differs according as the initial convulsions continue or cease. This, therefore, is an important point to consider in the prognosis for the relative recovery. It will also decide the question whether the child, while bodily more or less a cripple, is in addition to be mentally defective and totally useless to the community. What

conditions determine the continuance of the convulsions, whether this is influenced more by the nature of the lesions or more by their seat, we are unable to say.

As a rule, the attacks, even if they should have a tendency to continue, do not recur for months, for one, two, or even four years, after the acute period of the disease has passed off. Then, however, they may return on any provocation—after a fright, maltreatment, sometimes during the second dentition—at first at long intervals of months, then more frequently. At first they may be slight and of short duration, then more severe, until finally they resemble in every point the classical attacks of epilepsy—in other words, the hemiplegic or hemiparetic patient has now become an epileptic. As has been stated, the influence which these attacks have upon the mental development of the child is very detrimental. Much more often than is the case in idiopathic epilepsy does the patient become weak-minded. The condition of speech found in this disease is interesting. If the patient had fully acquired speech previous to the attack, it is only affected if the lesion is on the left side of the brain, in which case the symptoms do not differ from those which we have described under left-sided cerebral hæmorrhage. If, on the other hand, the patient has not yet learned to speak, he will, in case the fits continue, either not learn at all or only very imperfectly, and his talk will, even if his mind is only slightly impaired, be quite unintelligible ; but often the attacks do not recur, so that the mental development progresses normally. In such cases speech likewise reaches a gratifying degree of development even if it had not yet been fully acquired or had again been lost. The healthy hemisphere takes on vicariously the work of the injured one (cf. page 181, remarks on aphasia of children).

Independently of the epileptiform attacks there may occur changes in the extremities, which are in a way analogous to those described on page 228. An especially characteristic symptom is the pronounced spastic condition which manifests itself in an increase of the reflexes, rigidity and spasm of the muscles—hemiplegia infantilis spastica. This rigidity is especially well marked in the muscles of the hand and the calves, and leads, preferably in the former location, to contractures, which, however, differ from others inasmuch as they cease during rest and sleep and only appear on voluntary motion. Benedikt, above others, has pointed out that at one time one,

at another time another, group of muscles may be affected; that, e. g., in walking the foot may be held normally, while again in the same foot we may see a talipes calcaneus, or at another time a talipes equinus. Similar conditions are observed in the hands; thus the fingers, which appear to be in a state of immobile flexor contraction, may at other times present a remarkable degree of mobility. Under what circumstances contractures appear, why sometimes they are present, sometimes absent, is not known. Perhaps the extent of the cerebral lesion and the secondary degeneration in the corticomuscular tract depending on this has something to do with it. We shall shortly discuss carefully the entirely involuntary movements of the affected hand which are noted in the course of this disease.

In almost all cases of infantile cerebral paralysis an arrest of development or growth becomes apparent in the affected extremities. This may be only insignificant, so as to be hardly appreciable. On the other hand, the limbs may in all their dimensions be considerably smaller than the corresponding ones of the sound side. Occasionally the whole half of the body, trunk and head as well, share in this arrest, and we have what is called a general hemiatrophy.

The following illustrations of cases from my clinic represent different types of the cerebral paralyses of children :

Figs. 83 and 84 : Hemiatrophy of the whole left side of the body. Epileptiform attacks. Dementia.

Figs. 85 and 86 : Hemiatrophy of the whole left side of the body. Cause : Traumatism. No fits. Intelligence normal.

Fig. 87 : Atrophy of the left upper and lower extremity (resection of the knee-joint). Epileptiform attacks, with a moderate degree of dementia.

Figs. 88 to 91 : Atrophy of the paralyzed side, very slight, but perceptible. All three patients suffer from epileptiform attacks and are demented. All three present contractures on the affected side, either in the wrist (Fig. 88 and also Fig. 83) or in the ankle-joint (Figs. 90 and 91).

All these eight cases, which came under my observation, depended upon disease of the right hemisphere. Whether this side is altogether more frequently attacked, and, if so, how the fact is to be explained, I dare not at present decide.

Diagnosis.—The diagnosis of the disease is, as a rule, easy, as the acute onset with the consequent hemiplegia is charac-

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teristic enough ; but if the patient come from a phthisical family and is himself tuberculous some doubt may arise. We may have a case of tuberculosis of the brain to deal with, which sometimes resembles in its onset the cerebral palsy of

Fig. 83.

children. High fever and convulsions are not absent and severe motor disturbances occur also. The fact, however, that in cerebral tuberculosis generally the base of the brain with its nerves, especially the oculo-motor and abducens, are implicated, and, further, that it runs a rapid and fatal course, will enable us to make a correct diagnosis.

Spinal and cerebral infantile paralyses can not be confounded with each other if we keep in mind that in the latter one whole side of the body is affected ; that the muscles are rigid, the reflexes increased ; that convulsions occur not only

at the onset, but also in the further course of the disease ; that the mind becomes impaired, etc. In the spinal form, either one limb alone—arm or leg—is affected or both arms or both legs, and the reflexes in the paralyzed extremities are lost,

Fig. 84.

The patient, who is now thirty-six years old, was taken ill in early childhood with an acute violent fever and convulsions; the latter lasted for several days, but after that disappeared. From that time the left side did not develop as well as the right, so that now the left upper extremity, which can be moved, with difficulty only, in the shoulder and elbow joints, is seven centimetres shorter than the right, while the left lower extremity is three centimetres shorter than its fellow. The whole half of the body has shared in the atrophy, which is also well marked in the nates. The circumference of the left upper arm measures four centimetres and a half, that of the left leg three centimetres less than that of the corresponding extremity. When the patient was fourteen years old the convulsions reappeared, and he has still one or two epileptiform attacks a week. He is quite demented.

signs enough to enable us to differentiate between the two affections. A hemiplegia due to cerebral hæmorrhage can in most cases be excluded, owing to its rarity in childhood. Such, moreover, would usually not be associated with any muscular atrophy.
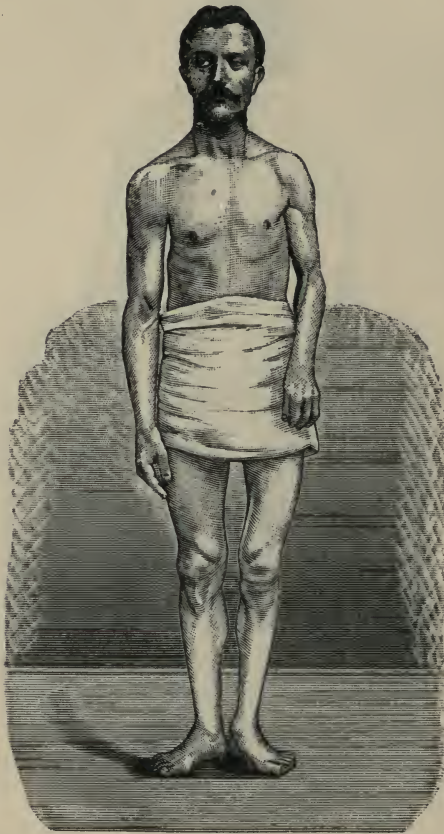

Fig. 85.

Prognosis.—The prognosis *quoad valetudinem* is absolutely, *quoad vitam* relatively unfavorable. Complete recovery is impossible, and has never been observed. If the patient does not succumb during the first days of the disease, he will remain a cripple all his life, his mental condition being good only in the most favorable cases. Under unfavorable conditions he may be epileptic and weak-minded, and to a greater or lesser extent deprived of the use of his limbs. The utmost we can expect is that the diseased side may atrophy only to a moderate degree,

that the patient may be sound enough in other respects, bodily and mentally, and thus be capable of making his own living (Figs. 85 and 86).

Treatment.—The treatment is, on the whole, entirely unsatisfactory. Even by the light of an early diagnosis we are
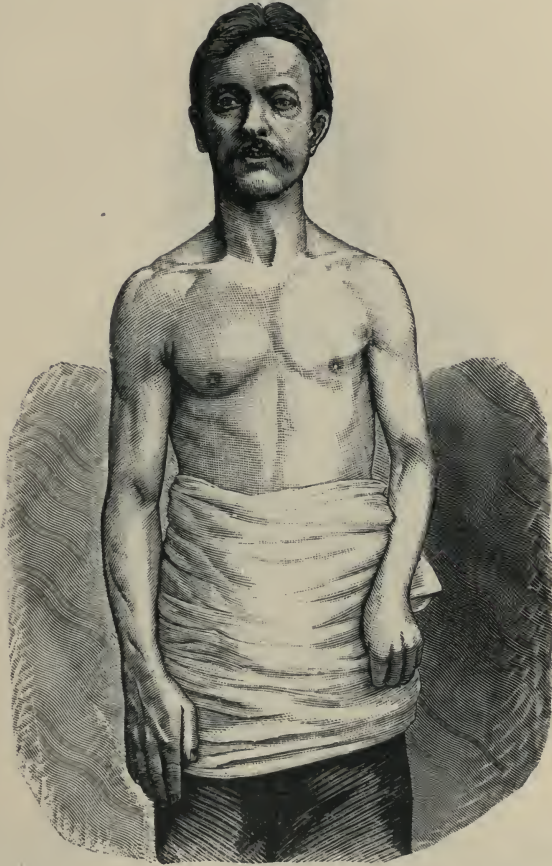
Fig. 86.

The patient now thirty-four years old, fell, at the age of three, from a high foot-stool and injured the right side of his head. He lost a considerable amount of blood, and was unconscious for quite a long time. Six months after the injury the atrophy of the left side of the body became apparent, first in the upper, then in the lower extremity. With the exception of this atrophy, which has now taken in the whole half of the body, including the thorax (compare the left with the right mamma), the patient is perfectly healthy. He has never had epileptiform attacks, there are no hemiathetoid movements, and no psychical alterations whatever.

not in a position either to prevent the continuance of the epileptiform attacks or to ward off the changes in the affected extremities, the symptoms of irritation, the atrophy, etc.   The

Fig. 87.

The patient, now eighteen years of age, had, when six months old, an "apoplectic stroke," and never learned how to walk properly, since the left half of the body was paralyzed up to her second year.   The power of motion has improved to a certain extent; the left leg, however, and the left arm have remained behind in development, so much so that the arm is eight centimetres, the leg twenty-five centimetres shorter than the corresponding limb of the right side. The shortening of the leg is partly due to a resection of the knee joint performed thirteen years ago (the reason for which procedure could not be made out).   Patient suffers from epileptiform attacks, occurring once a month; they last from a quarter to three quarters of an hour, and consist of more or less violent convulsions.   During these, consciousness is sometimes completely retained.   There is no trace of dementia.

symptomatic treatment of the epileptiform attacks by the different bromides and the galvanization of the atrophic parts is all that lies in our power, and, unfortunately, little enough is accomplished by these means.

Fig. 88.

The patient is now forty-four years old. The date of onset of the disease can not be definitely determined. She suffered from epileptiform convulsions from early childhood up to her tenth year; these have now entirely disappeared. At times, however, a "*tic convulsif*" (in the distribution of the left facial) is noted. The development of the left half of the body has been retarded, the upper extremity being two centimetres, the lower three centimetres shorter than the corresponding limb of the right side. There is also a difference of from four to five centimetres in the circumference of the limbs of the two sides. The shoulder, elbow, and wrist joints are contracted, the first being in a position of adduction, the second in one of flexion, and the third in extension. Marked degree of dementia.

Fig. 89.

The patient, who is now twenty-two years of age, was taken ill with convulsions in early childhood. They ceased, but after an interval of ten years reappeared in his fourteenth year, and have continued up to the present time, being quite severe and recurring quite frequently. From childhood he has suffered from a severe motor speech disturbance, and is only able to utter a few unintelligible syllables, and that with great effort; at such times almost all the muscles of the body are affected with associated movements. Atrophy of the left side is to be noted. The circumference of the left upper arm measures three centimetres, that of the left forearm two centimetres, that of the thigh four centimetres, and that of the leg two centimetres less than the corresponding measurements on the right side. The left arm is one centimetre, the left leg one centimetre and a half shorter than the right arm and right leg respectively. The left hand and fingers are in flexor contraction. Patient is moderately demented.

Ætiology.—Although nothing certain is known about the ætiology, it seems highly probable that the disease is of an infectious nature. In favor of this idea is the fact that it not rarely follows acute infectious diseases, especially scarlet fever. It has also been recorded after whooping-cough (Marie, cf. lit.). That traumatism may be the causative factor is proved by the case represented in Fig. 86. The patient at the age of three fell head first from a chair and injured himself in the right parietal region by striking against a sharp object. If the traumatism occur during intra-uterine life or soon after birth, the skull may become asymmetrical and the affected side often remain behind in development. Syphilis also deserves a place in the ætiology of infantile cerebral paralysis.

"**Athetosis.**"—It remains now for us to describe those peculiar involuntary movements above referred to which are known to follow cerebral infantile paralysis. The patients are absolutely unable to keep the fingers and the toes of the affected side still; they are in constant motion day and night, during waking and sleeping, without interruption. If we observe these movements more closely, we find them to be relatively slow, rhythmical, and monotonous. The fingers seem to be directed with a definite aim, as if they were attempting to seize something, and it is easily remarked that the normal limits of the movements are exceeded—the fingers are hyper-extended, the toes are elevated almost at right angles or fasten themselves to the floor like claws, etc. (cf. Fig. 92). All this is only possible in consequence of an unusual stretching of the ligaments, which also admits of positions of distinct subluxation. The will of the patient has hardly any influence over these movements, and only in light cases, and then but temporarily, may the patient succeed, by firm pressure of the affected hand upon the body, or by fixing the fingers with the unaffected hand, in restricting a little the abnormal excursions; as soon as the mechanical impediment is removed, they will, however, begin again with increased vigor.

The muscles of the forearm present a firmer consistence, a certain degree of hypertrophy. The arm feels hard and the surface temperature is 0.5° to 1° C. (0.9° to 1.8° F.) higher than on the opposite, sound, side; not but what the muscular strength is materially lessened and sometimes so much diminished that the examination with Duchenne's dynamometer yields astonishing results. With the affected arm the patient can hardly

lift five kilogrammes, notwithstanding the apparently good de-
velopment of the muscles, while with the well arm five to eight
times as much work can easily be done.   In the muscles of the
lower extremity a similar condition may be noted; not infre-
quently the ankle joint takes part in these movements of the
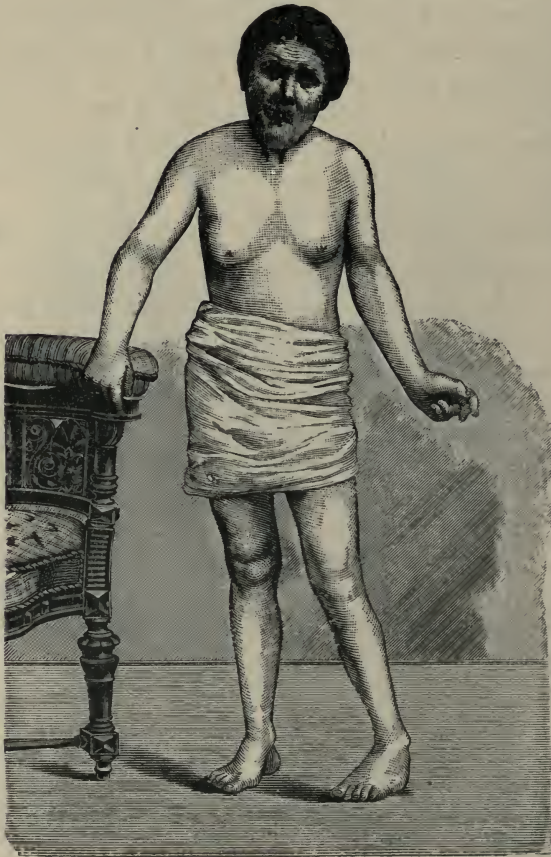

Fig. 90.

toes, and, in exceptional cases, the knee-joint as well.   Other
muscles than those of the extremities are not affected.

   The first who studied these movements carefully was Ham-
mond, of New York, in 1871.   He gave them the special name
athetosis (*a-τίθημι*) and raised them thus to the dignity of a sepa-
rate disease, which, in our opinion, they never deserve.   Athe-
tosis—and by this we mean the athetoid spasms—does not con-

stitute a disease, but merely a symptom. It is the expression of cerebral affections, the anatomical basis of which is variable. Only in the rarest instances, one could almost say never, do athetoid movements occur alone without any other symptoms. Almost always they are associated with other disturbances, either psychical (the patients are mentally undeveloped, de-

Fig. 91.

The onset of the disease can not definitely be fixed, since the mother of the patient does not remember it, and the patient herself, who is now eighteen years old, is demented and completely deprived of the power of speech. The fact, however, that the illness began in early childhood with convulsions is un-questioned; it is, however, not known how long they lasted nor what followed them. When the girl was five years old she was not yet able to walk, because the left leg was moved only with difficulty, and the foot gradually assumed an equino-varus position, which can still be noted. Patient now walks on the outer edge of her foot, and the leg is scarcely moved at the knee joint. The left upper extremity can be moved voluntarily in the shoulder and elbow joints; the fingers and the hand present athetoid movements, while in the facial mus-cles of the left side a marked "*tic convulsif*" is noted. Marked dribbling of saliva. Patient no longer suffers from epileptic attacks, but has from time to time periods of excitement, during which she becomes aggressive.

mented, sometimes of a changeable, irritable disposition) or somatic, such as paralyses or spasms in the distribution of different nerves—for instance, the facial—or contractures, etc. Again, the patient may be subject to epileptiform attacks which recur at intervals of various lengths.

If we thus affirm that every athetosis—be it the much rarer bilateral form (cf. the case of Bourneville and Pilliet, lit.), be it unilateral, the "hemiathetosis"—is only to be regarded as a symptom, we are, on the other hand, willing to admit that there are individual cases where the athetoid movements are such a prominent and dominating feature of the case that we may overlook others, or at least not be inclined to attribute any importance to them. So it is in an instance reported by Gnauck, who speaks of a primary—that is, idiopathic—athetosis, but who has noted a simultaneous paresis of the facial and a hemianæsthesia of the affected side. We can hardly call this an idiopathic affection, but must rather look upon it as a pre-hemiplegic phenomenon (cf. page 220); and, similarly, some explanation can be found for the few remaining cases published as "idiopathic" athetoses, some of which were congenital. These movements are always a symptom of cerebral disease. That they are occasionally met with in the course of other diseases—e. g., spinal affections, especially tabes—there can be no doubt. What is the pathological nature of the brain disease which gives rise to these spasms is still hypothetical. There are cases where no lesion at all was found at the autopsy. One thing, however, we know for certain, namely, that these movements may occur in different cerebral lesions—that is, that they constitute a symptom which is not confined to one particular disease, but is common to several. Among these, cerebral infantile paralysis, with the anatomical changes described on page 268, deserves the first place. Here athetosis (always only hemiathetosis) is relatively frequent, and here we have the best opportunity for studying its peculiarities. It has also been observed in connection with small foci of softening in the basal ganglia, the thalamus (Lauenstein), the corpus striatum (Schulz), and in the temporal lobes (Ewald), although we can in none of these instances be certain that the lesions found were actually the cause of the movements. After cerebral hæmorrhage where we have a lesion of the internal capsule, in old hemiplegias therefore, hemiathetoid movements are occasionally seen, yet, in comparison with the frequency of cere-

bral hemiplegias in adults, these are very rare, certainly much rarer than in the so-called infantile hemiplegias. We see, therefore, that cortical lesions and lesions of the cortico-muscular
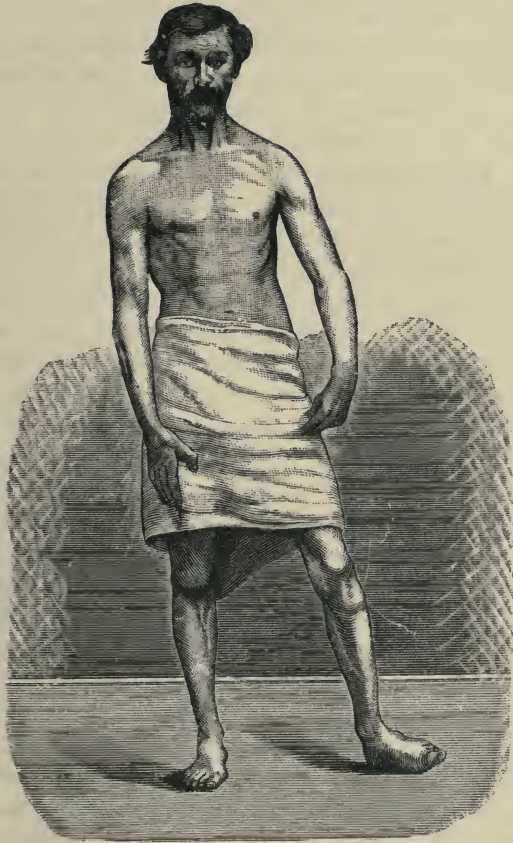

Fig. 92.

The patient, now twenty-nine years old, was taken at the age of six months with an "apoplectic stroke" followed by convulsions, which at first occurred at long intervals, later more frequently, viz., about once every two weeks; they presented all the characteristics of epileptiform seizures. Quite early, peculiar involuntary movements appeared in the left extremities, more particularly in the left arm, which must be considered as athetoid. At regular intervals the fingers are extended and again drawn into the hollow of the hand, this being repeated about fifty times a minute. In the left foot similar, although, of course, less pronounced movements, occurring especially in the ankle joint, are noted. At about the age of five the convulsions reappeared, although occurring with diminished frequency, i. e., from three to five times a year. The patient is excitable, irascible, and at times even violent. Intelligence is normal.

tract as well as of the basal ganglia may give rise to athetoid movements, although we do not understand the *nexus causalis*, if indeed such exist.   In our opinion, disease of the cortex undoubtedly plays the principal part in the causation of athetosis, and we can all the more reckon upon the occurrence of athetoid movements if the cortical disease has appeared in early childhood and has been either entirely confined to or has affected more particularly the motor region, the central convolutions, and the adjacent portions.   In lesions of the other parts of the brain, especially of the basal ganglia, the thalamus, the lenticular nucleus, and the caudate nucleus, athetoid movements are only exceptionally developed, the conditions which favor their occurrence being then wholly unknown.   That there is a cerebral lesion which produces no other symptom, whether psychical or somatic, than these movements is unlikely, and consequently, as we said, the name "athetosis," as indicating a separate disease, can not be held to be justifiable.

If one keeps, then, the characteristics of the movements which have just been described well in mind, and especially if one has had occasion to study their peculiarities, one can hardly mistake them for anything else.   A good point to remember is that they continue during sleep, so that the patients have to stop or at least impede them by mechanical appliances.

We shall give up the idea of chorea or hemichorea which we might entertain should the athetoid movements be accompanied by facial spasm, if after observation of the patient we have been convinced that the movements persist when the patient is asleep.   Furthermore, the duration of the disease and the fact that it resists all therapeutic measures, more especially the protracted use of arsenic, are facts not reconcilable with the diagnosis of chorea.   Other points of difference will be found in the chapter on the latter disease.

We can not speak of an ætiology of the affection, because, as we have said, athetosis is not an affection in itself, and the ætiological data therefore to be considered are those of the essential disease upon which the athetoid movements depend.

We possess no specific which will put a stop to these athetoid movements; their treatment is that of the primary disease, and, as this is usually beyond our reach, the outlook in athetosis is necessarily very gloomy.   If Hammond claims to have effected a cure by stretching the median nerve, we may be pardoned for asking how long this cure lasted, and if

Gnauck has seen the movements disappear after the use of the galvanic current and the internal administration of potassium bromide, we are justified in assuming that in his case the affection was due to a functional disturbance of the motor area. What lasting good results can be accomplished by hyoscine, a drug which has been used by Erb, I have not been as yet able to establish with the material at my disposal.

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BRAIN TUMORS.

Pathological Anatomy.—Brain tumors may be either sharp-
ly circumscribed or diffuse, in the latter case taking the place,
as it were, of the brain substance proper.   The most common
—e. g., the gliomata, the carcinomata, and the sarcomata—

Fig. 93.—GLIOMA TELANGIECTATICUM. (After ZIEGLER.) Frontal section through the
brain.   *a*, right centrum semiovale.   *b*, glioma in the left hemisphere.

occur in both varieties.   The clinical manifestations of brain
tumors depend upon the rapidity of their growth ; this,
again, upon their anatomical nature.   Among the most im-
portant and the most frequent forms of tumors must be men-
tioned :

The glioma, a form which is peculiar to the central nervous
system, but is found much more frequently in the cerebrum
than in the brain stem or the spinal cord.   It is formed by
an increase in the cells of the neuroglia, the axis cylinders
in the involved region first becoming swollen, and the nerve
fibres then destroyed.   If the newly formed cells are small
and comparatively few in number, and if their fibrilla-like
processes form a dense net-work, then the tissue of the growth

is firm and solid; if the cells are numerous the tissue is softer. On section, the glioma looks gray, grayish-red, or yellowish, sometimes variegated, and if, as is not uncommonly the case, it contain areas of hæmorrhagic softening, the tumor may be filled with opaque more or less fluid masses. The diameter of a glioma may measure from three to eight centimetres. The transition into the adjoining substance of the brain may be gradual or abrupt, and the tumor appear macroscopically sharply defined. The affected part of the brain is enlarged, but keeps its normal configuration while the ventricles are often dilated (Fig. 93).

The tumor nearest related in texture to the glioma is the sarcoma; it occurs in soft nodes, which, on section, present a marrowy, grayish-white appearance. It is seen much more

Fig. 94.—PAPILLARY CARCINOMA IN THE THIRD VENTRICLE. (After ZIEGLER.) Frontal section through the brain. *a*, tumor with cysts. *b*, right thalamus. *c*, lenticular nucleus. *d*, internal capsule. *e*, caudate nucleus. *f*, left thalamus. *g*, lenticular nucleus. *h*, internal capsule. *i*, dilated lateral ventricle.

frequently at the base than at the convexity of the brain, and not uncommonly is found to originate from the dura, from the periosteum of the skull bones, or from the skull itself (osteo-sarcoma). According to the character of the cells, we distinguish a round-cell sarcoma, a spindle-cell sarcoma, a fibro-sarcoma, etc. In size they may vary from that of a walnut to that of a man's fist, and may be solitary or multiple.

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The carcinoma, which appears usually in the brain as secondary to carcinoma of the breast, lung, or pleura, is found especially in the ventricles as a soft tumor (cf. Fig. 94), displacing the neighboring brain substance, and giving rise to hydrops ventriculorum.

Clinically of great importance are the tubercles and the syphilomata (gummata), which, although they show macroscopically as well as histologically much similarity, can with certainty be distinguished by the presence or absence of the tubercle bacilli. They also may be either sharply defined or infiltrate the tissue; they appear on section as yellowish, cheesy tumors consisting in part of granulation tissue. The "solitary tubercles," which may reach the size of a hazelnut, are single or multiple; they occur by preference in the pons, in the cerebellum, and in the cortex. Syphilomata more frequently originate in the dura mater, and thence invade the brain substance.

The psammomata, which, coming also from the dura, are characterized by calcareous concretions imbedded in them; the cholesteatomata, which on section have a lustre like that of mother-of-pearl; the lipomata, often found in the corpus callosum; the enchondromata, which originate especially from the bones of the base—all these are clinically of little importance, as they produce, owing to their relatively small size. either only insignificant or no symptoms at all. Hence we may well omit them in our description.

At the autopsy we can often demonstrate the consecutive changes produced by a general compression of the brain. The skull bones themselves in young people may appear perforated and riddled with holes, there may be gaps in the dura or signs of inflammatory irritation, certain areas may be rough and thickened, presenting a velvety appearance, the convolutions, flattened and pressed against each other, have lost their distinctness, the pia looks dry and anæmic. Certain alterations of shape seem always to occur if the pressure reaches a considerable degree; thus a pressure in one hemisphere exerting itself from above downward changes more especially the shape of the insula and the portions of the temporal and parietal lobes which cover it in (Wernicke). This effect must be attributed not only to the increase in volume of the tumor, but also to the increased amount of the fluid in the ventricles, the hydrops ventriculorum (internal hydrocephalus) which almost con-

stantly accompanies tumors. No doubt this internal hydro-cephalus itself is due to pressure on the venous trunks in the brain, and it occurs, therefore, earlier, and is more marked if the large venous trunks coming from the tela choroidea are pressed upon by the tumor (Wernicke).

On the cranial nerves signs of pressure have also been noted. The optic tract, the oculo-motorius, the abducens (Türck) have been found compressed by tightly stretched vessels, and an exudation into the sheath of the optic nerve has been observed (Leber). In some cases we find a more or less widely spread softening in the parts surrounding the tumor, in others this may be entirely absent; if the softening is of a hæmorrhagic character, this must be attributed to a cutting off of the arterial blood supply produced by the cere-bral compression and to venous stasis. Sometimes, in the neighboring vessels, there develops an arteriitis obliterans with its sequelæ (C. Friedländer). Cranial nerves in the immediate neighborhood of carcinomata and syphilomata are found to be infiltrated with the tumor elements (Wernicke).

Ætiology.—The ætiology of brain tumors is entirely ob-scure: we do not know in the least whether certain external influences increase the predisposition to tumors in the brain or not, just as we are entirely ignorant of the ætiology of tumors in general. Although the common idea exists that traumatism may be the starting point for a new growth, it is difficult to understand the connection; certainly, however, this factor plays an infinitely smaller part in tumor than in brain abscess, and the occurrence of a brain tumor following an injury is probably for the most part accidental. No doubt, in some kinds, hereditary predisposition must not be disregarded, as in carcinomata and tubercles, but even this loses some of its significance, because malignant brain tumors, especially car-cinomata, are usually secondary, as we have said. Nothing remains, then, but to inquire how far age and sex influence their occurrence. With reference to the former, it is supposed that some brain tumors, such as tubercles, predominate in the young, while carcinomata and sarcomata are chiefly found in older people; others—e. g., myxomata and sometimes gliomata —are congenital (Virchow). As to sex, older and more recent authors (Lebert, Friedreich, Hasse) agree that males are more liable to brain tumors than females, and Wernicke has calcu-lated that the proportion is about three to two.

Symptoms.—The symptoms we are wont to observe in brain tumors are due to the mechanical influence which the tumor exerts by general or local compression of the skull contents, and, further, to destructive or irritative actions which depend upon certain vital peculiarities of the growth, the irritation mostly accompanying the infective neoplasms. One or the other of these just-mentioned factors will influence the clinical picture of the disease in a more or less characteristic manner, and as one or the other is more prominent the whole aspect of the disease will vary.

With reference to the former, the increased intracranial pressure, if it appears acutely, we have first a displacement, then an increase of tension in the cerebro-spinal fluid. In chronic processes the latter does not necessarily occur, but as the skull cavity gradually becomes encroached upon, some of the fluid may be absorbed or the brain become atrophic. As the intracranial pressure becomes higher the circulation in the brain and its membranes is retarded. What is the cause of this retardation, whether the diminution in the tone of the vessel walls produces such an increase in the tension of the cerebro-spinal fluid that by compression a narrowing in the capillaries is produced or whether fluxionary hyperæmias come into play, we are not able to decide definitely. At any rate, if the blood current in the interior of the skull frequently undergoes a slowing, there is a tendency to increased transudation and lymph formation, and with it a danger of œdema of the brain (cf. von Bergmann, Die Lehre der Kopfverletzungen, Stuttgart, 1880, pages 316–364).

The symptoms to which this increase of the intracranial tension gives rise, and which one has frequently the opportunity of studying in the course of brain tumors, may be divided into general and focal. The former, for the knowledge of which we have to thank especially Leyden, Manz, and Duret, usually appear in a regular sequence and are always the same for the same degree of pressure.

The most conspicuous and earliest to appear is the headache. The patient complains of nothing but his head, which feels heavy and dull. Every movement causes pain, and this becomes at times so violent that the patient feels as if he were losing his reason. The pain seems diffuse and can not be localized. It is in front on the forehead, behind over the occiput, to the right, to the left; it torments him everywhere, and the

lightest tap with the finger anywhere upon his head is intensely disagreeable. Sometimes there comes an hour or two of relief, although the patient feels by no means well and is never without pain even in sleep. The seat of this pain, which is due to the general increase of the intracranial pressure produced by the tumor, is not known. It is, however, not likely to be in the substance of the brain itself, unless it be perhaps in the corpora quadrigemina and the thalami. We should rather look for its position in the dura, which derives its nerve supply from the trigeminus (cf. page 3). If the fibres of this nerve are compressed by the tumor in the posterior fossa, then there is not the vague pain taking in the whole head, but another well-defined headache referred by the patient to the back of the head and neck only, a trigeminal or occipital neuralgia which is not a general but a focal symptom. This double significance of the headache may become a very valuable point in the topical diagnosis. Entire absence of headache is rare, and we are only without this symptom when the growth of the neoplasm is slow. Its occurrence with unwonted vehemence has repeatedly been noted in aneurisms situated near the dura. Occasionally it disappears when definite focal symptoms become established, and it becomes more obscured in the later stages of the disease, when the patient becomes somnolent. Its existence is then only apparent from the fact that the half-unconscious sufferer frequently puts his hand to his head and moans.

A second general symptom is afforded by the epileptiform convulsions, which either affect the whole body or are confined to one side and during which consciousness may or may not be completely lost. They are by no means so frequently associated with brain tumors as headache, still their occurrence is common enough to be of diagnostic value. They, too, may constitute a focal symptom, as is, for instance, not rarely the case in cortical tumors of the frontal or parietal lobes, which, partly, exert local pressure, partly, irritate the cortex. We must not suppose that these two symptoms, although they are both of an irritative nature, always go hand in hand. Either one or both may be present, sometimes the one as a general, the other as a focal symptom. Convulsions occur in about fifty per cent of all cases of brain tumors.

The psychical changes constitute a third general symptom, which, however, disturbs less the patient himself than his

friends.   A certain slowness in thinking is occasionally noticed in the patient, at first temporary, but later more constant—an inability to appreciate properly the commonest details of daily life which had never been before remarked in him.   At the same time the features become dull and lose their animated expression, his movements become slow and awkward, he grows careless in all his doings, and this listlessness about everything going on around him may be carried to such an extent that he lets his urine and fæces pass from him, without showing any concern, or attempting to satisfy his needs in a proper manner. Gradually he begins to show occasional signs of bewilderment. Things that he meets with every day he no longer recognizes. His own house seems strange to him, he forgets the way to his dining and bed room and has to be shown there, etc. He even forgets how to read and to write, how to solve the simplest mathematical problems which would not give the slightest difficulty to an eight-year-old child, and gradually he becomes more and more demented, until this condition passes into one of deep coma and death.   In other cases the intelligence seems to remain intact for a long while, and only the weakness of memory strikes one.   The friends of the patient become alarmed on noticing that he forgets things which he has said or done only one or two days or even a few hours before, that he does not remember the visits of the physician who comes daily, but complains of not having seen him for a long time.   Yet although he may be troubled with bodily pain, the patient may seem at the same time cheerful, inclined to jest, and to look at things from the humorous side, and it is not until later that the other mental defects also begin to show themselves, and not infrequently the physician is not consulted until the friends discover that the patient is no longer capable of conducting his own affairs.   Actual speech disturbances do not usually occur.   Certain peculiarities of speech which do come on and make it different to that in health are due to the extensive loss of memory of the patient, owing to which he has difficulty in finding the right expressions, and often mixes them up, etc.   This makes him uncertain in speaking.   He talks slowly, and his deliberation becomes quite noticeable.

In consequence of the increased intracranial pressure, not rarely disturbances in the sensorium occur.   The patient is in a dazed condition, has a constant desire to sleep, and is drowsy. The pulse then is often slow at first (forty-five to fifty-five beats

per minute) and irregular, similar to that which we may observe in apoplexy. This retardation is finally followed by an increase in the frequency in the number of beats. In other words, the primary irritation has given way to paralysis of the vagus.

Together with the action of the heart, respiration is affected. During coma it is deep, slow, and often stertorous; with the continued increase of the cerebral compression it becomes irregular and shallow. Deep inspirations are interrupted by long pauses, in one of which the patient dies.

Slight vertigo, sometimes attended with vomiting, is not uncommon. The latter, which is cerebral in origin, has certain peculiar characteristics. It usually occurs on the slightest provocation. It may be provoked by a simple change in the position of the body, and often comes on in the early morning and without the existence of any stomach trouble. Without any retching large amounts of watery clear stomach contents are repeatedly thrown up, and after a short while the patient feels perfectly well. Sometimes the vomiting is the forerunner of apoplectiform attacks, in which the patient may be unconscious for hours. Such attacks are due to a sudden increase in the intracranial pressure, either from hæmorrhage into the substance of the tumor or from sudden hydrocephalic exudations (Wernicke).

That papillitis is extremely common in brain tumor we have said before. We may add here that it may exist without headache, for the increase in the intracranial pressure sufficient to produce papillitis does not necessarily produce an appreciable irritation of the dura, and, on the other hand, if headache exists without papillitis, it is not referable to the cerebral compression but to irritation of the dura. We should never forget that papillitis may exist without any visual disturbance, and hence never omit the ophthalmoscopic examination in suspicious cases, no matter whether the patient complains of trouble with his eyesight or not. Again, the patient may only complain of one eye, while the other seems to perform its function normally, and yet profound changes be found in either fundus.

If in the course of a brain tumor the patient develops in addition to papillitis an early blindness, then the amaurosis has to be interpreted as a focal symptom, and the tumor located in the cerebellum, as neoplasms in this situation are usually at-

tended with very marked internal hydrocephalus, especially of the third ventricle, the floor of which becomes distended and presses upon the chiasm situated under it (Türck). Moreover early amaurosis may be produced by tumors in the region of the corpora quadrigemina, especially those of the pineal gland, by basal neoplasms, which, just as those of the pituitary body, press on the chiasm and the beginning of the optic tract, or which raise the base of the brain from the base of the skull, so that the artery of the corpus callosum is made tense and compresses the optic nerve (Türck).

In considering the focal symptoms produced by brain tumors we must first of all state that these may be entirely absent, just as we have seen is sometimes the case in brain abscess. Instances of this kind have repeatedly come under observation, and it was on this very account found impossible to make a certain diagnosis during life. Absence of both general and focal symptoms is very rare, and only possible when the new growth is very limited, and situated at an indifferent place. Further, there are symptoms which we are justified in taking for focal symptoms, but which are in reality due to the general compression. The most important of this nature is hemiplegia. We may in a case of brain tumor find a well-marked hemiplegia, which persists without any amelioration, and be induced to call it a focal symptom, and yet, to our surprise, at the autopsy a tumor may be found in an entirely indifferent area—for instance, in the white matter of the frontal lobes—a connection which we could not reckon upon. An instance of this nature I had published in an inaugural dissertation. This was the case of a man fifty years of age who suffered from mitral insufficiency, and who was seized with a grave right-sided hemiplegia which persisted unchanged for months, associated with speech disturbances. Papillitis could never be demonstrated. The case was then supposed to be one of embolism in the left middle cerebral artery, but at the autopsy a round-cell sarcoma the size of a walnut was found in the white matter of the frontal lobe, in the pars frontalis media of the left hemisphere (Steinberg, Beitrag zur Localisation der Hirntumoren. Inaugural dissertation. Breslau, 1886). For the hemiplegia to be uncrossed—that is, to be situated on the same side as the tumor—is certainly very exceptional; in our case it was crossed. If focal symptoms make their appearance comparatively early we mostly have to do with basal tumors which

produce fatty degeneration and gray atrophy of the involved cranial nerves, notwithstanding the no inconsiderable power of resistance which such nerves possess. Besides the optic (unilateral papillitis) and the oculo-motor (ptosis), the fifth, the facial, the abducens, and the hypoglossus are then relatively frequently affected. Of the fifth, usually only the sensory portion is implicated; sensory disturbances in the face, tic douloureux, later anæsthesia in its area of distribution, occur much more frequently than paralysis of the muscles of mastication. The facial is, on the contrary, affected in its whole distribution, a fact which, in conjunction with the reaction of degeneration in the paralyzed muscles which also exists, is characteristic of the peripheral origin of the paralysis (cf. page 89). The whole hypoglossus is involved, which causes not only the tongue to be protruded to one side, but also leads to atrophy in the affected muscles; swallowing, mastication, and speech, are necessarily affected by this. The hypoglossus paralysis, however, is much rarer than that of the facial. Combined affections are found:

(*a*) Of the olfactory, the optic, the oculo-motor, and the first branch of the fifth in tumors of the anterior fossa.

(*b*) Of the chiasm, the oculo-motor, the first branch of the fifth, and the abducens in tumors of the pituitary body.

(*c*) Of the oculo-motor, the patheticus, the chiasm, in tumors of the middle fossa, if situated above the dura, of the three ocular nerves and the quintus, if situated below the dura; and finally,

(*d*) Of the facial, the trigeminus, the auditory, the glossopharyngeal, the vagus, the accessorius, and the abducens in tumors of the posterior fossa.

Diagnosis.—It is the object of our diagnosis in a given case to determine first the presence, then the position, and finally the nature of a tumor. The first question can, as is apparent from what has been said, by no means always be answered with certainty; especially is this difficult if either only general or only focal symptoms are present. Among the former, headache, we have said, plays the most important *rôle*. It may last for years without any other signs to lead us to suspect a tumor, and it is in such instances that we can easily understand how this may be mistaken for simple habitual headache or hemicrania, where the pain may also attain an almost unbearable intensity. Yet in hemicrania and its allied affections there

occur remissions, and there are considerable periods of time during which the patient is perfectly free from pain ; whereas in the course of a brain tumor this never happens. Here we find no intervals of relief, but the patient's sufferings are uninterrupted. Moreover a headache, no matter how severe it be, which is materially improved by the exhibition of salicylates, bromide, or caffeine, etc., we can hardly refer to a serious organic brain disease. If, however, it persists uninfluenced by all the ordinary therapeutic measures, this ought to put us on our guard, and make us look further for focal symptoms— unilateral papillitis, for instance—which may be present; yet we should, on the other hand, not lose sight of the fact that there are quite a considerable number of cases of pure migraine which do not yield to remedies, and which have to be regarded as incurable.

Convulsions, although less often than headache, may be the only striking symptom. If they last for months, appearing at moderately long intervals, we may, in the absence of any other symptoms pointing to a tumor, think of idiopathic epilepsy. Here, also, the therapeutic test may throw light upon the subject. Large doses of bromide usually diminish the frequency as well as the severity of epileptic attacks, at least for a time, and the favorable influence of the drug is often, indeed, quite striking, while if the seizures are due to an organic cerebral lesion, bromides, even if they be continued for a lengthened period, have but little effect. Such fruitless trials should direct our attention again to the possible existence of a tumor, and lead us to search for further symptoms which may help the diagnosis.

If the patient complains of nothing further than attacks of vertigo and vomiting, if psychical changes, headache, and convulsions, are absent, then the diagnosis remains uncertain, because vertigo can be produced by many different causes, and cerebral vomiting is met with in affections so different from one another that it is simply impossible to diagnosticate a brain tumor from these two symptoms alone. They even do not necessarily indicate a brain disease, as we may have to deal with Ménière's complication of symptoms, with a stomach-neurosis, or a spinal disease—e. g., tabes. The gastric crises of the tabetics may resemble very closely the attacks of vomiting in the course of a brain tumor.

Among the organic diseases of the brain which may be mis-

taken for a new growth are brain abscess and meningitis. The former—the abscess—is almost always associated with febrile movements, and rarely with papillitis; moreover, there are the characteristic remissions, so that the patient's general condition may be excellent for years. If we keep these points in mind, and if we make it a rule never to diagnosticate a brain abscess unless we can obtain in the history some ætiological datum, such as an otitis media, traumatism, etc., the differential diagnosis will usually present little difficulty. In meningitis fever is the most important symptom. Papillitis is more frequent here than in abscess, and hence of less value in the differential diagnosis between tumor and meningitis, yet the early delirium and the jactitations are sufficiently characteristic symptoms to be of diagnostic value.

Other diseases to be considered are chronic cerebral sclerosis associated with arterial disease, and lobar sclerosis. The absence of grave general symptoms, the usually much slower course, the appearance of multiple sclerotic foci, the absence of papillitis, are often points enough on which to base a diagnosis.

Finally, the possibility of confusing brain tumor with progressive paralysis of the insane (dementia paralytica) and with chronic alcoholism ought to be spoken of. This can, of course, only happen in those cases of brain tumor where apoplectiform attacks occur, where headache is either absent or only slight, where, however, the mental disturbances are marked, and where, owing to the defective memory, the alterations in speech become a prominent feature of the case. The course will clear up all doubtful cases. If we are dealing with a dementia paralytica we shall not have to wait long for the appearance of the characteristic delusions of grandeur, and the patient will become bewildered and have transitory periods of excitement, whereas, with the tumor, stupor and somnolence are developed. In chronic alcoholism tremor and the occurrence of stomach and liver affections are usual. Above all, a conscientious use of the history will guard us from an error in the diagnosis.

The seat of the tumor we can only attempt to determine when we have reliable focal symptoms to aid us, but, as we have observed, such may be absent, and, as it seems, this is more especially the case in soft tumors occurring in the ventricles and sometimes in the frontal lobes, which give rise to

symptoms of general compression only. It is furthermore per-
fectly certain that a great part of the basal ganglia, the lenticu-
lar and the caudate nucleus, also the anterior portion of the
thalamus, the corpus callosum, the fornix, the choroid plexus,
and finally the cerebellum, with the exception of the vermiform
process, may be the seat of neoplasms with a complete absence
of all focal symptoms. On the other hand, tumors of the motor
area, of the occipital and temporal lobes, of the pulvinar, of the
crus, the pons, the medulla oblongata, and of the vermiform
process of the cerebellum, often manifest themselves clinically
by characteristic focal symptoms, which we here need not de-
scribe, as they have been considered above in detail. Suffice it
only to add that destruction of the pulvinar, no less than de-
struction of the occipital lobe, may give rise to hemianopia,
that an early oculo-motor paralysis points to the existence of a
tumor in the crus, while severe general symptoms—tonic con-
vulsions, without the loss of consciousness, staggering gait—
indicate a neoplasm in the vermis of the cerebellum. Tumors
of the medulla oblongata may, if general symptoms are absent,
simulate bulbar paralysis in their course. Vertigo has often
been noted in connection with such tumors. Other symptoms
are changeable and uncertain; sometimes, indeed, there are no
symptoms at all. Paralysis of the abducens points to the pos-
terior fossa as the seat of the neoplasm. The affections of
other nerves, which are important in this connection, have
been mentioned above.

Where we have amaurosis, the pupillary reaction to light
ought to be carefully examined. Its presence denotes that the
optic nerve and tract are intact and the new growth can only
be situated in the central optic fibres, while if it is absent or
much diminished we have to deal with a lesion of the optic
nerve or tract. Even with the existence of papillitis the pupil-
lary reaction may be present. Then the occurrence of the
former with the central lesion must be considered as an acci-
dental coincidence. If we think it possible that the amaurosis
is due to double hemianopia, we may examine for the so-called
hemianopic pupillary reaction (described on page 34) to throw
light upon the question.

The existence of focal symptoms, however, does not always
facilitate the diagnosis as much as we might suppose. This is
especially true if the general symptoms are very grave and
pronounced. As we have remarked, a hemiplegia must not

always be taken for a focal symptom, and we must again insist that its presence is of no value for the topical diagnosis. We need not mention that various disturbances may be produced by indirect action which baffle all attempts at a topical diagnosis (cf. the lecture of Jastrowitz, the reference to which is given at the end of the chapter).

The nature of the tumor can in some cases not be determined, while at other times it may be very apparent. The course of the disease is of less value in this question than, for example, the history of the patient's previous diseases, and the fact that certain tumors show preference for certain portions of the brain, sometimes also the age of a patient, are likely to afford us valuable hints.

Where syphilis has existed, we have to think of gummata. If the family history be one of tuberculosis or carcinoma, brain tubercles or secondary carcinoma ought to be considered. A chronic cerebral affection in a child, attended with headache and convulsions, is strongly suggestive of solitary or of multiple cerebral tubercles. Tumors of the cortex are more likely to be of a syphilitic or tuberculous nature, while those of the base are preferably sarcomata; those of the white matter, the centrum ovale, gliomata.

Prognosis.—The prognosis in brain tumor is generally unfavorable, and death within one or two years after the appearance of the first symptoms may be predicted. Spontaneous recovery is unheard of, and improvement as a consequence of treatment is very rare and has only been observed in cases of gummatous or tuberculous neoplasms. Here it occurs beyond question, consequently the prognosis is much less gloomy in these than in other tumors. In general the course is, in spite of all treatment, steadily progressive. The patient's sufferings increase in severity and the agony is only blunted by the dulling of the sensorium. Death occasionally sets in suddenly, as a rule only after a protracted state of marasmus in consequence of exhaustion.

Treatment.—The treatment is in the vast majority of cases of no avail. Only in rare instances can we by a systematic administration of potassium iodide (5.0 to 8.0 (grs. lxxv to ℥ ij) daily in hot milk for one and a half to two months) effect a noticeable improvement. Whether this is due to the direct action of the iodide on the tumor or whether only the secondary changes, the softening, the œdema, the accumulation of

fluid in the ventricles are influenced thereby, we do not know. As a matter of fact, however, the improvement does occasionally occur, and, be it explicitly stated, not only in cases of gummata, but also in other, malignant, neoplasms. Besides iodide of potassium arsenic seems at times to have a beneficial action, yet sufficient positive observations are wanting on this point.

The question of operative interference, if such appear indicated, involves the same principles which we have set forth in connection with operation for abscess, and which ought to guide us here also. Symptoms pointing unmistakably to an exudation into the ventricles justify trephining and tapping of the lateral ventricles for the purpose of lowering the intracranial pressure. The posterior fossa is always a sort of a *noli me tangere* (Wernicke). Headache, vertigo, and vomiting are to be treated symptomatically.

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APPENDIX.—PARASITES OF THE BRAIN.

Among the parasites found in the brain the cysticerci and the echinococci are the most important.

The former—the cysticerci—are found quite frequently at the autopsy when their existence during life was not diagnosticated or even suspected, a proof that they may be present without giving rise to any symptoms, or that they may produce a clinical picture such as is often due to other causes. The cysts, which are rarely single, but mostly multiple, amounting as they may to one hundred or more in number, have their seat, some in the meninges, some in the substance of the brain, in the gray as well as in the white matter; sometimes they are free in the ventricles. They may be so numerous that the whole surface of the brain is studded with them. Their size may vary from that of a bean to that of a walnut, and but rarely exceeds that of the latter. They contain a serous fluid. At a place where the cyst-wall is somewhat thickened are situated the neck and head, the latter often darkly pigmented, and to be recognized on closer examination by a crown of hooklets and suckers. The parts surrounding the cyst are either perfectly normal or in a state of inflammatory softening. This latter is found as a rule only when the cysticercus is dead and has undergone changes. If the cyst sends out diverticula it assumes the form of a bunch of grapes, and hence is called cysticercus racemosus (Virchow, Marchand). It is estimated that the parasites live from three to six years. After their death they are changed into calcareous concretions, surrounded by a connective-tissue membrane, which in their interior contain cholesterine and fat.

Echinococci are usually found in single solitary vesicles on the free surface of the brain or the ventricles. Their yellowish mucoid contents, surrounded by a cyst-wall and a connective-tissue capsule, can break through to the outside, and be evacuated through the nose, the ears, etc., and a sort of spontaneous recovery take place.

It is impossible to sketch a clinical picture produced by cysticerci in the brain, because this varies, of course, with the seat of the cysts. I had occasion in the past few years to observe four cases in my clinic, and of these only one was diagnosticated during life, and this one, not because it presented characteristic symptoms, but owing to the history of the patient, from which we learned that he was in the habit of frequently eating raw pork. In all four cases the patients suffered from epileptiform attacks with convulsions, sometimes with, sometimes without loss of consciousness. Two of them were in the intervals between the attacks temporarily completely bewildered, and were sometimes for hours not able to find their way in the ward where they were staying, did not recognize their fellow-patients—in short, presented conditions which, considering the attacks which they were subject to, were looked upon as epileptic equivalents. Motor disturbances were not observed in any of the cases; all of them, however, complained at times of headache and vertigo. In one case three cysts the size of a pea were found imbedded in the left lenticular and caudate nucleus, the internal capsule being spared, and the patient had perfect use of the right extremities. In another case there was found a focus of softening the size of a pea, in which the calcified remains of a cysticercus could be demonstrated, in the left half of the middle segment of the pons immediately below the middle line, without there having been during life any noticeable symptoms of destruction. A third case showed, besides numerous vesicles imbedded in the gray cortex, cysticerci swimming free in the fluid of the ventricles, the amount of which was considerably increased. The high grade of hydrocephalus was probably responsible for the imbecility of the patient, a condition for which during life the epileptic attacks had been held accountable; these, in their turn, were doubtlessly connected with the parasites in the cortex. Cases presenting a course which resembles that of the

Fig. 95.—Cysticercus Racemosus. (After Marchand.)

progressive paralysis of the insane I have myself not had occasion to observe. According to Wernicke such instances are not rare (*loc. cit.*, 111, 373).

The ætiology of cysticerci in the brain is that of cysticerci in any other part of the body ; they will develop in persons who often give the parasites a chance to invade their body, as is, for instance, the case with butchers, and they hence occur relatively frequently in such individuals. Therapeutics in this case is powerless ; we have no means of destroying the parasite.

Echinococci of the brain often do not present any peculiar symptoms which could be used for diagnosis. The clinical picture by which they manifest themselves is usually that of a tumor, but when they have perforated to the outside we may be able to demonstrate on the protruding tumor fluctuation and pulsation. If they perforate into the orbit they give rise to œdema of the lids and exophthalmus. Westphal has observed a case in which over ninety cysts were evacuated to the outside.

That actinomycosis may occur in the human brain is shown by the publication of Bollinger (cf. lit.), where a tumor in the third ventricle is described which contained numerous characteristic granules.

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CONGENITAL DISEASES—HYDROCEPHALUS—MENINGOCELE—POREN-CEPHALY—ABSENCE OF CERTAIN PARTS OF THE BRAIN.

Our knowledge of the collections of fluid in the brain, which are described under the general term of hydrocephalus, is, on the whole, very defective, and this is even more true of

the causes which bring about the abnormal increase. We
know that the fluid is either contained between the meninges
or within the ventricles, and speak accordingly of a hydro-
cephalus externus and internus. We know further that it may
collect very rapidly or very slowly. In the former case we
have a hydrocephalus acutus, and in the latter a hydrocephalus
chronicus. Finally, we know that the conditions under which
it develops may sometimes exist during intra-uterine life, or,
again, may appear much later, and we consequently distinguish
the congenital from the acquired form. But, after all, the dis-
tinction which we gain by this is only superficial. About the
exact manner of development of any of these forms there pre-
vails a great difference of opinion, and the question under what
circumstances hydrocephalus may develop as an independent
idiopathic disease can not be satisfactorily answered. There
is no doubt but that in by far the greater majority of cases we
have to deal with a congenital disease, and, as a matter of fact,
this form plays in practice the most important *rôle*.

The congenital hydrocephalus is very rarely external, but
is much more often internal. It may be well developed at
birth, so that the circumference of the skull measures sixty or
seventy centimetres or more. The skull bones then are usually
so thin that their thickness scarcely amounts to that of a sheet
of paper. The fontanelles and sutures are separated by wide
gaps. The distention of the ventricles may be so enormous
that they form a large cavity which is surrounded by brain
substance one and a half to two centimetres thick. The lat-
eral ventricles are usually dilated to a much greater extent
than the third and fourth; still, these latter may also be mod-
erately distended. The whole brain, more particularly the
basal structures, presents the signs of an increased intracranial
pressure; they are flattened out, the commissures are stretched,
the foramen of Monroe is very large, the walls of the ventricles
are often covered with granulations, the ependyma inflamed
and in places slightly thickened. The colorless serous fluid,
the amount of which may be as much as one and a half litres,
contains 99 per cent. of water, 0.3 per cent. albumen, traces of
salts, and so forth, and the sp. gr. is 1.004 to 1.006.

The most conspicuous symptom of hydrocephalus is the pe-
culiar enlargement of the head. This is, however, not always
apparent in the first weeks. Sometimes one and a half or two
months may pass before the increase in size begins to be notice-

able. The circumference of the head, which at birth meas-
ures forty centimetres and a year later forty-four centimetres,
rapidly becomes greater, and every week a half or one centi-
metre is added to it, so that after a certain time, often only
after a few months, the head has reached in circumference a
size which it does not generally attain to before the age of
puberty—viz., fifty centimetres. If the distention of the skull
is equal on all sides it becomes spherical and forms a strik-
ing contrast to the smallness of the face, which, of course, does
not take part in the enlargement. If, however, this is more
marked in the sagittal diameter the skull assumes a dolicho-
cephalic form, and its appearance is no less bizarre. This is
still more accentuated by the enormously enlarged veins which
as blue cords run over the skull. The eyes are frequently di-
rected downward. This may depend upon an insufficient in-
nervation of the eye muscles. The appearance of a child with
a well-developed hydrocephalus, the enormous head, which if
the child is held erect rolls from side to side, the small trunk
which with its shrunken limbs looks as if it was only an appen-
dage of the head, the idiotic facial expression, are together
characteristic enough to warrant the diagnosis without any
further examination, which would reveal various motor dis-
turbances, spasms of the muscles, and sometimes increased re-
flexes. It need hardly be stated that the intelligence develops
only in a very imperfect manner or practically not at all. Most
of the children never learn to speak or at least only imper-
fectly. They are not able to play like others, their conduct is
silly and senseless, their habits are dirty, and they require
much painstaking care and nursing. In exceptional cases their
mental development reaches a somewhat higher stage and they
are able to comprehend certain things, so that under particu-
larly favorable circumstances, as in a well-conducted home for
feeble-minded children, it may be possible to give such children
an amount of knowledge and skill which is quite remarkable.
The appearance of epileptiform attacks, which are always
to be anticipated, often greatly interferes with such at-
tempts.

The course is either chronic or acute. The issue is always
unfavorable. The children either die during or soon after
birth or they attain an age of a few months, or finally they
may live four or five years, while it is very exceptional for
them to live longer or reach the age of puberty. If, how-

ever, this happens, the head ceases to grow and remains of the same size or becomes even a little smaller and the skull ossifies. If death occurs in an earlier stage this happens either during a convulsion or comes on gradually as a consequence of general marasmus. There is no question but that in face of this affection therapeutics is powerless. We may well omit the usual inunctions of the skull with mercurial ointment or the painting with tincture of iodine, as well as the internal administration of iodide of potassium, without any feeling of self-reproach, for, often as these measures have been used, rarely has any good result from them been seen. Good general nursing of the child, later a well-conducted simple instruction as far as this is feasible, finally, symptomatic treatment, more especially of the more dominating symptoms, as the epileptiform seizures, which are best met with bromides, is more rational than any other more or less futile measures, not excluding puncture of the head and other surgical interference. That we are ignorant of the ætiology we have said above, and would only add here that the statement, that syphilis and alcoholism in the parents are predisposing causes, is without foundation.

The idiopathic hydrocephalus which appears later in life may be connected with atheromatous processes and focal diseases in the brain. Owing to the rarity of its occurrence, however, it has been but little studied, and the possibility that even in such cases we have in reality to deal with the secondary, deuteropathic, hydrocephalus is by no means excluded.

The secondary hydrocephalus has at times to be attributed to disturbances of the circulation, at times to general disorders of nutrition. Among the former may be mentioned active hyperæmias of the brain, occurring in consequence of the abuse of alcohol, and venous stasis, as it is seen in valvular diseases of the heart and emphysema. There are, besides, the circulatory disturbances caused by circumscribed meningitides, tumors, and abscesses, by which, for example, obstruction of the aqueduct of Sylvius may be brought about (Seeligmüller). Among the disturbances in nutrition there are certain forms of anæmia, general dropsy, phthisis pulmonalis (Callender). The affection may run a very acute course and prove fatal in a few days. On the other hand, it may be eminently chronic, and then the symptoms need not by any means be characteristic, and it may be the more difficult to make a diagnosis, as the increase in the size of the head is wont not to take place. Some-

times the symptoms are those of brain tumor ; again those of a spastic spinal paralysis may predominate.

The so-called hydrocephalus ex vacuo, a form which develops in old people under the influence of a general atrophy of the brain, must also be looked upon as a secondary hydrocephalus. It is associated with more or less pronounced dementia. About ætiology and treatment nothing need be added to what has been said on congenital hydrocephalus.

Under certain circumstances there are found defects in the bony skull cap which allow the contents to protrude. By this the dura and galea as well as the skin are raised hemispherically, constituting what is called a brain hernia or cephalocele, and we speak of an encephalocele if the brain substance and the pia are both contained in the dural sac, while, if only the dropsical soft meninges are to be found in it, it is called a meningocele. Whether a local decrease of resistance of the membranous skull and defects of ossification or perhaps abnormal adhesions of the meninges with the amnion is the cause of such anomalies has as yet to be decided. Clinically they possess no significance.

The above-mentioned defects (page 267), which we call porencephaly (Heschl), may also be congenital. Some gyri may be entirely or partly absent, so that clefts or funnel-shaped openings or pits are formed. The defective areas, unless there be a communication with the ventricles, are covered with pia tissue, and the empty space is filled up with fluid which collects in the subarachnoidal tissue ; or, again, the neighboring convolutions are pressed together over the gap and instead of a hollow we only find a deep cleft (cf. Ziegler, Pathol. Anat., ii, 636).

Very remarkable is the fact that certain parts of the brain may be entirely absent. This has been observed for the corpus callosum, the fornix, the corpora albicantia, the gray commissure, and others. With reference to the absence of the corpus callosum various hypotheses have been put forward. It has been thought to be connected with the development of the base of the skull and to depend upon the angle which the petrous portions of the two temporal bones form with each other (Richter, Virchow's Archiv, 106, 1886). Recently Kaufmann has described a case where the corpus callosum was completely absent and where its formation had never even begun, so that the commencement of the disturbance in development

had to be referred to a time between the third and fourth months.   In this case the high grade of internal hydrocephalus which was present had to be looked upon as the cause (Arch. f. Psych. und Nervenkrank., 1887, xix, Bd. iii, page 769). This, in all probability, is more frequently, than is generally supposed, the immediate cause of congenital malformations due to arrest of development which is principally the result of traumatism during birth, protracted labor, asphyxia in consequence of compression, etc.   Deficiencies in the region of both fissures of Rolando are especially of interest because they may simulate in their clinical manifestations spastic spinal paralysis, although the resemblance is somewhat obscured by the simultaneous presence of cerebral symptoms ; and there is, of course, every possible gradation, from the pure picture of a spastic spinal paralysis in which only the lower extremities are affected to that in which the arms are implicated and cerebral symptoms are well marked.   Schultze (Deutsche Medicinische Wochenschrift, 15, 1889) has observed the spastic rigidity in the lower extremities in more than one member of the same family.

Sometimes certain parts of the brain are only imperfectly developed.   Such a condition has been found in certain gyri, the optic thalami, the corpora quadrigemina, the corpora striata, and others.   Schröter, among other writers, has described such a defect in the corpus callosum, which in his case was abnormally short (Allgem. Zeitung f. Psych., 1888, xliv, 4, 5). The cerebellum may also remain very much behind in development, so that under certain circumstances it scarcely attains the size of a walnut.   The causes of such local malformations are usually as obscure as their clinical manifestations during life.

DISEASES OF THE SPINAL CORD.

WHAT we have said above about the diseases of the brain holds good, with certain limitations, also for those of the spinal cord. The anatomy of the cord certainly offers less difficulty than that of the brain, and, especially as regards the finer structure of the organ, has been more minutely examined into and is better understood; but in the physiology there exist still so many points, some obscure, some still under discussion, that the pathology remains here also very incomplete. To give a description of the diseases of the spinal cord, especially when questions of its physiology and pathological anatomy are to be discussed, is an extremely difficult undertaking, and were it accomplished far better than I have been able to do it, would still stand in need of a lenient judgment. We shall adopt the same arrangement as in our account of the cerebral diseases, and divide the subject into three parts. The first will contain the diseases of the membranes of the spinal cord, the second those of the spinal or peripheral nerves, the third those of the white and gray matter of the cord.

PART I.

DISEASES OF THE SPINAL MENINGES.

THE spinal meninges are, on the whole, not frequently dis-
eased alone; more often the inflammation spreads from the
(soft) membranes of the brain to the pia of the cord, or from
the surrounding structures to the dura spinalis. The one of
greatest practical interest among the affections of the meninges
of the cord is the pachymeningitis cervicalis hypertrophica,
which we shall shortly describe.

Of the anatomy but little needs to be added to what has been
said on page 3. The spinal portion of the dura is thinner than
the cerebral; it widens into a large cylindrical sac, which is by no
means filled up by the spinal cord. This dural sac extends beyond
the lower end of the spinal cord (conus medullaris), and terminates
in a cone-shaped point at the level of the second sacral vertebra;
all these are points too well known to be dwelt upon here at length.
The conus medullaris ends in the filum terminale, a filiform process
which is accompanied by the longitudinal nerve bundles coming
from the lumbar and sacral portion of the cord, which constitute the
cauda equina. The so-called ligamentum denticulatum is a flat band
which by its inner edge is connected with the pia and externally by
a toothed edge to the dura mater; the arachnoid lies in such close
contact with the dura that the subdural space is only a capillary
space, whereas the subarachnoid space, situated between the arach-
noid and the pia, is of considerable width. The denticulate ligament
divides it, though incompletely, into an anterior and a posterior
half. In contradistinction to the pia mater of the brain, that of the
spinal cord presents two different layers of connective tissue, the
outer one of which, very well developed in man, passes into the sub-
arachnoideal trabeculæ, while the inner is made up of a single layer
of circular bundles of fibrillæ (Schwalbe) (*vide* Fig. 96).

CHAPTER I.

INFLAMMATIONS OF THE DURA MATER.

PACHYMENINGITIS SPINALIS.

WHILE in the cerebral portion of the dura the inner surface is the usual seat of the inflammation, we find that the spinal dura mater may be diseased on its outer as well as on its inner surface; yet the clinical recognition and separation of these two forms is very often impossible.

The inflammation of the outer surface of the dura, the pachymeningitis spinalis externa, or the inflammation of the
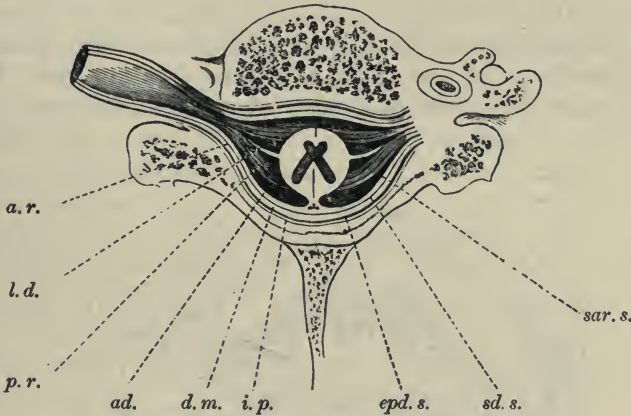
Fig. 96.—CROSS-SECTION THROUGH THE VERTEBRAL COLUMN AND THE SPINAL CORD (DIAGRAMMATICAL). *epd. s.*, epidural space. *sd. s.*, subdural space. *sar. s.*, subarachnoid space. *i. p.*, inner periosteum of vertebra. *d. m.*, dura mater. *ad.*, arachnoid. *p. r.*, posterior spinal root. *l. d.*, denticulate ligament. *a. r.*, anterior spinal root. (After EICHHORST.)

connective tissue between the dura and the vertebral column, peripachymeningitis, is a very rare disease, and probably only occurs secondarily. The inflammatory changes, which at times are most marked on the posterior surface, consist of a thickening and cellular infiltration of the dura; sometimes, also, the membrane may be found covered with dense cicatricial deposits (Eichhorst). The chief causes are caries or tuberculosis of the vertebræ, pleuritis, psoas abscess, syphilis, puerperal pyæmia, suppuration in the peritoneal cavity, and in exceptional cases the disease may have its origin in a neuritis

migrans. The clinical picture depends largely upon the impli-
cation of the nerve roots and of the spinal cord. If the cord is
compressed by the thickening, the symptoms of a pressure pa-
ralysis, to which we shall have occasion to refer later, make
their appearance. If the nerve roots are implicated, there are
violent paroxysmal pains which run along the vertebral column
and radiate into the extremities. Rigidity of the neck and
tenderness on pressure over the spinous processes of the verte-
bræ are rarely absent, but are not sufficient to warrant a diag-
nosis, as they may be found just as well in an inflammation of
the pia. To make a definite diagnosis will in all cases only be
possible, if accompanying signs are taken into consideration,
more especially those of any primary disease. It is always a
difficult, sometimes even an impossible task.

The inflammation of the internal surface of the dura mater
usually develops in the cervical portion of the cord; follow-
ing Charcot, who first described the anatomical and clinical
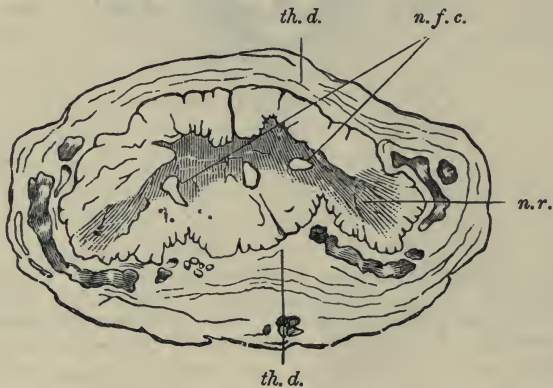
Fig. 97.—Cross-section through the Middle of the Cervical Enlargement in
Pachymeningitis Cervicalis Hypertrophica. *th. d.*, thickened dura. *n. f. c.*,
newly formed cavities. *n. r.*, nerve-roots. (After Charcot.)

features of the disease, it has been called pachymeningitis cer-
vicalis hypertrophica; the inflammatory new formation and
thickening of the connective tissue, which are most marked on
the posterior inner surface of the dura, exist in circumscribed
areas (Fig. 97); this compresses the nerve roots, which pass
through the membrane at these places, and finally even the
cord, and may give rise to the formation of channel-like cavi-

ties (*n.f.c.* in Fig. 97). If the compression continues for a considerable time it leads to secondary degeneration of the pyramidal tracts in the spinal cord, as well as of the motor nerves originating in the parts diseased, and to atrophy of the muscles supplied by them.

Symptoms.—The symptoms of the disease are mostly the outcome of the participation of the nerve roots and the spinal cord. The disease may well be divided into two stages, each having its characteristic symptoms. To the first belong the pains, to the second the paralyses (Charcot). The pains vary extremely in intensity and extent; as a rule they are confined to the region of the neck, whence, occurring in paroxysms of increasing severity, they radiate into the upper extremities and are accompanied by paræsthesias in the arms, tingling and formication in the finger tips. The grip is usually markedly diminished, and a test with the dynamometer shows that the patient is able to lift only ten to fifteen kilogrammes. Not rarely trophic disturbances, in the form of vesicular eruptions, roughness and desquamation of the epidermis, are noted. The sensation of stiffness in the neck and of difficulty in moving the head troubles the patient a great deal, and gives to him a stiff, quite characteristic appearance. He carefully avoids turning his head in any direction, and tries to make up for this rigidity of his neck by turning the whole body, which he does slowly and in a somewhat awkward way. The most careful examination of the cervical region, percussion of the spinous processes, hot sponges applied to the skin over them, and the like, does not always reveal an increased sensitiveness.

Gradually, that is to say, in the course of two or three months or more, the patient gets accustomed to his pains, so much the more so as they become less severe in the further course of the disease. On the other hand, he discovers to his great distress that the motor power of his upper extremities is becoming more and more impaired. The stage of paralysis, as a rule, is immediately preceded by a peculiar heaviness and stiffness in the shoulder and elbow joints. The patients notice that they are unable to raise their arms as high as before; if they are females, that they can not arrange their hair themselves any more, owing to the impairment in the upward and backward motion of the arms, movements which finally become totally impossible.

The elbow joint, too, becomes stiffened, and the motions

of the wrist and finger joints become visibly impaired. The disability is not, however, usually equal in both arms and hands, as one hand may be almost useless, while the function of the other is not much interfered with. Still, in some instances, the trouble may progress in both arms *pari passu.* Curiously enough, not all the muscles of the forearms become affected, but more especially those supplied by the ulnar and median nerves, while the extensors, which are supplied by the musculo-spiral, remain more or less intact. The affection of the muscles manifests itself by an increasing atrophy and weakness, which allows an overaction of the healthy antagonists—the extensors—so that the hand, although by no means in all, but only in the well-marked cases, assumes a very characteristic position. It is dorsally flexed, and the fingers, which are bent in the second and third joints, give to it the appearance of a claw (Fig. 98). About the development of this position
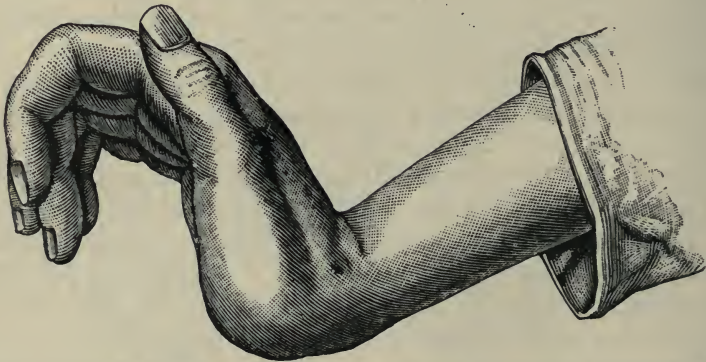
Fig. 98.—POSITION OF THE HAND IN PACHYMENINGITIS CERVICALIS HYPERTROPHICA.
(CHARCOT.)

we shall have more to say when speaking of the ulnar paralysis. The difficulties arising from this diminished motor power are considerably aggravated by the paræsthesias in the finger tips. The patients are unable to take hold of small objects—pins, steel pens, etc.—they are unable to attend to their own toilet because they can not feel small buttons, and so forth. They become more and more helpless, and, what is of the greatest significance for patients belonging to the working classes, they become incapacitated for work and unable to earn their living. This may indeed be the case at quite an early period, when the patient is otherwise in a comparatively fair

condition, especially in female patients who do fine hand-work (sewing, knitting, embroidering). The whole condition becomes worse and worse. Arms and hands become stiffer and stiffer, until finally, although not in all instances, a complete paraplegia of the upper extremities develops. Whether or not to these symptoms a paresis or paralysis of the lower extremities or bladder disturbances are added will depend entirely upon the extent to which the spinal cord takes part in the process. It can in no case either be predicted or excluded with certainty.

Course.—The course of the disease is always chronic and extends over years. After the period of pains has passed the patients are, as a rule, free from them forever, and only suffer from the helplessness which results from the motor disturbances. Owing to this they require scrupulous care, have to be dressed, undressed, fed, etc., by an attendant. Recovery or even an improvement is an extremely rare outcome. To be sure, I have seen a cured patient in the clinic of Charcot, but from the minuteness with which this case was described, from the feeling of justifiable pride which accompanied the demonstration, one could well see how extremely rare a cure must be. Remak, too, speaks of the curability of the disease (Deutsch. med. Wochenschr., 1887, No. 26). I myself am unable to present such a case. The patients in my wards, after unsuccessful trials of all proposed modes of treatment, have long given up all hopes of any marked improvement.

Diagnosis.—The disease may, especially in its onset, possibly be confounded with either spinal leptomeningitis or, as we shall later show, with syringomyelia. It is natural that tumors of the vertebral column, if they be situated in the region of the cervical enlargement, should produce in the initial stage the same symptoms as a pachymeningitis. The further course, however, will soon settle the diagnosis. Besides these there are two more diseases which may in the mind of the beginner give rise to some difficulties with regard to the differential diagnosis—namely, progressive muscular atrophy and amyotrophic lateral sclerosis. It is true that a patient with a pachymeningitis may sometimes present the appearance of a man suffering from progressive muscular atrophy; but the two diseases should never be confounded, inasmuch as in the latter affection the initial stage is not accompanied by pain, and the stiff neck has never been known to occur in it. The idea of

amyotrophic lateral sclerosis will probably also be discarded, as in this disease the lower extremities are implicated, and as difficulty in swallowing, a sign which indicates extension of the process to the medulla oblongata, will usually not be very late in appearing. We may say that the diagnosis of cervical pachymeningitis can, if the case is carefully examined and if the course of the affection is taken into consideration, almost always be correctly and definitely made out.

Ætiology.—We are wholly ignorant of the ætiology of the disease. Some maintain that the abuse of alcohol is of some importance in this connection, others the living in damp houses. Whether syphilis has any such influence, and, if so, what is its mode of action, is not as yet established. There is no doubt that the affection is more common among the working classes and the lower grades of society, but what are the conditions and influences which act as direct causes, if such there be, we are not able to say.

Treatment.—The treatment comprises local as well as general therapeutic measures. The former consists of the application of strong counter-irritants—e. g., the painting twice daily with tincture of iodine, of the use of irritative ointments or moxæ. The application of Paquelin's cautery, with which punctiform scars on the skin are produced (the so-called *points de feu* of the French), only deserves preference because it is less painful than the others. Any lasting result can not be expected from it. Not more is effected by general or internal treatment, and it is impossible to give the indications for any particular remedy. Iodide of potassium has been used in order that something might be done, without, however, producing anything else than disorders of digestion. If the patient insists on taking medicine, a placebo ought to be given—acids, bitters, etc. In no case were we able to see any beneficial effects from warm baths and hydrotherapy in general. The only measure which at least modified the symptoms somewhat, in that it gave the patients for a time more freedom of motion in their paretic extremities, was electricity, more especially the cutaneous faradization with the brush on the neck as well as up and down the limbs. If the constant current is used it ought especially to be applied to the muscles innervated by the ulnar and median nerves. By repeated closing and opening of the current muscular contractions should be elicited.

LEPTOMENINGITIS SPINALIS.

THE soft membranes are rarely ever by themselves the seat of inflammation, whether of an acute or a chronic type. Such, however, may exceptionally occur as the result of traumatism, of overexertion, carrying heavy loads, or as a consequence of exposure to cold after sleeping on the damp ground in camping out, etc. (Brown, cf. lit.). But in the greater number of cases we have to do with the extension of an inflammatory process of an infectious nature, as in epidemic cerebro-spinal meningitis, or in tubercular meningitis, both diseases which affect the membranes of the brain as well as those of the spinal cord. That there are instances of meningitis secondary to other diseases, and under what circumstances they occur, we have already stated on page 12. Here we only wish to draw attention to its connection with acute articular rheumatism, of which Krabbel (Inaugural Dissertation, Bonn, 1887) has reported an instance.

Pathological Anatomy.—Pathologically the acute spinal meningitis is divided into three stages. The first is characterized by a diffuse reddening and swelling of the meninges, more especially of the pia; the second by the appearance of a purulent or fibrino-purulent exudation upon this membrane. This may occur gradually, and may vary considerably in extent; it may be found over the whole length of the pia (always more on the posterior surface), or may be confined to circumscribed areas. In the third stage the pus becomes reabsorbed and thickening of the pia with the formation of adhesions between it and the dura takes place.

That the nerve roots also participate in the inflammation is evident from the hyperæmia of their blood-vessels, the infiltration of the interstitial connective tissue, and the eventual de-

generation of the medullated nerve fibres. If we remember the many processes by which the pia is united with the spinal cord itself, it is not surprising that the latter is implicated. On cross-section it looks in places injected, œdematous, and is seen to bulge ; on the other hand, there are undoubtedly instances in which the cord does not take part in the inflammation.

The chronic form, which seems very rarely to occur primarily, and then only after the protracted abuse of alcohol, is usually preceded by the acute disease or is secondary to different spinal lesions or various affections of the vertebræ. The pathological changes occurring here can well be compared with those of the acute form. In this condition we find thickening and opacity of the tissue, masses of newly formed connective tissue, and adhesions to the dura. There is turbidity of the spinal fluid, which is abnormally increased, and sometimes abnormal formation of pigment. The brownish-red and black specks often seen are to be looked upon as the remains of previous hæmorrhages, in which the coloring matter of the blood has undergone changes (Eichhorst). In consequence of the extraordinary development of the processes of the pia, this membrane adheres very firmly to the cord, so that it can not be stripped off without loss of substance of the cord. Here, again, the nerve roots are implicated, as is evident from their changed appearance. They look flattened and atrophic.

Symptoms.—In the acute form pain undoubtedly plays the principal part. Even in the initial stage, which does not differ from that of other acute diseases in most of the symptoms (chill, general malaise, loss of appetite, disturbed sleep, elevation of temperature), the pain along the spinal column is very marked. The patients are constantly troubled with it in whatever position they may lie, although it is especially sharp on any attempt to move or to sit up in bed. At the same time they feel an unwonted stiffness in the muscles of the back, and have difficulty on motion. On careful examination of the back we find that, although the spinous processes of the vertebræ are tender on pressure, and by the slightest tap or by the touch of a hot sponge pain is evoked, this is in no way comparable to that felt by the patient without any extraneous interference. This persists obstinately, and usually in the further course of the disease may radiate into the arms and legs, owing, of course, to the implication of the nerve roots. The

same factor also accounts for the different hyperæsthesias of the skin, the girdle sensation, the muscular pains, etc. Rigidity of the neck is only observed if the process has attacked the cervical portion. If the spinal cord itself becomes implicated, spinal symptoms, bladder disturbances, increased reflexes, and extensive sensory disturbances make their appearance. All these symptoms may persist unchanged for weeks, the patient feeling very bad, and complaining of constant violent pain. If the disease take a favorable turn the pains gradually abate and the patient gets relief; but, on the other hand, the symptoms of irritation may give place to those of paralysis, and as anatomical changes go on in the nerve roots (degeneration, atrophy), we have analgesias and anæsthesias, the muscles become more and more incapable of performing their functions, they undergo marked atrophy, and on electrical examination distinct reaction of degeneration is found. There is direct danger to life (1) if the process extends upward to the medulla oblongata; in that case death may occur in a few days. (2) If, owing to an extensive myelitis, bed-sores develop which lead to the utter exhaustion of the patient. Recovery may be complete or incomplete; in the latter case pareses, paræsthesias, and bladder disturbances are left behind as the result of irreparable anatomical changes.

The symptoms of the chronic do not differ much from those of the acute form. The pains only are occasionally less pronounced. They vary with regard to their violence and seat; sometimes they are most marked high up between the shoulder blades, sometimes lower down in the back, so as to interfere more or less completely with stooping; not rarely they are found to radiate toward the front of the thorax, sometimes on one, sometimes on both sides. Even slighter degrees of pain are sufficient to seriously interfere with the occupation of the patient, especially, of course, if the arms or legs, or what is, however, rather rare, all four extremities are implicated. Sensory changes are found in both the acute and the chronic form; an implication of the cord itself leads to the same symptoms of irritation or paralysis which we have before mentioned. The disease may drag out its course through a number of years and still there may follow a relative recovery; complete recovery I have never seen.

Diagnosis.—To make a correct diagnosis of this disease much experience and carefulness is necessary. Acute spinal

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meningitis may be mistaken for muscular rheumatism and lumbago, the chronic form for spinal irritation and cord diseases. A differentiation from the former may be facilitated by an examination of the spinous processes for tenderness on tapping or touching with a hot sponge. In simple muscular rheumatism the spinous processes are not sensitive, whereas the different muscles are found to be tender if pressed or kneaded. Lumbago pains are recognized by their greater severity, their frequent change in locality, and their lesser persistency. Spinal irritation should only be diagnosticated in very anæmic hysterical individuals; and the further course and final outcome of the disease will guard us against the assumption of the existence of a cord disease, for, if this be present, the issue is always unfavorable.

Treatment.—With reference to treatment little is to be added to what has been said on page 318. Here, too, local measures—counter-irritation, etc.—must first be tried, and in case they should be found of no avail, prolonged tepid baths (93° Fahr., for from half an hour to an hour and a half) should be substituted. Electricity should also be used in the form of the faradic brush applied over the painful muscles. Gentle massage, if practiced by a competent person, is strongly to be recommended, and ought to be continued for a long time. The administration of iodide of potassium, for which no indication whatever exists, is to be condemned.

CHAPTER III.

THE vessels nourishing the spinal meninges are the anterior and posterior spinal arteries, arising from the vertebral artery, which in its turn comes off from the subclavian. They join with a succession of small branches which enter the spinal canal through the intervertebral foramina and form median vessels, which run in front and behind the cord along the longitudinal fissure, having numerous horizontal anastomoses. Both of these arteries send constantly fine horizontal twigs into the substance of the cord, while others are distributed to the pia. The capillary network is decidedly denser in the gray than in the white matter.

The occurrence of a hæmorrhage between the membranes of the spinal cord ("intrameningeal"), or between the dura and the bony vertebral canal ("extrameningeal"), is, on the whole, very rare. If one of these two forms occurs more frequently than the other, it is the latter, the extrameningeal, the so-called apoplexia epiduralis, so named because the blood escapes into the epidural space (cf. Fig. 96). The hæmorrhages between the dura and the arachnoid—apoplexia subduralis— and those between the arachnoid and the pia—apoplexia subarachnoidalis—which break into the space filled with the cerebro-spinal fluid, are much more uncommon. If we find on the inside of the dura encapsulated foci of variable size which contain products of decomposition, hæmatoidin crystals, detritus, etc., then we speak of a pachymeningitis interna hæmorrhagica. The loose blood coagula may be found of such a size that they compress the cord and the nerve roots. On the other hand, there may be nothing more than punctiform extravasations of blood, in the neighborhood of which the vessels of the dura appear more than usually full. That these coagula are to a certain extent capable of being absorbed, and that

they do not necessarily irreparably damage the cord and the nerve roots, is proved by the cases which take a favorable course.

Ætiology.—With reference to the ætiology, it may be said that such hæmorrhages may be evoked by overexertion. They occur by preference in men, and more especially in laborers who do hard work, such as carrying heavy loads, and who drink a great deal of alcohol. Further, after traumatic influences, either direct injury to the bodies of the vertebræ or after severe concussions affecting the whole body, such as one might receive, for instance, in a collision between two railroad trains, in which case symptoms arise which simulate very much the clinical appearances of railway spine, which we shall describe later. Secondary meningeal apoplexies occur in the course of infectious diseases—scarlet fever, small-pox, typhoid fever, etc. Also in epileptics they are not rare, and, according to Hasse, are often associated with heart hypertrophy.

Symptoms.—The symptoms very closely resemble those of spinal meningitis, only that the onset is always very sudden—"apoplectiform." A person in perfect health may feel suddenly a violent circumscribed pain in the back which differs in degree and extent in different cases, and which if the hæmorrhage is extensive may in a few hours give place to complete paralysis of the legs (more rarely of the arms). In milder cases, while the pains gradually abate, sensory disturbances, paræsthesias and anæsthesias, gradually develop, also slight motor disorders, weakness in the muscles of the extremities, sometimes also signs of motor irritation—trembling, twitching, etc. The main characteristic of a meningeal hæmorrhage which is purely spinal is the complete freedom from disturbances of consciousness. The course and the duration of the disease depend upon the extent of the hæmorrhage and its capability of being absorbed. It is necessary to have seen, carefully studied, and analyzed several cases of this nature in order to properly understand and correctly recognize a new instance. The implication of the spinal cord itself necessarily gives rise to what are known as "spinal symptoms" (increased reflexes, bladder disturbances, persistent paralyses), as we have repeatedly stated.

Diagnosis.—The diagnosis is easy in the cases with characteristic onset if we are satisfied with the diagnosis of "meningeal apoplexy," whereas it is very difficult, nay, often impossi-

ble, to determine the exact kind, whether it is epidural or sub-
dural. Again, to determine its situation is comparatively easy
if we remember that in affections of the lumbar cord the legs,
bladder, and the rectum mainly suffer, whereas affections of
the dorsal region give rise to symptoms of irritation in the dis-
tribution of the intercostal nerves, and those of the cervical
portion to motor and sensory disturbances in the upper ex-
tremities. If the seat be still higher up—in the medulla ob-
longata—bulbar symptoms, disorders of respiration and deglu-
tition, will not be absent, and the case will be fatal in a short
time.

Prognosis.—The prognosis depends upon the extent of the
hæmorrhage. Cases with a favorable outcome have repeatedly
been observed. Implication of the cord and the nerve roots
makes the prognosis more unfavorable.

Treatment.—In the treatment we ought in a fresh case, in
the first place, to procure absolute rest in bed and apply ice
over the supposed seat of the trouble, to arrest the hæmorrhage
if possible or to prevent the return of it. If the irritation seems
to be localized, local bleeding may be indicated. The further
treatment is the same as in acute meningitis.

Tumors of the spinal meninges are of no practical impor-
tance, because they can never be diagnosticated with certainty.
Although we know well from the reports of autopsies that just
as in the cerebral we may in the spinal meninges find psammo-
mata, sarcomata, myxomata, gummata, carcinomata, etc., and
that their seat may be epidural, subdural, and subarachnoidal,
we are never able to recognize definitely from the symptoms
observed during life either the nature or the seat of a tumor in
the meninges of the cord. The reason is very simple. The
tumors, as long as they are very small, produce no symptoms,
and if they grow give rise to symptoms which depend upon
the compression of the cord and the nerve roots and can not
be distinguished from those produced by pachymeningitis and
leptomeningitis spinalis. They consist therefore of signs of
motor and sensory irritation and later of paralysis, which vary
according to the seat of the tumor. If, for instance, only one
half of the cord is compressed, this may give rise to a clinical
picture which resembles that of a unilateral lesion of the spinal
cord—viz., paralysis and hyperæsthesia on the side of the com-
pression, anæsthesia on the intact side. A case of this kind

has been reported by Charcot (cf. lit.). Innumerable variations are possible, according to the size and seat of the tumor, and the less we are able to fully diagnosticate the cases during life the more important and instructive it will be to examine and describe as carefully as possible what is found at the autopsy.

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PART II.

DISEASES OF THE SPINAL NERVES.

THE nerves of the spinal cord, which are called spinal or periph-
eral nerves, arise, as is well known, by an anterior smaller, and a
posterior larger root. These are flat bundles of fibres, loosely sur-
rounded by the arachnoid, which pass into the intervertebral foram-
ina, where the posterior roots form a swelling, the ganglion inter-
vertebrale, and emerge from the spinal canal, the two roots having
united to form a common round trunk. This again divides after its
exit from the canal into two branches, an anterior and a posterior.
The anterior, usually the larger, forms numerous anastomoses with
the branches above and below it, the so-called ansæ, which are col-
lectively called plexuses. The posterior, smaller nerves, pass back-
ward between the transverse processes of the vertebræ, and are
distributed to the muscles and the skin of the back.

Of the thirty-one pairs of spinal nerves, there are eight cervical,
twelve dorsal, five lumbar, five sacral, and one coccygeal. The pos-
terior as well as the anterior branches contain fibres from both roots.
The anterior roots are motor (Charles Bell, 1811). They supply,
besides all the muscles of the trunk and extremities, the unstriped
muscles of the internal organs and the unstriped muscles of the
vessels. The posterior roots are sensory, but we should keep in
mind that the anterior most probably contain, besides the motor,
also trophic and secretory, and the posterior roots, besides the sen-
sory, also fibres for the reflexes.

The peripheral nerves, just as the cranial, may be affected
independently or secondarily, and as the result of some primary
disease in other parts. In cases of the first class overstrain
plays an important *rôle*, often also, as we have seen to be the
case in diseases of the cranial nerves, exposure to cold and
traumatism, while in those of the second class a great many
factors come in, more especially infections, intoxications, and
general cachexiæ; of these we shall speak when we treat of
the individual nerves.

If we inquire into the anatomical character of the disease we shall in many instances have to admit that we are unable to find any anatomical changes whatever in the affected nerves. This is true in many cases of mild neuralgias, but also in some of the severe, even of the severest, types. The examination of pieces of the trigeminus, for instance, which were cut out where a resection had been made on account of intolerable pain has by no means always revealed appreciable changes in the nerve; on the contrary, this has on microscopical, as well as on macroscopical, examination repeatedly been found to be absolutely normal. In other instances, however, an inflammation—i. e., a neuritis—could be demonstrated as having been the cause of the trouble. In such cases there is seen in the acute stage an exudation in the interstitial tissue and an abundant infiltration of the same with round cells, a condition which gives rise to a swollen and œdematous appearance of the nerve ("purulent neuritis"). If this inflammation continues for some time the process goes on to degeneration, under the influence of which a part of the myeline sheath is destroyed and compound granular corpuscles are formed. The axis cylinders generally remain for some time intact. In some bundles there may be found nerve fibres completely atrophied, while the sheath is somewhat thickened and irregularly contracted, presenting a wavy outline. This increase and condensation of the connective tissue makes the nerve look more and more like a cord of connective tissue, which is thinner or thicker than normal according to the amount of the newly formed tissue; sometimes, also, it is in places irregularly thickened (neuritis nodosa). The pigment deposits found have to be looked upon as the remains of previous hæmorrhages. Even after extensive destruction of the nerve fibres by the connective tissue, regeneration is to a certain degree possible, as the peripheral nerves possess this power to a considerable extent, a point which is of importance for the prognosis. According as the advance of the process is centrifugal or centripetal we speak of a descending or an ascending neuritis. A neuritis migrans has also been described. If the process occur simultaneously at different places, we speak of a multiple or a disseminated neuritis (Leyden, Roth). From the researches of Scheube we should be led to regard the so-called beri-beri, or kak-ke, a disease which occurs epidemically in Japan, as a multiple neuritis. In very chronic cases the inflammatory

changes in the connective tissue are so slight in comparison to the degenerative process in the nerve fibres that it is preferable to speak in those cases (as Strümpell has proposed) of a " primary chronic degenerative atrophy of the nerves," instead of a neuritis.

The symptoms of neuritis, of course, vary according to the position and the function of the affected nerve, as we shall show in the following pages. The symptomatology of the primary multiple neuritis *par excellence* we shall describe later.

The peripheral nerves may also be the seat of neoplasms, which in them usually develop, starting from the connective tissue. Only rarely do they consist of newly formed nerve fibres, and deserve properly to be called neuromata ; much more frequently they are fibromata, which may be found as solitary or as multiple new growths, and which not uncommonly may give rise to thickenings and nodular swellings, which can be easily demonstrated and felt on the nerves. Extensive tumors, where numerous nerve trunks are united by connective tissue into a compact mass, the so-called plexiform neuro-fibromata, are rare. Malignant neoplasms, carcinomata, and sarcomata of the peripheral nerves are sometimes met with. That here, also, the symptoms depend on the seat of the new growth is self-evident (cf. Krause on Malignant Neuromata and the Occurrence of Nerve Fibres in them. Volkmann'sche Sammlung klin. Vorträge, 293, 294, 1887. Deutsche med. Zeitung, 1888, No. 15).

We shall first speak of the affections of the motor and the sensory nerves which innervate the muscles of the extremities and the trunk, and certain internal organs which are not connected with the cranial nerves, and after that we shall turn our attention to the trophic, the vaso-motor, and the secretory fibres as far as our scanty knowledge on these points will allow. An appendix will be devoted to the primary affections of the muscles supplied by the spinal nerves.

A. DISEASES OF THE MOTOR AND SENSORY NERVES.

I. Diseases of the Cervical Nerves.

Of the four upper (smaller) cervical nerves, the first, which is called the suboccipital, emerges between the occipital bone and the atlas. The anterior branches of these four form the plexus known as the plexus cervicalis, which is situated opposite the correspond-

ing vertebræ. From this plexus come, besides the muscular branches to the scalenus, the longus colli, etc., the phrenic, which chiefly is formed by the fourth cervical nerve, and which for the most part
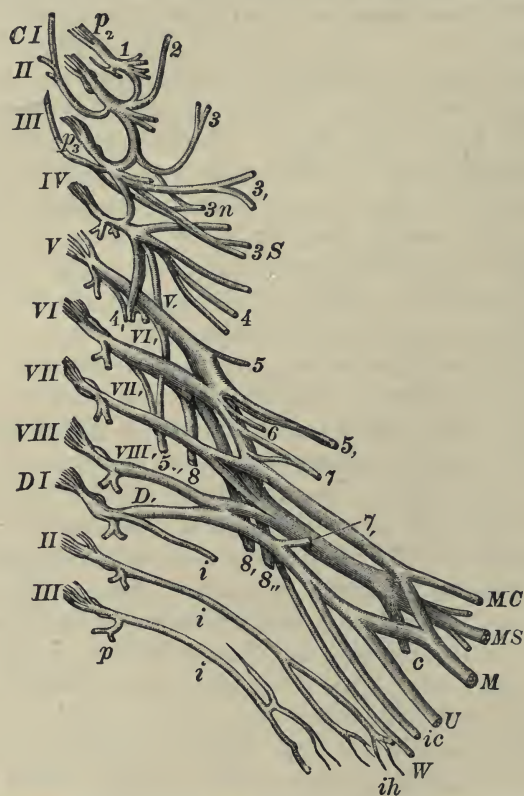
Fig. 99.—DIAGRAMMATIC OUTLINE OF THE CERVICAL AND BRACHIAL PLEXUSES. (After SCHWALBE.) *CI-VIII*, roots of the cervical nerves. *DI-III*, roots of the first three dorsal nerves. *pp*, posterior branches—p_2, of the second, p_3, of the third cervical nerve. 1, anterior branch of the first cervical nerve and loop of union with the second. 2, small occipital nerve. 3, great auricular nerve. 3_1, superficial cervical nerve. $3n$, communicating branches to the descendens noni from the second and third. $3S$, communicating to the accessorius from the third and fourth nerves. 4, supraclavicular nerves. 4_1, phrenic nerve. Brachial plexus: V_1–$VIII_1$, and D_1, the five roots of the brachial plexus. 5, rhomboid nerve. 5_1, suprascapular. 5_{11}, posterior thoracic. 6, nerve to the subclavius muscle. 7, 7_1, inner and outer anterior thoracic nerves. 8, 8_1, 8_{11}, subscapular nerves. *MC*, musculo-cutaneous. *M*, median. *U*, ulnar. *MS*, musculo-spiral. *ic*, internal cutaneous. *W*, nerve of Wrisberg. *c*, circumflex. *i, i*, intercostal nerves. *ih*, intercosto-humeral nerve.

is a motor nerve, the superficial cervical, the auricularis magnus, the occipitalis minor, and several communicating branches to the upper cervical ganglion and the gangliform plexus of the vagus (Figs. 99 and 100).

The anterior branches of the four lower (the stouter) cervical nerves, after they have passed between the anterior and middle
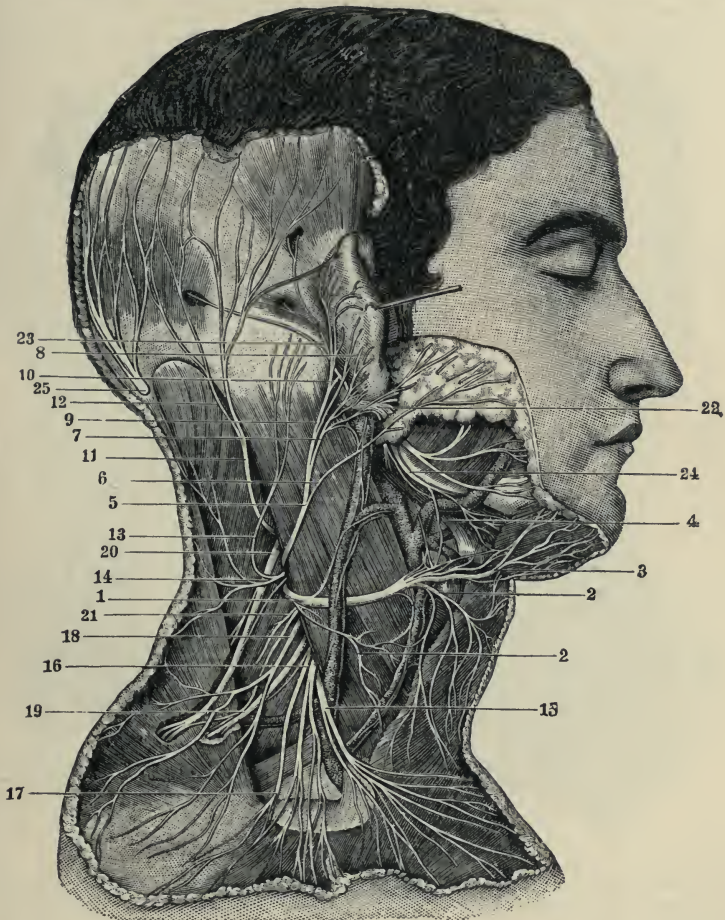
Fig. 100.—THE SUPERFICIAL BRANCHES OF THE CERVICAL PLEXUS. (After HIRSCH-FELD and LEVEILLÉ.) 1, superficial cervical nerve; 2, its inferior branch; 3, its superior branch; 4, its union with the facial. 5, great auricular nerve; 6, one of its facial branches; 7, its branch to the lobule; 8, twig which pierces the auricle to pass to its outer surface; 9, branch to the deep surface of the pinna; 10, its union with the posterior auricular of the facial nerve. 11, small occipital nerve; 12, its branch which unites with the great occipital nerve; 13, a mastoid branch or second small occipital; 14, twigs from this to back of the neck. 15, inner, 16, 17, middle, 18, outer branches of the supraclavicular nerves. 19, branch of cervical nerves passing into trapezius muscle. 20, accessorius spinalis. 21, branch to the levator scapulæ. 22, trunk of the facial nerve; 23, its posterior auricular branch; 24, its inframaxillary branch. 25, great occipital nerve.

scalenus and have reached the supraclavicular fossa, form, in conjunction with the anterior branch of the first dorsal nerve, the so-

called brachial or subclavian plexus (Fig. 101), which may be divided
into a smaller or supraclavicular portion, situated above, and a larger
infraclavicular portion situated below the clavicle. From the former
are given off, besides the suprascapular, only the three subscapular
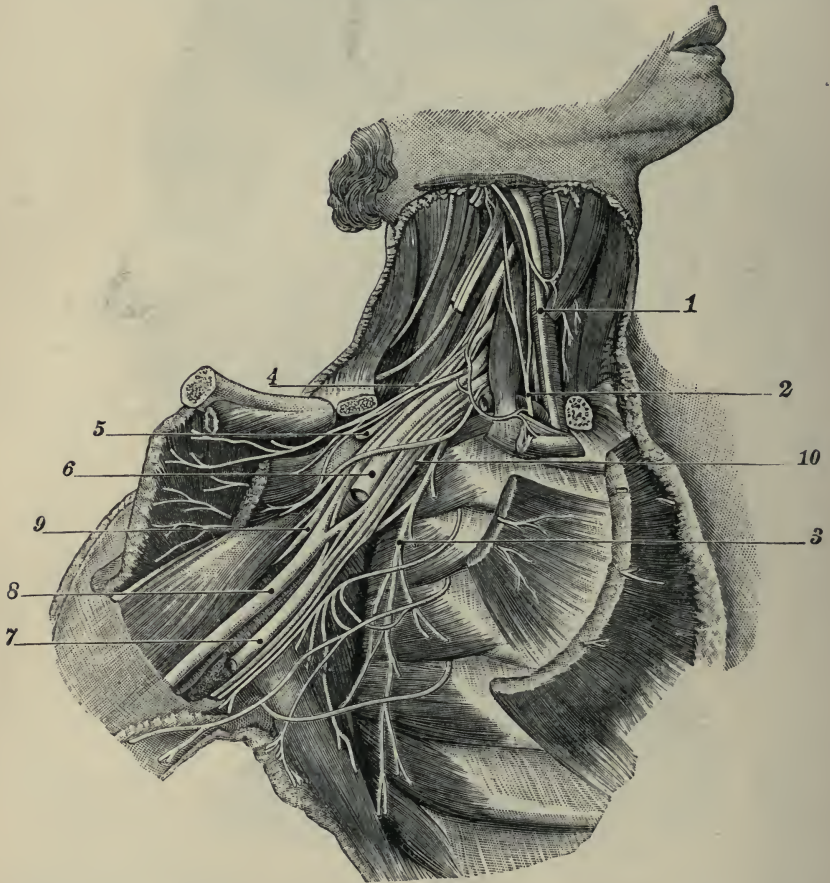
Fig. 101.—Deep Dissection of the Axilla, showing the Brachial Plexus. (After
Hirschfeld and Leveillé.) 1, vagus. 2, phrenic, passing down to the inner side of
the scalenus anticus muscle. 3, posterior thoracic nerve distributed to the serratus mag-
nus. 4, external anterior thoracic nerve passing into the pectoralis major. 5, supra-
scapular nerve. 6, musculo-spiral nerve. 7, ulnar nerve. 8, median nerve. 9, muscu-
lo-cutaneous nerve. 10, small internal cutaneous nerve (nerve of Wrisberg).

nerves, the anterior and posterior thoracics, and the rhomboid nerve.
The larger portion, which has also been called the axillary plexus,
furnishes the large nerves which supply the entire upper extremity,
the circumflex (axillaris), the median, the ulnar, the musculo-spiral
(radial) and cutaneous branches, namely, the nerve of Wrisberg, the

internal, and the (longest) external or musculo-cutaneous nerve, which has also been called perforans Casseri (cf. Figs. 102 and 103).
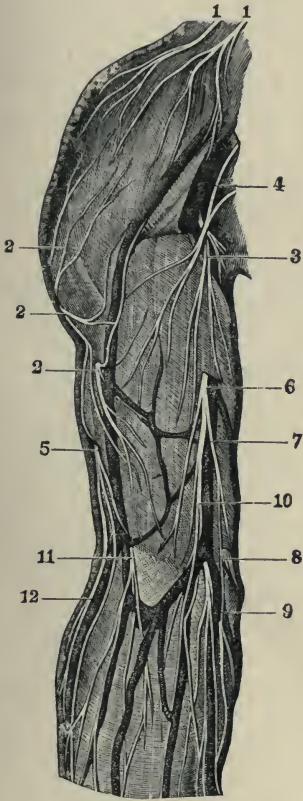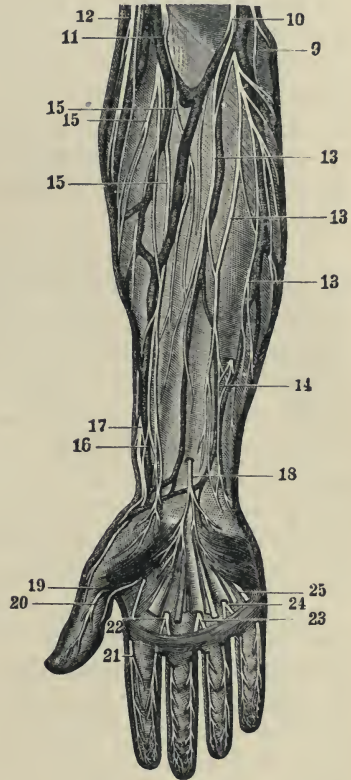
Fig. 102.   Fig. 103.

Fig. 102.—CUTANEOUS NERVES OF THE SHOULDER AND OF THE UPPER ARM. (After HIRSCHFELD and LEVEILLÉ.) 1, 1, supraclavicular nerves from cervical plexus. 2, 2, 2, cutaneous branches of the circumflex nerve. 3, 4, upper branches of internal cutaneous nerve. 5, upper external cutaneous branch of musculo-spiral. 6, internal cutaneous nerve. 7–10, branches of the internal cutaneous nerve. 11, musculo-cutaneous nerve. 12, lower external cutaneous branch of musculo-spiral nerve.

Fig. 103.—CUTANEOUS NERVES OF THE FOREARM AND OF THE PALM OF THE HAND. (After HIRSCHFELD and LEVEILLÉ.) 9–11, as in Fig. 102. 12, cutaneous branches of the musculo-spiral nerve. 13–14, further branches of the internal cutaneous nerve. 15 and 16, terminal branches of the musculo-cutaneous nerve. 17, the radial nerve. 18, palmar cutaneous branch of median nerve. 19–23, digital branches of the median nerve. 24 and 25, digital branches of the ulnar nerve.

Just as we have seen in speaking of the cranial motor nerves—e. g., the oculo-motorius, the abducens, and the facial —the motor disturbances of the spinal nerves may be of a

paralytic or of an irritative character. In the former case the mobility of the muscles supplied by the affected nerve is diminished (paresis) or completely lost (paralysis). In the latter we have symptoms of motor irritation which are not under the control of the will, the so-called spasms. These consist either of transient muscular contractions or of a lasting state of spasmodic contraction of one or of several muscles. The former we call clonic, the latter tonic spasm.

On the whole, paralytic symptoms are much more common in the distribution of these plexuses than symptoms of irritation.

In the sensory disturbances we can equally distinguish paralytic from irritative conditions, the former giving rise to anæsthesia, the latter to hyperæsthesia. The anæsthesia is characterized by the fact that external (mechanical, chemical, or thermic) stimuli are either not perceived at all or with diminished acuteness, whereas in hyperæsthesia, on the contrary, even very weak stimuli are felt to be abnormally strong and unpleasant. The latter condition is usually attended with symptoms of sensory irritation, namely, either by pronounced pains or by paræsthesias—that is, abnormal sensations of pricking, formication, numbness, or a " furry feeling."

The affections of the sensory fibres of the spinal nerves chiefly manifest themselves by symptoms of irritation. They are always associated with more or less pain and are called neuralgias. That these also occur in the cranial nerves has already been stated, and the trigeminal neuralgia (cf. page 68) may be taken as a type of them. The neuralgic pains are usually very violent, but are rarely or never constant. They appear periodically and follow fairly accurately the distribution of the affected nerve. The diagnosis is rarely difficult. Peripheral anæsthesias—that is, such as are only due to affections of the peripheral nerves or their end organs—are, as we said, rare.

CHAPTER I.

THE cervical plexus is, on the whole, much less frequently affected with motor disturbances than the brachial. Among the nerves belonging to it, it is especially the phrenic which may present symptoms of paralysis or of irritation; yet neither paralysis nor spasm of the diaphragm due to disease of the phrenic is of any great practical importance, since neither affection scarcely ever occurs by itself, but is much more often met with only when associated with other diseases. Paralysis, for instance, is observed in the course of progressive muscular atrophy, in hysteria, probably also in lead poisoning. Traumatism or mechanical compression produced by tumors or abscesses in the neck may be the cause. Among the signs of paralysis of the diaphragm there is chiefly one which is very conspicuous—namely, the faulty expansion in the epigastric region during inspiration. Instead of becoming prominent, as is the case in the normal condition, the epigastrium is drawn in, and when we lay our hand on it we can feel that the diaphragm does not descend. If only one of the phrenic nerves is thus affected this phenomenon is present only on one side, while the other half of the diaphragm performs its function properly. Besides this, hardly any other symptoms are observed in uncomplicated cases if the patient remains at rest, whereas if he exerts himself a distinct dyspnœa and an increase in the frequency of the respiration become apparent. The obstinate constipation which such patients complain of can well be understood if we remember the part which the diaphragm takes in the abdominal pressure.

Spasm of the diaphragm, at least the tonic form of it, is not more common than paralysis. Patients affected with this suffer from great shortness of breath and quickly become cyanosed. The markedly prominent epigastrium remains with the dia-

phragm immobile and is tender to the touch, and only the upper part of the thorax shows shallow respiratory movements. In some cases of tetanus, tonic spasm of the diaphragm seems to be the cause of death. It occurs almost never by itself without some accompanying or underlying affection, except in hysterical persons. On the other hand, the clonic form of the spasm, the so-called hiccough (singultus), is extremely common. Everybody is familiar with the short clonic movements of the diaphragm, which are accompanied by inspiratory sounds and which vary in frequency and severity, occurring sometimes in such rapid succession that eighty or even a hundred may be counted in one minute. Severe protracted hiccough may become very troublesome, indeed, even dangerous, if sleep is for a long time seriously interfered with. This is, however, only the case if singultus occurs as a symptom in the course of other diseases—e. g., in apoplexy, in peritonitis, in chronic gastric catarrh, etc. As a rule it is arrested without any interference on the part of the physician by popular methods, such as holding the breath, closing the glottis, and then attempting an expiration.

Therapeutics is almost powerless in the face of affections of the motor fibres of the phrenic. In paralysis electrical stimulation of the nerve, in the (tonic) spasm chloroform and morphine have been recommended; yet these measures have by no means been effectual.

The sensory fibres which the phrenic takes up in its course, and which are distributed to the pleura, the pericardium, and the peritonæum, may also be affected. Neuralgia of the phrenic is rare, or perhaps we had better say is undoubtedly but rarely recognized. The pains, starting at the base of the thorax at the points corresponding to the insertions of the diaphragm and radiating in all directions, are taken for rheumatism of the chest muscles or intercostal neuralgia, and it is only in cases where the pain is felt directly over the scalenus anticus and corresponds to the course of the nerve that the diagnosis is made correctly. Valleix's painful points can occasionally be demonstrated on the spinous processes of the upper cervical vertebræ and at the points of insertion of the diaphragm. Respiration is only interfered with if the mobility of the diaphragm is at the same time impaired.

The ætiology of the disease is obscure; more especially are we ignorant of the conditions under which it may occur in-

dependently. It seems not to be a rare accompaniment of Graves' disease, of angina pectoris, and of sclerosis of the coronary arteries.

Another apparently more important neuralgia in the region of the cervical plexus is the occipital or cervico-occipital neuralgia, which attacks by preference the occipitalis major, but also the minor, further the auricularis magnus (cf. Fig. 100), and the superficial cervical. The patients complain of pain in the whole occipital region, in the neck, often, too, in the ears. Much more rarely the pain radiates in a forward direction to the cheek and the lower jaw. In the intervals the sufferer is perfectly comfortable, while during the attacks the pain becomes very intense, although it never reaches the pitch of some trigeminal neuralgias. Such patients dread every motion of the head and carefully avoid every cause for laughing, as this, as well as sneezing, chewing, and so forth, is liable to bring on an attack. The consequent rigid position of the neck is quite characteristic for this form of neuralgia. Painful points can sometimes be found at the exit of the occipitalis major— that is, about halfway between the mastoid process and the spinous processes of the cervical vertebræ. Where they are absent the disease may be confounded with torticollis rheumatica; yet such a mistake may be avoided by remembering that the neuralgia is not constant, but characterized by intervals of perfect ease.

The course of an occipital neuralgia is often tedious, but, on the whole, it is not unfavorable, and complete cures are not rare. The prognosis is only bad if there be some organic lesion of the nerve, caused, for instance, by disease of the cervical vertebræ. If, too, as is usually the case, no definite cause can be found, energetic counter-irritation to the skin, local bleeding, or galvanization, are sufficient to effect a cure or at least an improvement. Subcutaneous injections of morphine we shall probably in most cases be able to dispense with. The removal of every deleterious cause should, of course, be insisted upon. Under certain circumstances the occupation has something to do with it. Thus, I have found that the stevedores of the London docks, who carry extremely heavy weights on their backs which press upon the neck and the occiput, are frequently subject to occipital neuralgia (cf. Hirt, Krankheiten der Arbeiter, iv, 91).

22

THE brachial plexus may be diseased in its supra- or infra-clavicular portion. The affections of the latter, undoubtedly the more frequent, are of greater practical importance than those of the former.

Here, too, the motor disturbances are more prevalent, sensory disturbances in the region of the brachial plexus, especially neuralgias, being decidedly exceptional.

In the supraclavicular portion, above all, the posterior thoracic—which, coming from the fifth and sixth cervical nerves, supplies the serratus magnus—is affected in an interesting and very striking manner.

The so-called serratus paralysis is quite frequently due to the calling of the patient, as certain occupations seem particularly to predispose to it. If prolonged pressure is frequently exerted on the nerve—as, for instance, is the case in people who carry heavy loads on their shoulders, or if the shoulder muscles, especially the serratus, are overexerted, as happens, for instance, in mowing, in certain manipulations of tailors, shoemakers, etc.—the paralysis has been known to develop rapidly. Occasionally such ætiological factors are absent and we are forced to fall back upon the still obscure influence of what is called "catching cold."

The condition is quite characteristic whether the arm be in a state of motion or at rest. In the latter position the scapula appears elevated and approaches with its lower angle the vertebral column more than normally, the inner median margin having an oblique upward and outward direction. The cause of this deformity is to be sought in the overaction of the antagonists—the rhomboids, the levator anguli scapulæ, and the trapezius (Fig. 104). On moving the arm, the patient, we find, can raise it only to the horizontal position, owing to the absence

of the action of the serratus, which pushes the scapula forward. As soon as we produce artificially the action of this muscle by fixing the shoulder blade and pushing it forward, complete elevation of the arm is possible. If the patient attempts this same motion himself the scapula is approached to the spinal
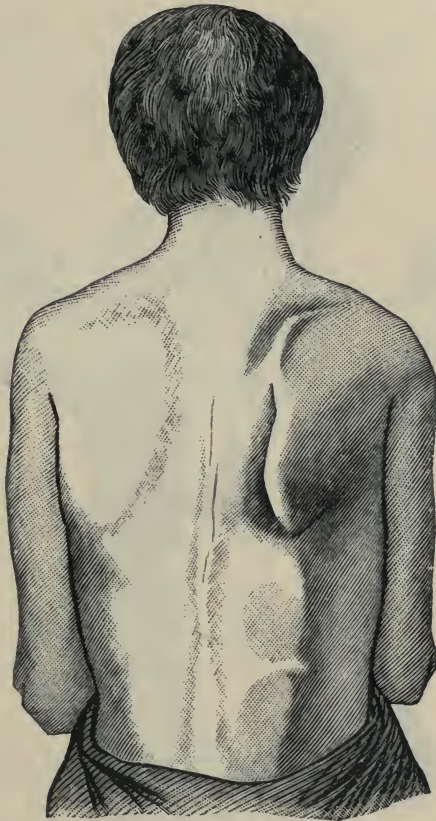
Fig. 104.—CASE OF RIGHT-SIDED SERRATUS PARALYSIS IN A MAN THIRTY-FIVE YEARS OF AGE. Position of the scapula with the arm hanging down. (After EICHHORST.)

column. If the arm is raised in front of the chest the inner edge of the scapula is elevated and stands off from the thorax in a wing-like fashion, so that we are able to touch the inner surface of the bone (Fig. 105). This appearance, which is very characteristic, is perfectly sufficient in conjunction with the other signs to assure the diagnosis. Besides a moderate impairment in adduction, which somewhat interferes with the folding of the arms across the chest, there are no other abnormi-

ties to be mentioned. Especially is it to be noted that there are no decided sensory changes to be perceived in a pure serratus paralysis. As this affection is not rarely met with in the course of progressive muscular atrophy—sometimes this disease begins with a serratus paralysis—it is not to be wondered

Fig. 105.—The same Case with the Arms raised.

at that the muscle at fault is sometimes found to be wasted. In the traumatic paralysis the atrophy comes on very late—many years after the traumatism. The muscle remains intact, electrical reactions are normal—reaction of degeneration being by no means always demonstrable—and yet there is no improvement. The prognosis, on the whole, is bad; the disease even in the most favorable cases is of very long duration, and may last for weeks, months, or years. Often it is not curable at

all, and the patient is, as it were, maimed for the rest of his days.

Not too much hope ought to be placed in the electrical treatment, no matter in what form electricity be employed; in grave cases, at least, such hopes are doomed to disappointment.

The paralyses of the pectoralis major and minor (anterior thoracic nerves), of the rhomboidei and the levator anguli scapulæ (muscular branches from the third, fourth, and fifth cervical nerves), of the latissimus dorsi, subscapularis, and
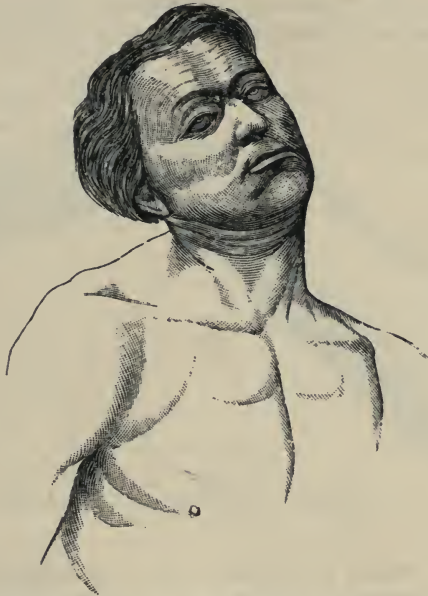
Fig. 106.—Position of the Head in Spasm of the Splenius Capitis on the Right Side.

teres major (subscapular nerves), finally, those of the supraspinatus and infraspinatus (suprascapular nerve) have alone no practical importance, and will as far as is necessary be mentioned in the consideration of progressive muscular atrophy. A lesion of the left suprascapular has been described by Hoffmann, one of the right by Bernhardt (Sitzung der Berliner Gesellschaft für Psych. und Nervenkrankheiten, March 11, 1889).

Spasms of the muscles concerned here are also unusual. A characteristic position of the head is evoked by a unilateral

spasm of the splenius capitis (cf. Fig. 106). Bilateral spasm of the deep muscles of the neck produces a strong retraction of the head, while spasm of the rhomboids alters the position of the shoulder-blades, etc.

Of the nerves belonging to the infraclavicular portion of the brachial plexus none is so frequently the seat of disease as the continuation of the posterior trunk of the plexus, which becomes the musculo-spiral or radial nerve, and supplies the skin and the muscles of the extensor surface of the arm.

The musculo-spiral paralysis can better than any other form be recognized at a glance. A patient with paralysis of the extensors is unable on stretching out the arm to raise the hand,
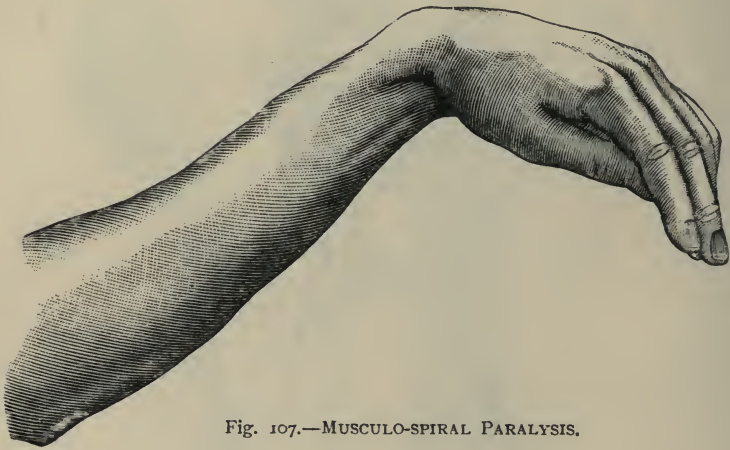
Fig. 107.—MUSCULO-SPIRAL PARALYSIS.

while lateral motion is difficult. Dorsal flexion, which is performed by the extensor carpi radialis and the extensor carpi ulnaris, is impossible, abduction and adduction difficult, the hand hangs down flaccidly ("wrist-drop") (cf. Fig. 107), and when rested upon the table can not be raised. On a more careful examination it is noticed that the first phalanx of the flexed fingers can not be extended without assistance, but that if this phalanx is passively extended the patient can straighten out the others himself. The first condition is due to the paralysis of the extensors, which, as is well known, on the dorsal surface of the first phalanx pass into an aponeurosis; the second to the preservation of the function of the interossei, which are supplied by the ulnar nerve. Since its extensors are also implicated, the thumb, of course, can not be actively

extended, neither can it be abducted, because the muscles con-
cerned are also paralyzed. Some interesting conditions will
be found on examination of the forearm in extension and flex-
ion. If, for instance, the forearm is extended and pronated,
supination is impossible, because the supinator brevis is para-
lyzed. During flexion of the forearm, however, the biceps,
which is intact, can perform supination without difficulty. If
the forearm is in a position of supination it is easily flexed by
the intact muscles, the biceps and the brachialis anticus, while
if it is half pronated flexion is imperfect, owing to the paral-
ysis of the supinator longus. The characteristic prominence
formed by the belly of this muscle when the forearm is flexed
is absolutely wanting. Any participation of the triceps in the
paralysis is only observed if the lesion is high up ("crutch
palsy"). Usually the injury is situated where the nerve turns
over the humerus or lower down, in which case naturally the
normal function of the triceps is not interfered with. Perma-
nent trophic disturbances, shown by pronounced wasting of
the affected muscles, are rare in cases of pressure paralysis,
while they are frequent in the paralysis developed as a conse-
quence and in the course of lead poisoning.

The flexors, otherwise perfectly healthy, also become weak-
ened, because their points of insertion are approximated to the
points of origin more closely than under normal conditions, on
account of the constant drooping of the hand, and hence the
interference with motion is aggravated. The patient can hardly
use the hand at all, he is unable to take hold of anything, the
finer manipulations necessary for writing, drawing, etc., are
impossible, and in the majority of cases he is unfit for work or
for making a living during the whole course of the disease.

Sensory changes are rarely sufficiently marked to add much
to his troubles. Sometimes paræsthesias may be complained
of—a feeling of cold, numbness, formication, and the like; some-
times, also, there is a distinct decrease of sensibility, so that
zones of anæsthesia can be made out; but these changes are
of relatively small importance, and, as we said, inconvenience
the patient but little. On the other hand, a source of great
annoyance is found in the peculiar painless swellings of the
extensor tendons on the back of the hand. These node-like
swellings have been described by Gubler as tenosynovitis hy-
pertrophica, and are to be attributed to mechanical influences
acting injuriously on the tendon.

The duration and course of a musculo-spiral paralysis may vary greatly, and it is often hard to give an opinion on these points at the very onset of the affection. An electrical examination, which reveals the reactions of the muscles and nerves to the faradic and galvanic current, is the only means by which we can arrive at an opinion as to the duration of the disease. The conditions are the same as those we described as existing in facial paralysis, and it suffices, therefore, to refer the reader to that chapter. But here again be it once more stated a prognosis should never be given without a previous electrical examination of nerves and muscles.

The ætiology of musculo-spiral paralysis is interesting from the fact that it is fairly well understood. While, as all confess, the cause of most nervous diseases is absolutely unknown, and we therefore are forced to fall back on uncertain explanations, such as exposure to cold, it seems according to our present knowledge that musculo-spiral paralysis always can be traced back to one of two kinds of causes—viz., mechanical or chemical. There are quite a number of lesions due to mechanical or traumatic causes. Frequently a man, when greatly fatigued, drunk, or exhausted, goes to sleep, using his arm, usually the left, as a support for his head; the latter, pressing on the nerve in the lower third of the humerus, gives rise to an injury in a relatively short time, or the arm supporting the head of the sleeper may press with its outer side against a chair or the like and a paralysis be the result. This is the so-called "sleep palsy." Next we have compression happening to the patient as a consequence of his daily occupation, due to pressure from ropes, handles of water-jars (as in the water-carrier paralysis of Rennes, etc.); sometimes in infants this paralysis occurs from too much compression on the arms by too tight swathing-clothes; sometimes too tight plaster-of-Paris bandages have been the cause; and, finally, all direct injuries to the nerve—stab wounds, blows, gunshot wounds, and compression of the nerve by abnormal callus formation after fracture of the humerus—must also be mentioned.

The lesions due to chemical causes may be the result of the action of certain poisons, among which lead deserves to be mentioned first. It is a fact no less remarkable than well authenticated, to which we shall again refer when speaking of lead poisoning in general, that this agent acts by preference on the muscles which are supplied by the musculo-spiral nerve.

This musculo-spiral paralysis, however, unlike the form which is produced by mechanical action, is not an independent disease, but merely a symptom of a general intoxication. According to the commonly received opinion (Leyden and others), the paralysis depends upon a degenerative atrophy of the motor peripheral nerve fibres, to which is often superadded a spinal affection. It differs in its clinical aspect from the mechanical lesion, inasmuch as the supinator longus and the triceps remain intact. Of late years several cases have been published where, after subcutaneous injections of ether into the extensor surface of the forearm for therapeutic purposes, a musculo-spiral paralysis appeared (Falkenheim, Arnozan, Remak, H. Neumann,
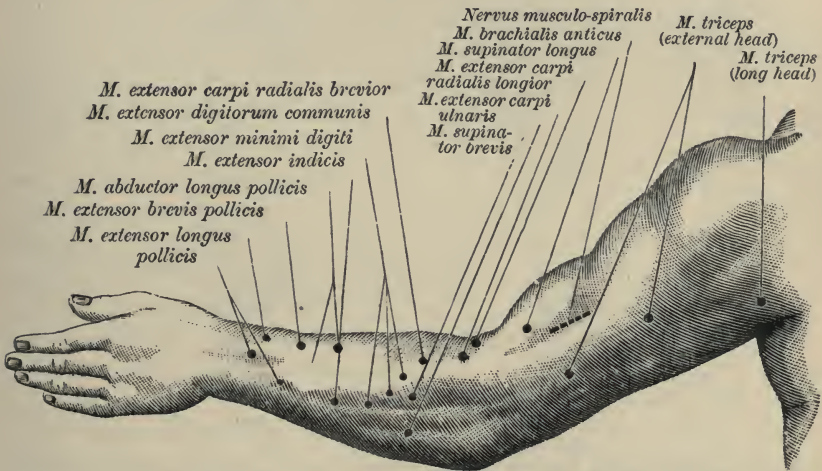
Fig. 108.—MOTOR POINTS OF THE MUSCULO-SPIRAL NERVE AND THE MUSCLES SUPPLIED BY IT.

cf. lit.). In using the drug in this way this possibility ought to be thought of.

In contradistinction to the frequency with which paralysis is found, signs of irritation in the distribution of the musculo-spiral nerve—that is, spasms—are extremely rare. They have especially been observed after manual overexertion—gymnastics, etc. (Hochhaus, Deutsche. med. Wochenschr., 1886, 47).

In the treatment electricity not only plays the chief, but the only *rôle*. From the motor points (Fig. 108) the muscles ought to be stimulated with the constant current, and besides this frequent extensive applications of the faradic brush to the skin

of the affected arm ought to be practiced. That the cause, if
such should be present—for instance, pressure of crutches, of
dislocated bones, etc.—ought to be removed is self-evident.
The sensory disturbances which may be found in the distribu-
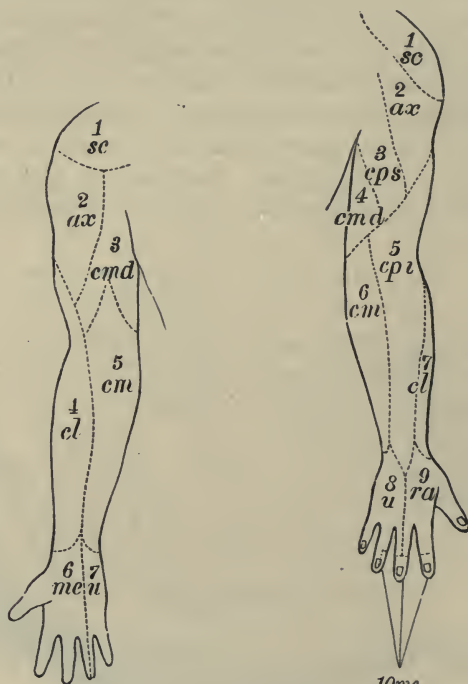
Fig. 109.   Fig. 110.

THE DISTRIBUTION OF THE CUTANEOUS NERVES OF THE ARM AND HAND. (After
EICHHORST.) Fig. 109, volar surface of the upper extremity. *1sc*, supraclavicular
nerve. *2ax*, circumflex nerve. *3cmd*, internal cutaneous nerve. *4cl*, external cutaneous
nerve. *5cm*, cutaneous medius. *6me*, median nerve. *7u*, ulnar nerve. Fig. 110, *1sc*,
supraclavicular nerve. *2ax*, circumflex nerve. *3cps*, superior posterior cutaneous nerve.
5cpi, inferior posterior cutaneous nerve. *4cmd*, internal cutaneous nerve. *6cm*, median
cutaneous nerve. *7cl*, external cutaneous nerve. *8u*, ulnar nerve. *9ra*, musculo-spiral
nerve. *10me*, median nerve.

tion of the musculo-spiral we shall mention when considering
cervico-brachial neuralgia. The mode of distribution of the
cutaneous nerves of the upper extremity is illustrated in Figs.
109, 110, 111.

The median and ulnar nerves supply together the innervation of
the muscles and the skin of the inside of the forearm and the hand,
the former innervating almost all the flexors of the forearm, the

pronator radii teres, and the pronator quadratus, the flexor carpi radialis, the flexor sublimis digitorum, and a part of the profundus, leaving the flexor carpi ulnaris to the ulnar. Among the thenar muscles the median supplies the abductor brevis, the opponens, the outer head of the flexor brevis, further, the three first lumbricales, while it again leaves to the ulnar, besides the one flexor mentioned, the antithenar, the adductor brevis pollicis, the deeper head of the flexor brevis pollicis, the fourth lumbricalis, and all the interossei.

Both nerves have this in common—that they only rarely become affected by themselves, much more rarely than the musculo-spiral, and that they are, unlike the latter nerve, liable to disturbances not only in their motor, but also in their sensory fibres. We shall have to speak, therefore, not only of paralyses, but also of neuralgias. With regard to the ætiology, we may consider it as the rule, just as in musculo-spiral paralysis, that motor disturbances only occur as a consequence of mechanical injury, provided there be no other disease present—e. g.; progressive muscular atrophy and the like—while neuralgias may appear under other circumstances—e. g., after acute diseases, after exposure to cold, sometimes also

Fig. 111.—Distribution of the Sensory Nerves on the Back of the Fingers (Krause). *r*, musculo-spiral nerve. *u*, ulnar nerve. *m*, median nerve.

without any demonstrable cause. The ulnar paralysis may be caused by certain occupations, as Duchenne has already observed repeatedly in workingmen who are obliged to press the elbow firmly upon a hard surface. It is not a rare occurrence in those who have to use the ulnar side of the hand—hypothenar eminence—a great deal to strike certain instruments (cabinet-makers, dyers, cobblers, etc.).

A pure median paralysis is chiefly characterized by the inability to pronate the forearm and to flex the hand, as we can easily understand from the anatomy of the parts. A very slight flexion of the hand toward the ulnar side is, however, rendered possible by the action of the intact flexor carpi ulnaris. The terminal phalanges can not be bent, but in the first phalanges, which are under the control of the interossei, this motion is not impaired. The part of the flexor profundus digi-

torum which is supplied by the ulnar makes it possible for the patient to seize some objects ·with the third, fourth, and fifth fingers. The extended and adducted thumb, which lies in close apposition to the index finger, is as good as useless.

On the other hand, we find in ulnar paralysis that the thumb can not be pressed against the index finger on account of the paralysis of the adductor pollicis, that the terminal phalanges of the fingers can not be straightened, the first ones not flexed (paralysis of the interossei), and that the little finger is almost wholly useless. With the median paralysis the ulnar
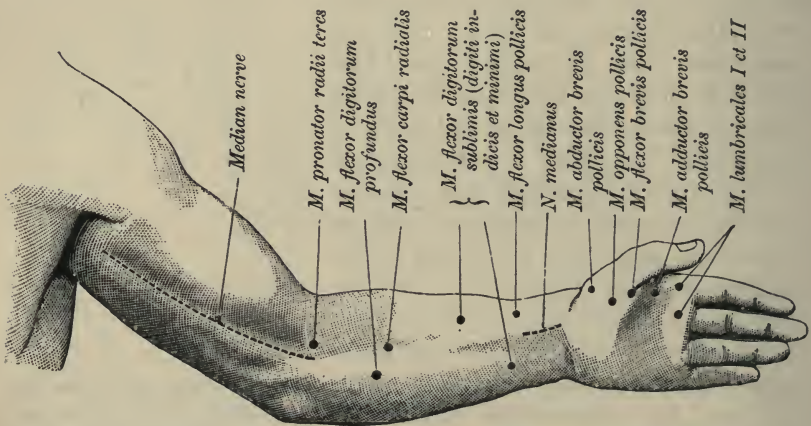
Fig. 112.—Motor Points of the Median Nerve and the Muscles supplied by it.

form has this in common, that flexion at the wrist joint is greatly impaired. In the latter especially lateral movement toward the ulnar side is interfered with owing to the paralysis of the flexor ulnaris. Lastly, the difficulty which is experienced by the patient in spreading his fingers apart and bringing them together again, movements which are indeed almost impossible, greatly facilitate the diagnosis of ulnar paralysis, which, however, for that matter, is always simple.

Muscular atrophies not uncommonly develop in both of these paralyses, but more frequently in the ulnar form. The interosseal spaces on the back of the hand become sunken in, and, if the wasting affects chiefly the interossei and the lumbricales, the hand assumes a peculiar appearance. It becomes not unlike a claw, since the healthy antagonists—the extensor digitorum communis and the flexor digitorum—produce a dorsal flexion of the first phalanges and a complete palmar flexion

of the second and third (cf. Fig. 115). This is called the " claw
hand," the " *main en griffe* " of the French.

Atrophy confined to the antithenar eminence I have repeat-
edly observed in cabinet-makers. They themselves attribute
it to the continued use of the plane.

The affections of the sensory fibres of the median and ulnar
nerves may either occur alone or be found associated with
those of the motor fibres. In the latter case we have to contend
with disturbances of sensibility, paræsthesias, numbness, anæs-
thesia, and pains, sometimes quite well pronounced, which es-
pecially appear in the initial stage of the paralysis. In the
former there are genuine neuralgias, acute, spontaneous, lanci-
nating pains which follow the course of the nerve and which
are intensified by pressure upon it. Such pains are more fre-
quently observed in the distribution of the median than in that

*M. lumbri-
cales III et IV*
M. opponens min. dig.
M. flexor minimi digiti
M. abductor minimi digiti
M. palmaris brevis

N. ulnaris *M. flexor carpi ulnaris* *Nervus ulnaris*
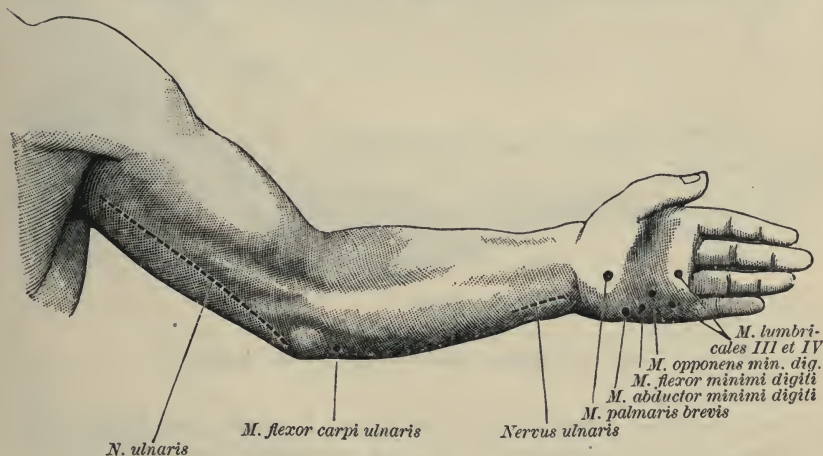
Fig. 113.

of the ulnar, but they are not common in either of these
regions. I have known them to occur occasionally after acute
diseases, especially after typhoid fever. In their course they
differ in no way from other neuralgias. The only fact remark-
able is that atrophy of the interossei and the " claw hand "
may develop in their course even when there are no motor
disturbances present. A relapse in a case of ulnar neuralgia
may occur after an interval of years, but no satisfactory ex-
planation for this has been discovered.

Lastly, we have to consider in the upper arm the musculo-
cutaneous and the circumflex nerves (Fig. 116), either of which

may be affected by itself or in connection with other nerves of the plexus. The former supplies the coraco-brachialis, the brachialis anticus, and biceps; the latter, the deltoid.

Lesions of the motor fibres of the musculo-cutaneous, which are only met with independently after injury due to surgical operations, impair and completely prevent flexion of the fore-arm on the upper arm. In lesions of the circumflex, motion of the arm away from the trunk is difficult, and even rendered impossible, if, as often happens in the course of the disease, the deltoid atrophies. This atrophy is readily recognized by the flattening of the shoulder, and is often associated with reaction of degeneration. Occasionally the participation
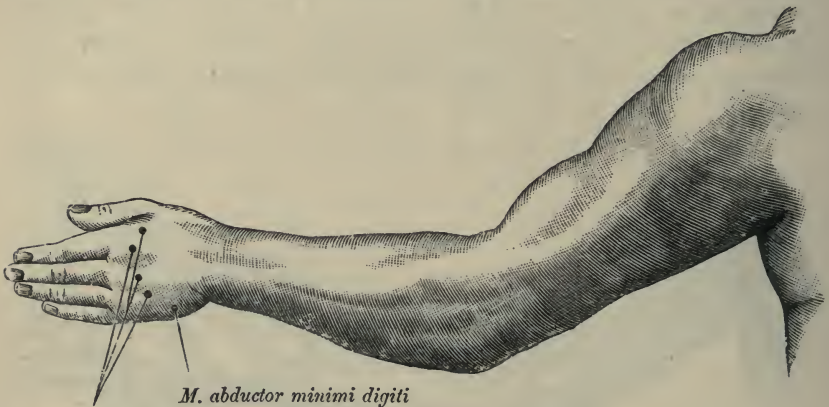
M. abductor minimi digiti

M. interossei I–IV

Fig. 114.—MOTOR POINTS OF THE ULNAR NERVE.

of the sensory fibres of the circumflex is more prominent; the patients then complain of violent neuralgic pains (Heon, cf. lit.), which are aggravated if any attempt is made to move the arm. It is important in such cases to make a careful examination of the shoulder joint, and frequently we shall find a chronic inflammation here to be the cause of the neuritis.

In other cases, again, we can not make out any organic changes in the joint, and we have to think of a joint neurosis. For information on this point the reader is referred to the chapter on hysteria. A severe concussion, a fall upon the shoulder, which at first produces hardly any symptoms, may give rise to disease, lasting for years, in which both the joint and the nerves of the plexus take part.

In any one of these affections of the nerves of the arm we

should in the treatment, besides aiming at the removal of the cause if such be found, make use as soon as possible of the galvanic current. It is a mistake to lose time with other measures, such as bathing, massage, rubbing, and the like. Where

Fig. 115.—CLAW-HAND. (After DUCHENNE.)

the electrodes are to be applied may be learned from Figs. 108 to 116, where the motor points are accurately given. We need hardly say that, besides the electricity, various placebos, rubbing and passive motion, may be used to quiet the patient's mind.

Not uncommonly several nerves of the brachial plexus are paralyzed at the same time. Duchenne was the first to describe such instances in children in consequence of obstetrical

N. musculo cutaneus
M. brachialis anticus
M. biceps brachii
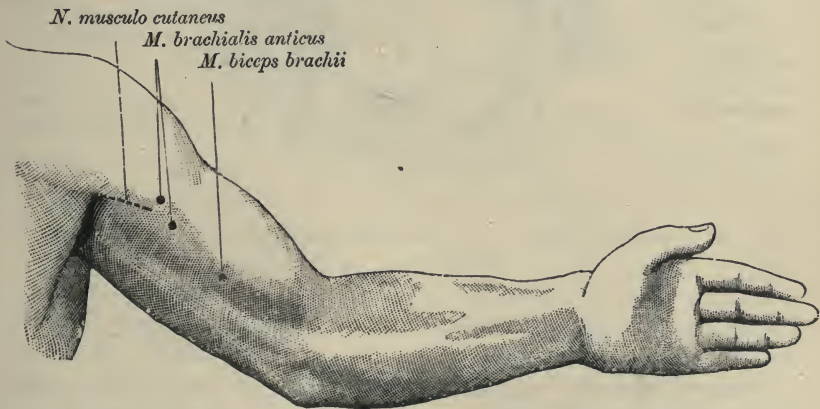
Fig. 116.—MOTOR POINTS OF THE MUSCULO-CUTANEOUS NERVE AND THE MUSCLES SUPPLIED BY IT.

operations, such as version and subsequent extraction, the Prague method of extraction, etc., and designated this form as "paralysie obstétricale infantile du membre supérieur," or birth palsies. Independently of the French investigator, Erb

has given us an excellent well-defined picture of such a paralysis. The lesion which affects the plexus gives rise to a simultaneous paralysis of the deltoid, the biceps, the brachialis anticus, and the supinator longus, and the patient can neither move his upper arm away from the body, nor approach the forearm to the upper arm. The whole extremity hangs down flaccid, while the fingers and hand retain their mobility. The

N. dorsalis scapulæ
Circumflex nerve
Post. thoracic nerve (M. serratus magnus)
Phrenic nerve
Anterior thoracic nerve (M. pector.)
Brachial plexus   *Erb's supraclavicular point*
Fig. 117.

lesion in such cases must be situated at a point where the circumflex and the musculo-cutaneous and the musculo-spiral are still close together—i. e., at about the exit of the sixth cervical nerve—between the scaleni, and it is from this so-called "Erb's" or "supraclavicular" point (cf. Fig. 117) that we are able to stimulate simultaneously all these four above-mentioned muscles. If the infraspinatus is also taken in, the arm is in a position of internal rotation, and can not be turned outward.

This paralysis, which Erb has aptly termed "combined shoulder-arm palsy," is often a very tedious and troublesome

affection. The longer it lasts the more the nutrition of the muscles suffers, and the most varied degrees of atrophy, which is often especially marked in the deltoid, are seen. On electrical examination we find that the faradic and galvanic excitability of the nerves, although not completely lost, is diminished, as is also the faradic excitability of the muscles, while the galvanic excitability of the same has undergone qualitative as well as quantitative changes, a condition which Erb has designated as partial reaction of degeneration. Sometimes, also, there is present complete reaction of degeneration (cf. page 91). If the sympathetic is also implicated (Seeligmüller), the ensuing paralytic symptoms, contraction of the pupil, narrowing of the palpebral fissure, and retraction of the bulb on the affected side, are further sources of annoyance to the patient. How the participation of the sympathetic is to be explained, whether, as Klumpke (cf. lit.) holds, by a lesion of the communicating branch of the first dorsal, we can not decide. If the sensory fibres are also implicated, the patient complains, in addition to the motor, also of sensory disturbances, not only of great difficulty in moving the arm, but also of pains, numbness, and formication.

The treatment, of course, consists in the use of electricity, galvanic stimulation from Erb's point, and the application of the faradic brush, which, acting reflexly, often give very good results.

Peculiar and very curious motor phenomena in the upper extremities are observed in connection with and as a direct consequence of certain callings. Such occur in cases where no particular exertion of the muscles might lead us to think of a peripheral lesion of the plexus as the result of overstrain, but in those whose occupations bring into play complex, co-ordinated movements. Since in many cases—but by no means in all—a faulty co-ordination of the movements is the cause of the affection, we may for the present accept the name of "co-ordination occupation neurosis," which was proposed by Benedikt, at the same time insisting upon the fact that it only fits a certain small number of cases.

Among the occupations which relatively frequently give rise to the disturbance in question the most important certainly is writing, and writer's cramp—mogigraphia, graphospasmus—is one of the nervous diseases to which most careful study has been devoted. Nevertheless, our knowledge is extremely

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limited, and we must confess that we have not as yet gotten beyond the description of the symptoms. The pathogenesis and therapeutics are *terræ incognitæ.*

In the first place, we ought to state that only in a fractional number of cases have we to deal with a cramp or spasm; more often the conditions are the following: The patient, after having for weeks, perhaps months, noticed that while writing the hand becomes tired more easily than before, finds one day that he is utterly unable to write another line without great strain; as soon as the pen is taken into the hand the sensation of fatigue comes on; hand and arm drop as if paralysed, while at the same time the patient may complain of more or less intense pain in the forearm, upper arm, and possibly in the shoulder. The writer's cramp in such cases is in reality a writer's paralysis. In other instances, as soon as the penholder is clasped, the hand begins to tremble and the handwriting becomes uncertain and tremulous, which is all the more striking because on examination the patient's hand, especially the right, proves to be quite steady if it is not used in writing. Sometimes there is an actual spasm when the penholder is seized, which attacks the muscles of the hand as well as those of the forearm, so that hand and arm make involuntary movements or they become stiff and immobile (clonic and tonic spasm). The pen is either irregularly jerked to and fro or firmly pressed against the paper—in both cases writing is absolutely impossible. On further examination nothing else is discovered, and what is more especially interesting, the patient is able to do anything else with his hands, even the finest work. He is able to draw (with a pencil), play the piano, etc.; moreover the electrical examination of the apparently seriously affected muscles seldom reveals anything abnormal worthy of mention—Dubois (Schweiz. Correspond. Blatt, 1887, 5) found the excitability for both currents, especially in the thenar muscles, increased. Sensibility is, on the whole, normal. Pains only occur on forced attempts to write; in short, the patient can do anything demanded of him except write.

Analogous to the affections just described are the conditions of fatigue in the muscles of people, chiefly professionals, who play the piano a great deal. In them not only the right hand, but, especially in female patients, the left also is affected. Pain and weakness may become so marked in both hands that piano-playing has to be given up completely. This becomes

the more necessary when the symptoms persist during rest as well, and not only when the patient is playing. Such disturbances are also noted in telegraph operators, cigar-makers, dairy-maids (as the result of milking cows); also, but rarely, in tailors it is produced by the frequent handling of the heavy shears, etc. In all cases it is evident that the occupation is the sole cause, although we do not know how and upon what organs it acts injuriously. It is very unlikely that the disturbance is of a peripheral nature, the negative result of the examination of muscles and nerves and the uselessness of any treatment seeming to indicate this. We can not accept either the theory which attempts to explain the symptoms by a primary weakness of certain muscles and a secondary spasm of the antagonists (Zuradelli), or that which assumes the spasm to be of a reflex nature, starting from the sensory nerves of the skin (Fritz); or, finally, the explanation that we have to deal with a disturbance in conduction of the nerve muscle apparatus used in writing; but we are rather of opinion that the weakness and the motor disturbances of the upper extremity arising in consequence of the occupation are of a central nature and are to be referred to the brain cortex. The situation of the centres concerned in writing and in other movements which depend upon a co-ordinated action of the muscles of the hands is unknown. These centres in consequence of overexertion, but also often without any appreciable cause, are thrown into a state of paralysis or irritation which gives rise to corresponding disturbances in the extremities. Perhaps this may at times arise simply as the result of a general increased nervousness which may have a hereditary origin. It is evident that besides those affections which are due to a functional disturbance of the cortex there are those in which anatomical lesions, whether of the central organs or of the peripheral nerves, may be the cause of the same symptoms as those now under consideration. Thus we may sometimes meet with cases of old almost cured hemiplegias in which as the only remaining disturbance a slight difficulty in writing or similar occupations may be present. The same may happen in slight disseminated scleroses of some collections of fibres in the spinal cord, or, finally, as I have had occasion to observe repeatedly, in the initial stage of tabes, and the disturbance at the first glance may suggest to us writer's cramp. Hence we should, first of all, endeavor to decide whether the trouble is an independent affection or whether

it is to be regarded merely as a symptom of an underlying disease.

The prognosis is usually unfavorable. Only in the rarest instances are we able to afford the patients any decided lasting relief, a fact of which we should inform the friends before taking charge of the case. Only when we are able to get hold of the patient in the earliest stages of the trouble and can insure him perfect rest and the removal of the exciting cause, such as writing, piano-playing, telegraphing, etc., for weeks and months, is it sometimes possible to effect an absolute cure. If this can not be done, and if the rest is not complete, the success of all our attempts becomes very uncertain and the result will usually be disappointing. We may try massage, as has been done also by some non-professional specialists with transient success. Galvanism, faradism, rubbing with different external applications, hydrotherapy, gymnastics, may be advised. The result is usually the same as if strychnine or atropine is injected hypodermically or if the different nervines be given internally for months. Writing may be facilitated by using a penholder passed through a potato or through a wooden ball fitted to the hollow of the hand, or by using Nussbaum's bracelet. The advice to educate the left hand to write is always good because it gives the right hand a rest. Yet the value is by no means lasting, because the motor disturbance, as a rule, shows itself soon in that hand also, a fact which is an additional argument in favor of the central nature of the disease.

The simultaneous affection of several sensory nerves of the brachial plexus, analogous to the motor disturbance in the shoulder-arm palsy, is not common. When it does occur the pains are very violent and deprive the patient of the use of the extremity. The cervico-brachial neuralgia may affect all the sensory branches of the brachial plexus, so that the whole upper arm, forearm, and hand are painful; but it may also be confined to the area of distribution of one nerve, often the musculo-spiral or median.

Painful points can sometimes be demonstrated in the region of the circumflex nerve over the scapula, of the median in the bend of the elbow, of the musculo-spiral in the lower third of the humerus, and of the ulnar at the internal condyle. Vaso-motor and trophic changes may be entirely absent, yet the skin of the fingers not rarely looks glossy and atrophic ("glossy

fingers "). Here, again, traumatism, mechanical pressure—by tumors, aneurisms, etc.—are the most prevalent causes of the neuralgia. It may occur reflexly after amputation of the fingers or the forearm. A bilateral neuralgia of this kind is suggestive of a spinal disease, more especially of pachymeningitis cervicalis hypertrophica.

The treatment is in the main the same as in other neuralgias. Besides narcotics the electrical treatment should be begun as soon as possible. Descending currents through the diseased nerve, as well as the application of the anode over the affected plexus, are to be recommended. The faradic brush is usually borne well and is of use, although the manipulation itself may not be very agreeable to the patient. In rare instances we must have recourse to energetic counter-irritants to the skin. We have repeatedly made very successful use of the *points de feu* with Paquelin's cautery.

Paræsthesias and anæsthesias are quite common in the distribution of the brachial plexus. They are not always confined to one nerve, Upper arm and forearm, the hands also, are frequently affected, particularly when the occupation necessitates overexertion of them—e. g., in brick-makers. Again they are caused by the action of cold and hot water, often also by water containing lye (anæsthesia lavatricum, and the *mal des bassins* of the women engaged in unwinding the silk from the cocoons in the silk-spinning mills, etc.). For such patients the only remedy lies in abstention from this kind of work.

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II. *Diseases of the Dorsal Nerves.*

The anterior (ventral) divisions of the twelve dorsal nerves are called the intercostal nerves, since they run in the intercostal spaces. They supply the intercostal muscles, the levatores costarum, the serrati postici, and the three broad abdominal muscles. To the integument of the chest and abdomen they supply cutaneous branches. The posterior divisions of the dorsal nerves are divided into internal and external branches. The former are distributed to the deep muscles of the back, sending nerves to the rhomboidei and the latissimus dorsi; the latter, passing between the longissimus dorsi and the sacrolumbalis, also furnish numerous muscular branches, and, together with the internal, supply the skin of the back as far down as the crest of the ilium.

The sensory as well as the motor fibres of the dorsal nerves may become the seat of disease, but, and this is practically of

much importance, the anterior, the intercostal nerves, are more subject to sensory disturbances, while the diseases of the posterior branches are almost exclusively motor affections.

The disease of the anterior branches, the so-called intercostal neuralgia, is found with relative frequency in the female sex, especially in those of middle age. Ætiologically, occupation and hard work in general are of some importance. Servant girls and women of the poorer classes suffer more frequently than others. I have seen many such instances, and have found it besides in the course of phthisis pulmonalis when associated with peripheral neuritis. Traumatism, aortic aneurism, and spinal affections, may also give rise to intercostal neuralgia.

The pain appears in paroxysms and attacks more frequently the left than the right side, and almost exclusively the anterior or lateral, rarely the posterior portion of the nerve trunks. It often follows the course of the nerve and at times reaches a degree of intensity most distressing to the patient. The respiratory movements, more especially coughing and sneezing, cause great agony. Three tender points can usually be demonstrated—one close to the vertebral column, one in the middle of the course of the nerve, and one close to the sternum—called respectively the vertebral, lateral, and sternal points. The fact that frequently after cessation of the pain a herpes zoster appears is of great interest, although the question whether we have to regard the latter as a genuine trophic disturbance or simply as an extension of the inflammation from the nerve endings to the skin, as Dubler thinks, is still unsettled. For the prognosis it is without significance. In all cases of intercostal neuralgia the prospect for complete recovery is slight. Although we may succeed sometimes in cutting short the individual attacks, we can never be certain that they will not recur, and there are persons who all their life long are condemned to suffer from this disease.

The diagnosis is not always simple. Rheumatism of the chest muscles can easily be taken for intercostal neuralgia, and *vice versâ*. In such cases we shall find it useful to observe whether motion has any influence on the pain or whether this exists independently. If there is a history of traumatism, neuritis is always to be thought of, only we must beware of being deceived by malingerers, and to avoid this the condition of the abdominal reflex and the pupil should be examined into. The

former in the case of neuritis is increased, the latter often di-
lated on the side of the pain.   This fact was first established
by Seeligmüller, and shows that the sympathetic is often im-
plicated here just as in the affections of the brachial plexus
(Deutsch. med. Wochenschr., 1887, 45).

In the treatment morphine plays the most important *rôle*,
and, as a matter of fact, it is of much more value than the much-
lauded subcutaneous injections of osmiumine (one syringeful
of a one-per-cent solution at a dose), for this not only fre-
quently disappoints us, but also produces local troubles, small
abscesses, etc., so that the patient is left almost in a worse con-
dition than before.   The faradic brush, the *"points de feu"*
with Paquelin's cautery, blisters applied to the painful points,
may be tried ; but, on the whole, these means effect but little.

Among the intercostal neuralgias, the so-called mastodynia
(the irritable breast of Cooper), a neuralgia of the mamma, is
to be included.   This is a not very frequent affection of the
female after puberty and may be connected with lactation.   It
is a very painful and distressing trouble, against which usually
all remedies are tried in vain, so that in desperate cases the
patient herself suggests amputation of the breast to get rid of
the dreadful suffering.   The ætiology is obscure.   Traumatism
is rarely the cause.   Ill-fitting corsets may have some influence,
but women with well developed and those with small breasts
are equally liable to the affection.   The hyperæsthesia of the
skin often hinders a careful examination by palpation.   With
the tips of the fingers we should endeavor to determine
whether there are hard nodules in the tissue, which to the in-
experienced often suggest beginning carcinoma.   In some
cases my patients have derived some transient benefit from
suspension of the breast and the application of hot cloths.
Here also morphine is indispensable (cf. Terrillon, Des neu-
ralgies du sein ; Progr. méd., 1886, xiv, 10).

The motor disturbances affecting the muscles of the back
supplied by the posterior branches of the dorsal nerves are
generally paralyses.   We are far from being familiar with the
symptoms of the affections of every one of these muscles, and
must content ourselves for the present with mentioning the
paralysis of the erector spinæ, the sacro-lumbalis, and the lon-
gissimus dorsi, which may be affected in the lumbar, dorsal,
or cervical portion of the vertebral column.   Bilateral paraly-

sis causes curvature of the spine backward (kyphosis), unilateral paralysis lateral curvature (scoliosis). Paralysis or paresis of the erectors in the lumbar region gives rise to a characteristic walk and a characteristic position of the body. The upper part of the body is bent strongly backward, so that the lumbar part of the vertebral column is markedly curved forward. If by any movement the upper part of the body is brought forward so that its centre of gravity is no longer behind that of the whole body, the patient falls forward, or, if the patient sits on the floor, he has the greatest difficulty in getting up. The manner in which he raises himself is so
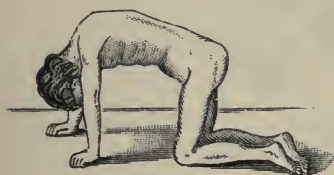
Fig. 118.
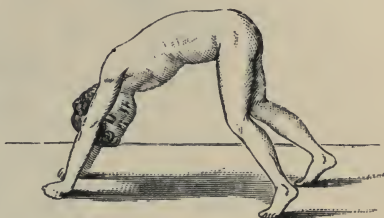
Fig. 119.
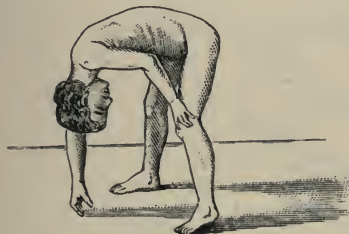
Fig. 120.

Fig. 121.

Figs. 118–121 illustrate the manner in which a child whose erectores spinæ are paralyzed gets up from the ground. (After GOWERS.)

characteristic of paralysis of the erector muscles that we have represented it in Figs. 118 to 121. The patient first gets upon all fours, and then climbs, as it were, with his hands up his own legs, constantly endeavoring to bring the upper part of the body as far back as possible by movements in the shoulders and the arms so that the abdominal muscles may resume the duty of balancing the body. This mode of getting up can best be studied in pseudo-hypertrophic paralysis.

III. Diseases of the Lumbar Nerves.

The posterior lumbar nerves are, like the dorsal, divided into outer and inner branches, which are distributed to some of the mus-

cles of the back and the skin of the lumbar and gluteal region. The
anterior, by far the stouter, are connected each with the correspond-
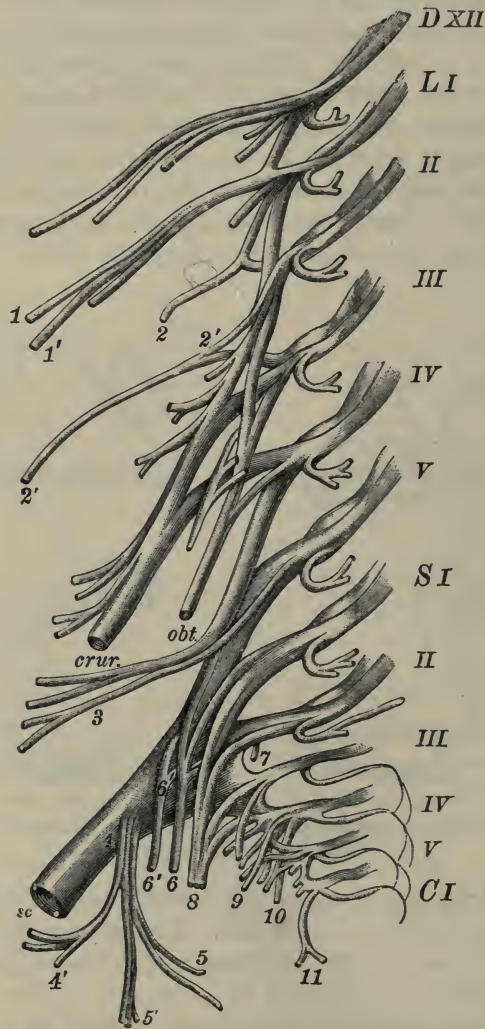
Fig. 122.—DIAGRAMMATIC OUTLINE OF THE LUMBAR AND SACRAL PLEXUSES. *D XII*,
last dorsal nerve. *L I–V*, the five lumbar nerves. *S I–V*, the five sacral nerves. *C I*,
the coccygeal nerve. 1, ilio-hypogastric nerve. 1′, ilio-inguinal nerve. 2, genito-cru-
ral nerve. 2′, external cutaneous nerve of the thigh. *crur*, anterior crural nerve. *obt*,
obturator nerve. 3, superior gluteal nerve. *sc*, great sciatic nerve. 4, small sciatic
nerve. 4′, inferior gluteal nerve. 5, inferior pudendal nerve. 5′, posterior cutaneous
nerve of thigh and leg. 6, 6, branch to obturator internus and gemellus superior. 6′, 6′,
branch to the gemellus inferior, quadratus femoris, and hip joint. 7, twigs to the pyri-
formis. 8, pudic nerve. 9, visceral branches. 9′, twig to the levator ani. 10, perforat-
ing cutaneous nerve. 11, coccygeal branches.

ing ganglion lumbale of the sympathetic. They form the lumbar plexus which lies behind and in the psoas muscle. Its branches are (Fig. 122): (1) The ilio-hypogastric nerve, for the transversalis and
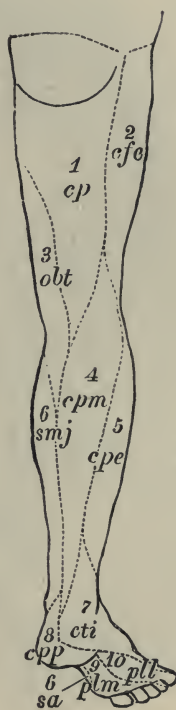
Fig. 123.   Fig. 124.

AREAS OF DISTRIBUTION OF THE CUTANEOUS NERVES OF THE LOWER EXTREMITY.
(After HENLE.) Fig. 123, anterior surface. 1, middle cutaneous nerve. 2, external cu-
taneous nerve. 3, ilio-inguinal nerve. 4, genito-crural nerve. 5, external spermatic.
6, posterior cutaneous nerve. 7, obturator nerve. 8, internal saphenous nerve. 9, com-
municating peroneal. 10, superficial peroneal. 11, deep peroneal. 12, communicating
tibial. Fig. 124, posterior surface. 1, posterior cutaneous nerve. 2, external cutaneous
nerve. 3, obturator nerve. 4, median posterior femoral cutaneous. 5, communicating
peroneal. 6, saphenous nerve. 7, communicating tibial. 8, plantar cutaneous. 9,
median plantar nerve. 10, lateral plantar nerve.

the internal oblique; (2) the ilio-inguinal, for the skin of the pubes and the genitals (N. scrotales et labiales anteriores); (3) the genito-crural, which divides into the external spermatic or genital branch and the lumbo-inguinal or crural branch, the former supplying the spermatic cord, the cremaster muscle, and the testis, the latter the

skin in the inguinal region ; (4) the external cutaneous, for the skin down to the knee; (5) the obturator, which gives off a posterior branch to the obturator ext. and adductor magnus and an anterior branch to the skin of the inner side of the thigh ; and (6) the anterior
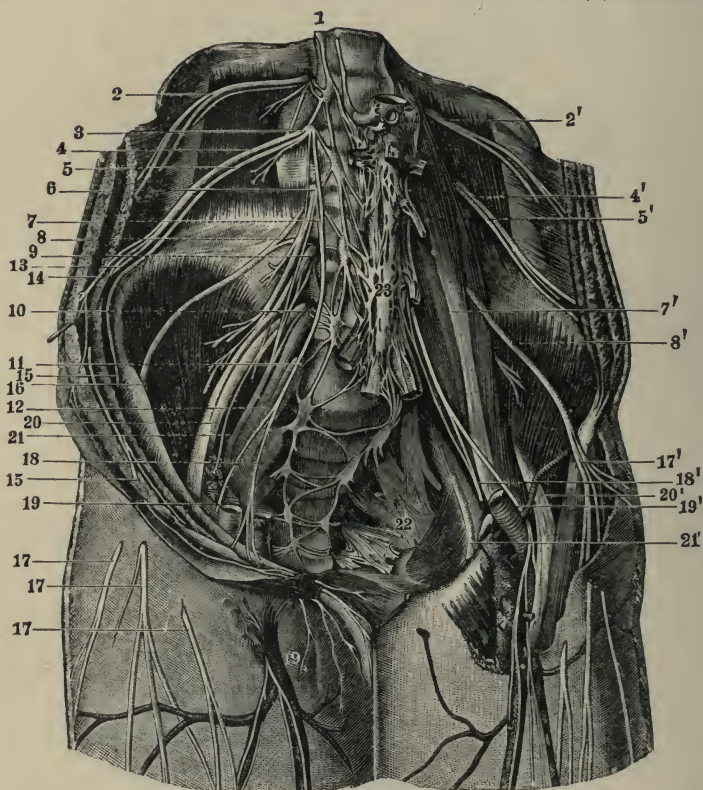
Fig. 125.—Branches of the Lumbar Plexus. (After Hirschfeld and Leveillé.) 1, lumbar part of sympathetic cord. 2 and 2′, twelfth dorsal nerve (anterior division). 3, first lumbar nerve. 4, 4′, ilio-hypogastric. 5, 5′, ilio-inguinal. 6, second lumbar nerve. 7, 7′, genito-crural nerve. 8, 8′, external cutaneous nerve of thigh. 9, third lumbar nerve. 10, fourth. 11, fifth. 12, lumbo-sacral cord. 13, iliac branch of ilio-hypogastric. 14, its abdominal branch. 15, ilio-inguinal nerve. 16–17′, external cutaneous nerve. 18, 18′, genital branch of genito-crural. 19, 19′, its crural branch. 20, anterior crural nerve. 21, 21′, obturator nerve. 22, left sacral plexus. 23, aortic plexus of the sympathetic.

crural (five millimetres in width), giving muscular branches to the anterior periphery of the thigh and having also cutaneous branches —middle cutaneous, internal cutaneous, and the long or internal saphenus nerve (cf. Figs. 123 to 126).

All the nerves of this plexus contain sensory as well as motor fibres, and may hence be affected in both ways. How-

ever, these affections do not often appear independently, where-as they are frequently observed as symptoms of central, more especially of spinal diseases, and, above all, of tabes. Our description of them, therefore, will here be very brief.

Among the sensory disturbances we have first to mention the lumbo-abdominal neuralgia, in which the hip joint is affected in much the same way as the shoulder joint in cervico-brachial neuralgia, so that the whole lumbar region down to the buttock is intensely painful. Of greater practical importance is what Cooper has described as "irritable testicle," neuralgia spermatica or neuralgia of the testicle, which either only forms a part of the lumbo-abdominal neuralgia, or, as Eulenberg and others assume, is a neuralgia of the sympathetic nerve. The spontaneous pain and the tenderness may attain such a degree as to lead to states of temporary psychical exaltation. Generally only one testicle is affected and most of the instances are found in young people. Further, we would mention the crural neuralgia and the obturator neuralgia, affections which manifest themselves by pain following

Fig. 126.—DEEP NERVES OF ANTERIOR AND INNER PART OF THE THIGH. (After HIRSCHFELD and LEVEILLÉ.) 1, anterior crural nerve. 2–3, muscular branches. 4, internal and middle cutaneous nerves, divided to show the deeper branches. 5, 6, nerves to the pectineus. 7, cutaneous filament from the last. 8, inner branch of internal cutaneous nerve. 9–11, muscular branches. 12–14, internal saphenous nerve and its branches. 15–19, obturator nerve and its branches. 20, lumbo-sacral cord. 21, its union with the first sacral nerve. 22, lumbar and sacral part of the sympathetic nerve. 23, external cutaneous nerve.

exactly the course of the respective nerves. The existence of tender points is not constant and their seat varies.

The treatment must be carried out according to the principles which we shall describe later in our account of sciatica.

Even less frequently than the sensory do the motor disturbances occur by themselves. If present, they are mostly of spinal, rarely of peripheral origin. Paralyses in the distribution of the crural nerve, which interfere with the function of the ilio-psoas and the quadriceps, make it impossible for the patient to bend the thigh at the hip joint and to extend the leg after it has been flexed on the thigh. Paralyses of the obturator nerve interfere with the adduction of the thigh, and the patient is no longer able to cross the affected leg over the other. Disorders in the nerves of the gluteus, the tensor vaginæ femoris, and the pyriformis impair rotation of the thigh inward and outward. Abduction is also hindered, while the actions of walking, standing, and more especially climbing stairs, are performed awkwardly. For details the reader is referred to Duchenne-Wernicke, page 261 and following, where the normal and pathological physiology of these muscles is carefully discussed.

IV. *Diseases of the Sacral and Coccygeal Nerves.*

The posterior small branches of the sacral nerves, four of which leave the vertebral canal through the posterior sacral foramina and the fifth through the foramen between the sacrum and coccyx, form numerous anastomoses, and thus constitute what is known as the posterior sacral plexus. The anterior, much larger, branches pass into the pelvis, where the first three and a part of the fourth, together with the lumbo-sacral cord (resulting from the junction of the fifth and a part of the fourth lumbar nerves), go to form the (anterior) sacral plexus. The plexus is triangular in form and rests upon the pyriformis muscle. The several nerves unite without much interlacement into an upper, large, and a lower, small, cord or band. The upper is formed by the union of the lumbo-sacral cord with the first and second and the greater part of the third sacral nerves and is continued into the great sciatic nerve. The lower becomes the pudic nerve. The plexus gives origin to a number of collateral branches—the superior and inferior gluteal, the small sciatic, and perforating cutaneous nerves and branches to the pyriformis, obturator internus, gemelli, and quadratus femoris. The great sciatic nerve, the largest nerve of the body, divides into the internal popliteal and external popliteal or peroneal, the latter again dividing into the anterior tibial and musculo-cutaneous, the former, which becomes the posterior tibial, terminating in the internal and external plantar

nerves. The pudic nerve divides into the inferior hæmorrhoidal, the perineal nerve, and the dorsal nerve of the penis or clitoris.

The anterior branch of the coccygeal nerve is distributed to the integument over the back part and the side of the coccyx. It is joined by a branch from the fifth sacral nerve, while the posterior division is lost in the fibrous structures on the back of the coccyx.

The affections of the sacral plexus, which appear independently of any other disease, are chiefly sensory in nature. Motor disturbances, although they are perhaps numerically as common as the former, are in the great majority of instances symptomatic of spinal diseases.

Sciatica.

Among the sensory disturbances there is especially one disease which, owing to its relative frequency and obstinate resistance to treatment, has attained to much practical importance—namely, the affection of the sensory fibres of the sciatic nerve, the sciatic neuralgia or sciatica, malum Cotunnii (Cotugno, 1764). This may, as autopsies have shown, be due to an organic disease of the nerve, a genuine neuritis, or to a functional neurosis. In the former there exist varicose dilatations of the blood-vessels of the nerve, swelling, increase in volume, alterations in consistency, and a collection of serous exudation in the nerve sheath (Cotugno, Jasset). In the latter no anatomical changes can be detected. The neuritis may be due to disease of the neighboring structures, to a tenosynovitis in the lower leg (Erb), to affections of the vertebræ (spondylitis, carcinoma), or may appear independently, in which case, leaving cold out of consideration for a moment, we have usually to deal with mechanical injuries, either as the consequence of wounds, fractures, or as the result of protracted pressure (tumors of the pelvis, aneurisms, hernia, uterus gravidus, engorgement of the venous plexus of the pelvis, habitual constipation, etc.). The occupation must, moreover, be taken into consideration in the ætiology of sciatica. It may exert an injurious influence in one of two ways, either through the overexertion which it entails or through the exposure to frequent sudden changes of temperature. Of the former we have instances in those who work with the sewing machine for weeks and months for several hours a day and in those who are always lifting heavy weights (stevedores, blacksmiths, etc.). To this

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class is thought to belong "*le lumbago des forgerons*," described by Maisonneuve (Hirt, Krankheiten der Arbeiter, iv, 90). Of the latter we have instances in puddlers and those who work at smelting furnaces, etc. Sciatica is frequently seen among such people, and seems to affect more commonly the left leg, probably because in throwing the coal into the furnace it has to be extended more forcibly (Chiene, of Edinburgh, and Hirt).

As a symptom sciatica is often seen in spinal affections (myelitis, spinal meningitis), in diseases of the general nervous system, especially in tabes, where it often appears bilaterally, also in diabetes. As a sequela it has been described as following typhoid fever. Whether malarial intoxication can ever be the cause of it is uncertain. It is sometimes seen in the course of syphilis. In lead and mercury poisoning it plays an entirely secondary *rôle*.

Symptoms.—Among the symptoms of sciatica pain is the most important. The motor disturbances which sometimes occur in the course of the disease—tremor, clonic spasmodic movements, the difficulty and awkwardness in moving which interfere to a greater or less extent with standing and walking—have to be looked upon simply as the result of the pain. This varies greatly. At first it may be dull and quite bearable, but later boring in character, extending over the whole lower extremity and persisting without intermission, so that it constantly occupies the attention of the patient and forces him to a frequent change of position ; or, again, it may appear in attacks, with intervals of comparative ease, so that the patient feels fairly comfortable and is able to follow his occupation. During the seizures it may be of such excruciating intensity that it can only be compared with Fothergill's faceache or the lancinating pains of tabes.

The patient suffers usually more intensely at night after going to bed, or at least he complains more at that time, often because he can not bear the extension of the leg, often perhaps because his attention is then less liable to be distracted. Yet even in the daytime the pain may reach a considerable pitch, especially when the patient has been making attempts to walk or has been standing too much. He may have perfect ease for hours when lying quietly, and yet a few movements of flexion and extension of the affected extremity are sufficient to throw him back into the most distressing condition. The extent of the

pain also varies; generally it is felt over the whole posterior surface of the thigh and the distribution of the external popliteal nerve. It may radiate into the region of the healthy sciatic and the lumbar plexus of the affected side. The posterior tibial nerve usually remains intact. Examination almost always discloses the existence of tender points, one, for instance, at the exit of the nerve from the pelvis, one at the lower margin of the gluteus, one in the popliteal space, one on the capitulum fibulæ. There may be others, but their occurrence is uncertain and their position changeable.

Sometimes patients with sciatica are seen to put all their weight upon the healthy leg in order to diminish the pain in the affected extremity. This causes the trunk to be bent toward the healthy side and the costal margin to approach the ilium, a position which may become so habitual that a genuine scoliosis may be developed (Albert, Nicoladoni, Babinski, cf. lit.). I have known several cases in which this secondary scoliosis persisted after considerable improvement of the primary affection, whereas in other instances I have seen it disappear when the cure of the sciatica was complete.

If we have to deal with a genuine neuritis trophic changes will be found to develop, especially more or less marked atrophy of the muscles, with reaction of degeneration (Nonne). The patellar reflex seems in such cases to be considerably diminished. An exaggerated knee jerk in the course of a peripheral neuritis has, on the whole, to be looked upon as exceptional (Strümpell, Möbius). In sciatica I have never seen it. If the trouble is purely functional the muscles and reflexes remain, even after years of suffering, unaltered. Other sensory changes—diminution of the sensibility, anæsthesias, paræsthesias—occur, but take a very secondary position to the dominating feature of the disease, which is pain.

Course.—The course as well as the duration vary greatly, but we may state as an undeniable fact that it is exceptional to find cases which last but a short time and end with complete recovery. Mostly it is a question of months and years before any decided lasting improvement is brought about. On the other hand, remissions are not rare. They may last for months and the condition of the patient may be such that he begins to be confident of a permanent cure, when suddenly, often without any appreciable cause, sometimes in consequence of a long walk, the pain again makes its appearance with un-

diminished intensity and the treatment has to be started all over again. The more frequent such relapses, the more gloomy becomes the outlook for complete recovery.

Diagnosis.—Great care should be exercised in the diagnosis, and we should first endeavor to decide whether the trouble has to be regarded as an idiopathic affection or as a symptom of another malady, and more especially in bilateral sciatica should we be on the lookout for a spinal disease or a disease of the general nervous system, such as tabes. The examination of the urine for sugar should never be omitted. If this proves negative, and if we can exclude general nervous diseases with certainty, we should proceed to analyze the pain, to examine into its nature, the time of its occurrence, its seat and extent, and should keep in mind that there are other than nervous affections that are associated with violent pain in the lower extremities, for instance, acute rheumatism of the lumbar muscles, lumbago, also inflammations in the hip joint, chronic hip disease, malum coxæ senile, as well as gouty affections and psoas abscesses. In all such instances the immobility of the extremity, which also exists in a pure sciatica, makes the examination difficult, and only after persistent repetitions shall we be able to obtain a clear idea as to the true nature of the trouble. Although it may be going too far to say with Hutchinson that out of twenty cases diagnosticated as sciatica in nineteen there exists no trouble whatever in the nerve (Medical Times and Gazette, 1882, vol. i, No. 1648, page 35), there can be no question but that here many diagnostic sins are committed and that there are many cases called sciatica after a superficial exploration which later prove to be something entirely different.

Treatment.—The treatment of sciatica should vary according as the neuralgic pains constitute merely a symptom or result from an independent affection of the nerve itself. In the former case our therapeutic measures, of course, must be directed against the underlying disease (diabetes, tabes, syphilis, etc.). If we have to deal with sciatica as an affection by itself our treatment should be systematic and carried out on definite lines. Our first rule should be never, or at any rate only in exceptional instances, to withdraw blood. If there are old scybalous masses in the bowel which press upon the nerve and thus cause the pain, considerable and lasting improvement may be brought about by the removal of these, and a course at Carlsbad or Marienbad may cure sciatica in such cases

more quickly and surely than the most careful use of electrici-
ty.   Next, especially when we have grounds for suspecting
an inflammatory condition of the nerve, we should try the ap-
plication of counter-irritants to the skin, fly-blisters or the so-
called *points de feu* (with Paquelin's thermo-cautery).   The
former more particularly, which have been used by Cotugno
and Valleix, deserve to be recommended, as they prove gen-
erally very effectual if used early in the disease ; they may be
applied along the course of the nerve on the thigh or in the
sacral region (Anstie).   Less benefit is usually derived from
irritating inunctions and plasters, which may, however, be
given a trial ; for instance, we may employ one of Betz's plas-
ters—empl. oxycroc, 15.0 (℥ ss.) ; arg. nitr. pulv., 1.0 (grs. xv)—
allowing it to remain on the skin until it drops off of its own
accord.   Among other drugs for inunctions besides veratrine
[0.1 : 10 lard] the narcotics (preparations of opium, belladonna,
hyoscyamus) may be useful.   Finally, the chloride-of methyl
spray may be recommended.   This, however, should be used
with great care ; otherwise it may be followed by a cellulitis,
erysipelas, or even gangrene.   The desired effect does not al-
ways follow.

From internal medicines I have never seen any lasting good
results ; besides antipyrine and antifebrine, iodide of potassium
has been used from time immemorial, also quinine and all the
nervines.   Recently solanine has been recommended, fifteen to
twenty centigrammes (grs. ijss.–iijss.) a day.   In my own expe-
rience this drug does not possess much value ; neither does the
oil of turpentine given internally in capsules containing fifteen
minims ten or twelve times a day.   In short, I consider all in-
ternal medicines, unless the case be one of syphilis, as useless
and inadvisable, for, owing to the long duration of the trouble,
they would have to be taken for months with great detriment
to the stomach and to the digestion.   More is accomplished
by external measures—massage and electricity.   Both have the
disadvantage, however, that they act very slowly and that their
application causes more or less violent pain, a remark which
applies more particularly to a systematic and an energetic use
of massage (Schreiber and others).   The faradic brush and the
combined current used by De Watteville are also very painful,
but both can be recommended with a clear conscience.   With
regard to the best manner in which the electricity should be
given, as we have already said before we now repeat that every

one has his own method, in which he has most confidence because he is most familiar with it.

If we are forced to send our patients to the springs, we may first of all recommend non-medicated hot springs or hot brine springs. Among the former may be mentioned Gastein, Johannisbad, Teplitz-Schönau, and Wildbad; among the latter, Wiesbaden, Nauheim, Rehme, and Baden-Baden. At hot sulphur springs, for instance, Landeck, Teplitz-Trencsin, and Pistyán in Hungary, such patients do very well, but it is advisable not to raise their expectations too high, as often the results of a stay at the springs are not very conspicuous. Sea-bathing is not always borne well by patients with neuralgia. At any rate, it is well to begin with places on the Baltic and to select first those where warm sea-water baths can, if necessary, be also obtained—e. g., Colberg, Misdroy, Zoppot, and others. In severe cases, particularly if there occur transient attacks of intense pain, morphine can not be dispensed with. Subcutaneous injections in proper amounts and at the proper time will do the patient no harm, but will afford him unspeakable relief, such as can be expected from no other drug.

Far less frequently—we might say, only exceptionally—are the individual branches of the sciatic nerve the seat of neuralgia; thus we may have an affection of the plantar nerves, and sometimes the hyperæsthesia in their distribution may be so marked that the patient is absolutely prevented from standing or walking. Barbillon (cf. lit.) has devoted a careful study to this so-called plantar hyperæsthesia without, however, being able to decide whether the disorder is of spinal origin, or whether it has to be regarded as a so-called dermatalgia, or again as a disturbance in the nutrition of the fine nerve endings. The first explanation is supported by the fact that usually both feet are affected; the last that it often occurs in people who have to stand a good deal. It has often been known to occur as a sequela of typhoid fever. Cures are said to have been effected by blisters, or by the application of a spray of methyl chloride; bathing the feet for some time in hot salt solution has also been recommended.

The pudic nerve, which supplies the bladder, the rectum, the perinæum, and the external genitals, is often the seat of neuralgias which are sometimes purely cutaneous and show themselves by an extreme tenderness of the skin of the penis, the

scrotum, the region of the anus, and the mons Veneris. In many instances the testicle is affected and, as we have pointed out above, becomes very tender and the seat of violent paroxysmal pains. Although there may be intervals in which the neuralgia disappears, the tenderness and irritability remain as long as the disease of the nerves is present.

Other nervous affections of the male urinary apparatus have been studied by Oberländer (cf. lit.), who has called attention to the fact that varicocele, chronic gonorrhœa, hydrocele multilocularis, tuberculosis, carcinoma, etc., frequently give rise to such disorders, and indeed not only do the just-mentioned cutaneous forms occur, but also a peculiar neuralgia of the urethra, which becomes particularly distressing during coitus and micturition, is frequently known to develop under the influence of such affections. The remains of a gonorrhœa together with chronic dyspepsia may produce a chronic hyperæsthesia of the mucous membrane of the bladder, to which little attention has been paid as yet. The pain appears periodically, affects the whole bladder region, and radiates into the urethra and the ureters. Slight errors of diet may evoke violent exacerbations of the trouble. Neuralgia of the bladder is found in neurasthenia, but also at times in the initial stage of tabes; hence it would be necessary to decide, if we have diagnosticated a neuralgia of the bladder, whether it is due to a cystitis or a spinal disease, or whether, on the other hand, it constitutes an affection by itself.

Anæsthesia of the mucous membrane of the bladder and of the urethra as well as loss of the muscular sense of the bladder make it impossible for the patient to say with the eyes closed whether he is voiding urine or not. It may happen to tabetics, in whom the condition is not infrequently met with, that, having given up all attempts to micturate after unsuccessful straining, they pass their urine involuntarily and become only conscious of the fact when they feel the dampness of their clothes. This anæsthesia does not seem, however, to occur as an independent disease, but would appear to be always of central origin.

The motor disturbances affecting the muscles which expel the urine and those which close the bladder may be of an irritative or a paralytic nature, the former constituting what is known as strangury; the latter are by the laity comprehended under the name of " weakness of the bladder." Both may be symptoms of chronic inflammation of the urethra or of certain

spinal diseases, and may also occur independently, as purely nervous affections. The desire to urinate every few minutes, a desire which is increased after drinking alcoholic beverages, is not infrequently alternated by spastic conditions of the muscles of the bulb which give rise during micturition to spasmodic excruciating pains in the perinæum which radiate to the thighs and the buttocks.

In all cases of this kind the treatment is generally begun with the usual anti-neuralgic remedies, of late years also with cocaine. However, the result is often very unsatisfactory. We should always carefully search for possible underlying abnormities, such as an elongated adherent preputium, insufficiently dilated or tight strictures, flexion or version of the uterus, or pathological changes in the rectum. If such be found the neuralgia is to be regarded as a reflex neurosis and we have to direct our therapeutic efforts to the primary cause, by which procedure we may be able to improve and eventually cure the neuralgia. To the same class of reflex neuroses belongs the enuresis nocturna, which is rather common among children. The trouble can usually be traced to irritation in the urethra or at the orifice, such as inflammatory conditions, slight adhesions of the mucous membrane far back in the urethra, too narrow an orifice of the urethra, and the like. Here, of course, attention to such primary disorders is the first step in our treatment, and dilatation of the posterior portions of the urethra with dilators made for the purpose will often be followed by striking results (Oberländer, Berliner klinische Wochenschrift, 1888, 31).

By coccygodinia we mean a neuralgia which is characterized by pain over the region of the coccyx. The affection is more frequently met with in women than in men, and the pain, which shows paroxysmal exacerbations and comes on more particularly during the act of defecation, may attain to a frightful pitch. The causes of the affection are obscure, yet we are probably not far from being correct in assuming that in many cases it is of reflex origin, as in men especially treatment of the genitals—a diminution of an abnormal sensitiveness of the pars prostatica ureth., etc.—may be followed by surprising results. In some cases the pains appear during sleep without any appreciable cause, in others they have been known to occur after traumatism. I have repeatedly observed them in neurasthenics and hysterical patients. The excision of the coccyx, an opera-

tion which in desperate cases has been undertaken for relief of the pain, should, of course, not be resorted to until all other means, particularly energetic application of the faradic brush, have been thoroughly tried.

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As to the motor disturbances—and of these we will take up first the paralyses which occur in the distribution of the sacral plexus—here, too, those of the sciatic nerve are the most important. Such lesions may affect the nerve high up in the pelvis, or soon after its exit from it, or still lower down in its branches. The first are almost always caused by traumatism or pressure exerted for a comparatively long time—e. g., by a pregnant uterus, the child's head during labor (Vinay), tumors, etc. The others often constitute a symptom of some other disease.

External popliteal (peroneal) paralysis, in which the muscles of the anterior surface of the leg are affected (the extensors

of the toes, the tibialis anticus, and the peronei), is easily recognized. The foot hangs down flaccidly, it can neither be
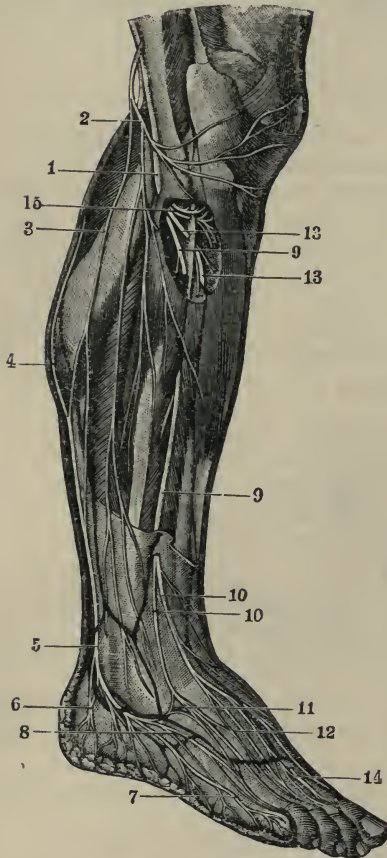
Fig. 127.

Fig. 128.

Fig. 127.—CUTANEOUS NERVES OF THE LEG AND FOOT. (After HIRSCHFELD and LEVEILLÉ.) 1 and 2, external popliteal nerve and its cutaneous branch. 3, peroneal communicating branch which unites with 4, the tibial communicating, in 5, the external saphenous nerve. 6, calcaneal branch of the external saphenous nerve. 7, external dorsal digital branch to the fifth toe. 8, dorsal digital branch to the fourth and fifth toes. 9–12, musculo-cutaneous nerve. 13, anterior tibial nerve. 14, its inner terminal branch between the first and second toes. 15, recurrent articular nerve.
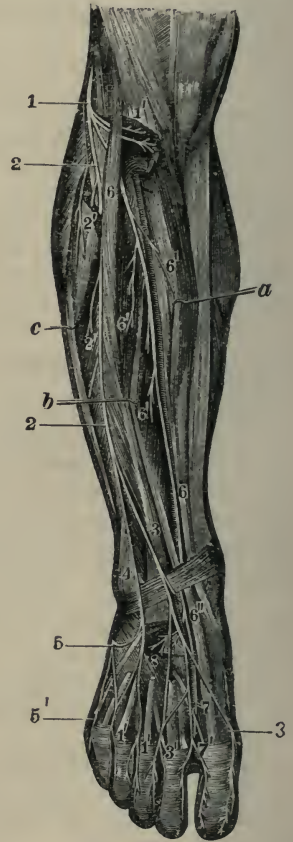
Fig. 128.—DISTRIBUTION OF THE BRANCHES OF THE EXTERNAL POPLITEAL NERVE. (After HIRSCHFELD and LEVEILLÉ.) *a*, tibialis anticus muscle. *b*, long extensor of great toe. *c*, peroneus longus. 1 and 1′, external popliteal nerve. 2 and 2′, musculo-cutaneous nerve. 3, 3′, internal branch of musculo-cutaneous nerve. 4, its external branch. 5, external saphenous nerve. 5′, its branch to outer side of fifth toe. 6, 6′, 6″–8, anterior tibial nerve.

dorsally flexed, abducted, nor adducted. As a result of this, walking is very much impaired, since the point of the foot

often trips over prominences on the floor, but by raising the thigh higher than usual the patient somewhat overcomes the difficulty. As the point of the foot or the outer margin is first put to the ground in an awkward manner, the gait is very peculiar and highly characteristic of this form of paralysis. Contractures of the calf muscles, which may later develop sec-

N. cruralis
M. tensor vaginæ femoris

N. obturator
M. pectineus
M. adductor magnus
M. adductor longus

M. quadriceps femoris
M. rectus femoris
M. cruræus
M. vastus externus
M. vastus internus

N. poplitealis externus
M. tibialis anticus
M. extensor longus digitorum
M. peroneus longus
M. peroneus brevis
M. extensor brevis pollicis

M. gastrocnemius
M. soleus

M. flexor longus pollicis
M. abductor minimi digiti

M. extensor brevis digitorum

M. interossei dorsales

Fig. 129.—MOTOR POINTS FOR THE NERVES AND MUSCLES OF THE ANTERIOR SURFACE OF THE LEG.

ondarily, give rise to a permanent position of talipes equinus or talipes equino-varus. External popliteal paralysis may be brought on by the occupation of the patient. It has been seen as the result of pressure in those who, from the nature of their work, have to be constantly in a kneeling position, as, for instance, asphalt pavers (Bernhardt).

Internal popliteal paralysis, which concerns the muscles of the back of the lower leg (the flexors, the tibialis posticus) and the muscles of the soles of the foot (adductor and abductor hallucis and the interossei), interferes with the plantar flexion of the foot and with flexion and lateral motion of the toes. As a result the patients are unable to stand on tiptoe. If the interossei take part, a condition is developed similar to that
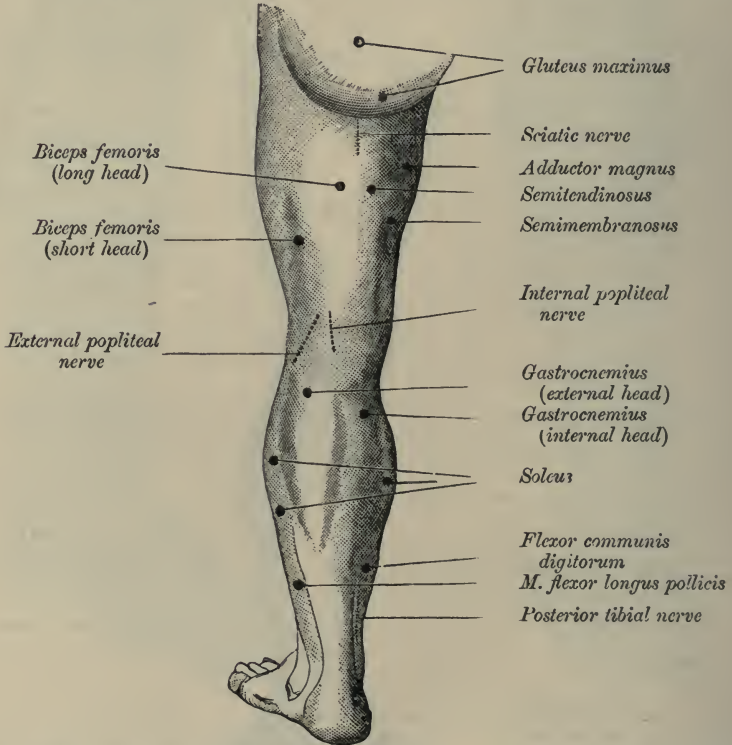
Fig. 130.—MOTOR POINTS FOR THE SCIATIC NERVE AND THE MUSCLES SUPPLIED BY IT.

which is seen in the hand and which we have described on page 348. The toes assume a claw-like position owing to the fact that the first phalanx is dorsally flexed while the second and third are in plantar flexion. Here also secondary contractures may appear (of the tibialis anticus, triceps suræ), which give rise to a paralytic clubfoot (pes planus, pes equinus, pes calcaneus).

Paralysis of the whole sciatic, which also, as we know, supplies the flexors of the lower leg (the semitendinosus, the semi-

membranosus, and biceps femoris), makes it impossible for the patient to flex the lower leg on the thigh, to approach the heel to the buttock, and to rotate the thigh (M. obturator internus). Paralysis of one sciatic alone does not make walking absolutely impossible, because the leg fixed in the knee joint is
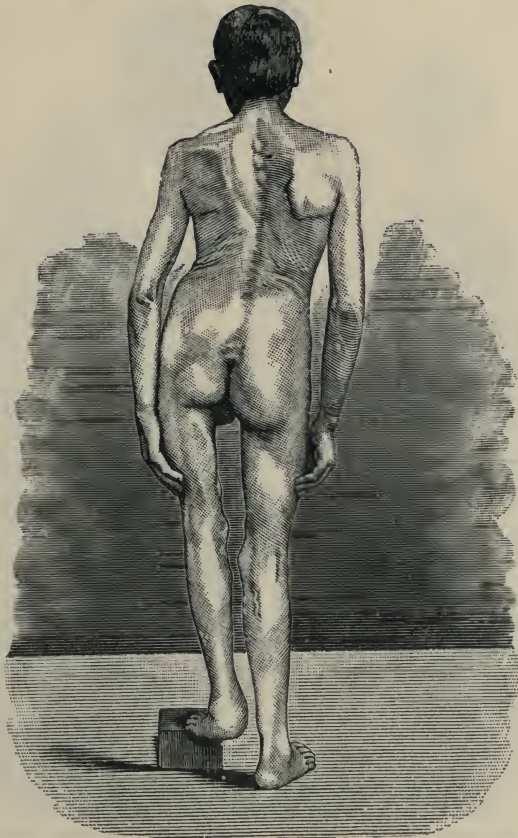
Fig. 131.—Case of Peripheral Neuritis of the Sciatic Nerve with Shortening and Atrophy of the Affected Extremity (personal observation).

moved forward by the muscles of the thigh, and so is used as a stilt (cf. page 228, gait of the hemiplegic). After a certain time muscular atrophies begin to be noticeable, and later become very marked. The affection of the hip joint, which sometimes develops in the course of the paralysis, but which also at times has to be looked upon as the forerunner or immediate cause of the paralysis, produces more or less marked shortening, so that the patient with his affected lower extremity presents a picture

like one of those shown in Figs. 131 and 132. This peripheral
affection of the sciatic, which has to be regarded as a neuritis
in the sense described above on page 328, can hardly be mis-
taken for anything else. The difficulty in moving one leg,
which may amount to an actual paralysis, may, it is true, also
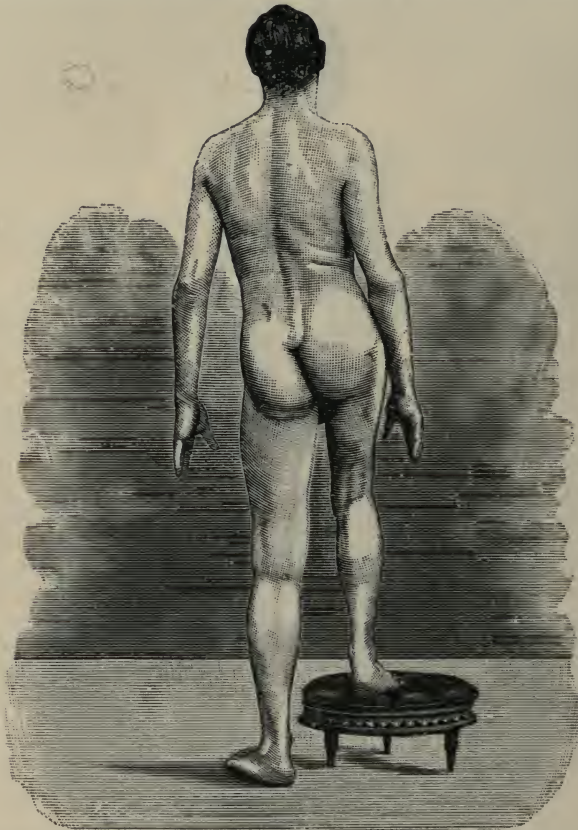
Fig. 132.—Case of Peripheral Neuritis of the Sciatic Nerve with Shortening
and Atrophy of the Affected Extremity (personal observation).

be the consequence of a central cortical affection—a mono-
plegia or monoparesis. In this case, however, the pains are
by no means a prominent symptom, nor do we find—and this
is the most important point of distinction—either atrophy or
shortening. The differential diagnosis between cortical and
peripheral paralysis has been spoken of on page 183. The
treatment of the affection is to be conducted according to the
principles which we have discussed in speaking of other periph-
eral paralyses.

The observations which some years ago were published by Westphal about a periodically recurring paralysis of all four extremities have as yet no practical importance, since we do not know anything about its nature. The same may be said about the peculiar paresis of the lower leg and foot which Zenker has described (Berliner klin-

Fig. 133.   Fig. 134.

Figs. 133–134.—CONTRACTURE IN THE QUADRATUS LUMBORUM (personal observation).

ischer Wochenschrift, October 8, 1883), and which has to be regarded as an occupation neurosis. It occurs not rarely in persons who have to remain a long time in a kneeling or squatting position, and such instances have been known to occur in potato pickers. It manifests itself in a more or less pronounced sensory or motor paralysis of the lower part of one or both lower extremities.

Spasms of the nerves of the sacral plexus may be tonic or clonic in character. They are, however, rare, and since treat-

ment can do but little against them they are not of much prac-
tical interest.

Spasmodic tonic contraction of the hip muscles has been
described by Stromeyer as spastic contraction of the hip. A
case of spasm confined to the quadratus lumborum has come
under my notice in an hysterical woman. It is illustrated in
Figs. 133 and 134. Tonic spasm of the quadriceps gives rise to
extension of the leg in the knee joint; it is sometimes known
to occur in neuralgias of the joint. The very painful cramp in
the calf muscles, which sometimes occurs after great exertion,
sometimes also in the course of certain grave general diseases
—for example, cholera—is well known.

Clonic spasms of the muscles of the lower extremities may
be observed in hysterical patients. The so-called " saltatory
spasm " (Bamberger, Wiener medicinische Wochenschrift, May
4, 1859), which forces the patients whenever their feet touch
the ground to jump, is not an independent affection, but only a
symptom of central disease. The increase of the reflexes,
which is generally present, is in favor of this view. Of the
treatment we shall speak in the chapter on hysteria.

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V. Neuritis involving Several Spinal Nerves at the Same Time—
Multiple Neuritis—Polyneuritis.

Just as we have seen that several of the cranial nerves can be affected at the same time, so none the less is this true of the spinal nerves. It is, however, not many years since it has been shown that such multiple nerve affections may occur primarily, that they are often of an inflammatory nature, that they give rise to numerous symptoms which may, under certain circumstances, be misinterpreted, inasmuch as they may simulate those of central lesions. The affection is known as multiple neuritis, and, as we said, our knowledge of it is of quite recent date (Duménil, Eisenlohr, Leyden, Strümpell, Vierordt, and others). We may confidently expect that in the near future we shall obtain further information upon certain points in connection with this disease which have not as yet been cleared up.

As we have above, on page 328, devoted some time to the description of the anatomical features of the disease, it remains for us here to speak first of the symptoms of multiple neuritis. It is remarkable to note that the onset frequently resembles that of an acute infectious disease: there·is fever, general malaise, dull headache, apathy, etc.; soon pains make their appearance, first in the lumbar region and the back, then in the course of the large nerve trunks. These are followed by an impairment of mobility, especially in the lower extremities, which makes the patient very anxious; the legs are heavy, they are moved only by a strong effort, and not without pain, and the patient is easily fatigued. The reflexes are diminished or lost, electrical excitability is decreased, but the pains—and this should be emphasized—usually soon abate and other sensory disturbances, paræsthesias and anæsthesias, are only exceptionally met with, the disorder chiefly affecting the motor apparatus. Repeatedly cases have been observed in which the motor disturbances made their appearance quite suddenly, an onset which we could almost call apoplectiform. Without any premonitory symptoms there come on violent radiating pains, with motor paralysis and trophic changes. Muscular atrophy and reaction of degeneration can be demonstrated. The muscular atrophy sometimes shows itself early and attains a high grade, sometimes not until late, and may be so slight that it almost escapes notice. In many in-

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stances a certain amount of ataxia is observed, and this symptom may indeed be very marked, so that it dominates the whole picture and makes it resemble that of tabes. In such cases the term pseudo-tabes peripherica, instead of simply multiple neuritis or polyneuritis, is very appropriate.

No description of the course of the disease which would fit all cases is possible, because this varies and presents peculiarities according to the pathogenesis. For example, if a multiple neuritis occurs in the course of another disease, its manifestations are not the same as when it is a primary affection, which has developed under the influence of some special cause. Among the conditions in which polyneuritis may develop we would mention phthisis pulmonalis, diabetes, tabes, articular and muscular rheumatism, polyarthritis, and finally the puerperal state. It has repeatedly been described as a sequela of typhoid fever, of small-pox, scarlet fever, and of diphtheria (the "infectious form" of Leyden). As an independent disease it may be caused by overexertion. Two cases which we have described were due to prolonged work with the sewing machine (cf. lit.). It may also appear, and this is unquestionably much more common, as a consequence of the action of certain poisons, more especially alcohol, carbon monoxide, bisulphide of carbon, lead, arsenic, and mercury (the "toxic form" of Leyden). Besides these two there is, according to Leyden, a third variety, the so-called atrophic (anæmic, cachectic) form, which develops after a long and severe sickness, somewhat in the manner recently described by Oppenheim and Siemerling.

Sometimes sensory, sometimes motor disturbances are the predominating symptoms. In the neuritis of phthisical patients both are marked to about the same extent. Occasionally certain nerves seem to be more liable to suffer—for instance, according to Möbius, during the puerperal state, the median and ulnar, the terminal branches of which are affected either in both hands or only in the one which is used more extensively, as a rule the right. In tabes, on the other hand, no region seems to be exempt, and, as Oppenheim, Siemerling, Pitres, Vaillard, and others have observed, not only the peripheral spinal, but also the cranial nerves may be attacked by the neuritis—for example, the vagus and its laryngeal branches, and the ocular nerves.

The neuritis which occurs in the course of joint affections often leads to considerable atrophy in those muscles which are

supplied by the affected nerve twigs. Chronic inflammation of the synovial membranes caused by sprains, chronic inflammations of joints, articular rheumatism, frequent attacks of gout, fractures which give rise to some impediment in circulation— all these causes may bring about extensive muscular atrophies. References bearing on these affections and upon "reflex atrophies," which we shall soon mention, will be found on page 393. The case which we have illustrated in Figs. 135 and 136 was that of a young man who suffered from a panarthritis, and who in consequence of his joint affection developed muscular atrophy in all four extremities, more especially in the upper arms and thighs. The hip and shoulder joints, as well as the knee and elbow joints, had been swollen and painful for years. That this atrophy, which may be due to an inflammation of the fine end twigs of the nerves, may also be caused reflexly by the joint affection has been shown by Charcot. If the hip joint is attacked, the flattening of the buttock, the abnormally high position of the gluteal fold, the marked prominence of the trochanter on that side, are striking features. If the upper extremities, especially the hands, are the seat of the disturbance, the atrophy gives rise to deformities which are either of the extensor or the flexor type (Charcot).

Peculiar and manifold are the manifestations of that variety of neuritis which is produced by the abuse of alcohol. For the sake of simplicity we may distinguish two cardinal forms of this affection, although the clinical pictures of the two can often not be well separated from each other. In the first the motor disturbances and the atrophies, in the second the sensory disorders, are the prominent symptoms. In the former case the patients complain of violent tearing and drawing pains in the lower, more rarely in the upper extremities, which are relatively rapidly followed by a marked difficulty in walking. The gait of the patient is distinctly ataxic and resembles most closely that of a tabetic, with the exception that in the latter no diminution in the strength of the muscles can be noted, while in alcoholic neuritis it can undoubtedly be demonstrated and is to be explained by the muscular atrophy which occurs comparatively early and which is particularly seen in the extensors. The degree to which walking in particular and motion in general is interfered with is very variable. Sometimes the patient can hardly raise himself in bed without assistance, sometimes he may for months be able to get about fairly well

without help.   It is interesting to note that the patellar reflex
is lost very early and completely, a circumstance which may
lure not the inexperienced alone into making a diagnosis of
tabes dorsalis.   This is still more likely to occur, and the mis-
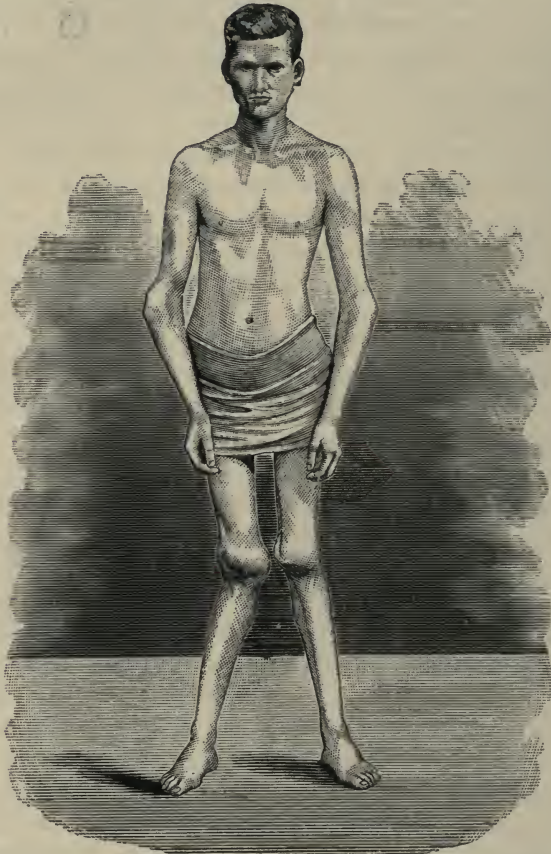
Fig. 135.—Panarthritis with Secondary Multiple Neuritis.

take is more excusable, if the action of the alcohol has also
manifested itself on the ocular nerves, so that, e. g., we may, in
addition to the symptoms mentioned, encounter a paralysis
of the abducens, which I have myself seen several times in
alcoholic neuritis, and which Suckling (cf. lit.) and others have
described ; or, again, the oculo-motor may be implicated and the
patient may complain of diplopia.   Vagus neuroses have been
reported in this connection, especially tachycardia, by Déjerine.
If we add to this the frequency with which Romberg's sign

(swaying while standing with the heels and toes together and eyes closed, in consequence of the disturbance of the muscular sense) is found in the disease, if we remember that stomach symptoms occur in both affections—in alcoholism as vomitus

Fig. 136.—Panarthritis with Secondary Multiple Neuritis.

matutinus in consequence of a chronic gastritis, in tabes as gastric crises in consequence of disease of the vagus nucleus—we can not be surprised at the frequency with which alcoholic paralysis is taken for tabes. Nevertheless it is not so difficult to avoid such a mistake, more especially if we have a chance to examine the patient repeatedly and do it carefully enough. We should particularly note the condition of the pupils. The absence of the Argyll-Robertson sign and the absence of bladder symptoms, both of which are very common in tabes,

will be significant features. In alcoholic neuritis, further, the nerve trunks are usually painful and the course of the disease differs in the two maladies. In tabes, as we know, the outcome is very unfavorable, while in alcoholic neuritis, if the cause is removed, it is usually good. Even the individual symptoms may, if analyzed carefully, give us some valuable diagnostic hints. For example, it will hardly be very difficult for the careful examiner to distinguish the morning vomiting of alcoholics from the paroxysmal spontaneous vomiting of tabes, which appears now and again and may not reappear for months.

The second form of alcoholic neuritis may run its course without giving rise to any decided motor disturbances. The patient then only complains of pains which sometimes run along the nerve trunks, becoming very violent, and may resemble the lancinating pains of tabes. He may complain of localized hyperæsthesias and anæsthesias, of formication and numbness, all of which symptoms are especially marked in the lower extremities. Various vaso-motor and trophic disturbances are not uncommon. Œdema may occur and disappear again, skin eruptions, circumscribed areas of hyperidrosis, the falling out of the nails and hair, may be noted. In all cases the psychical condition ought not to be neglected. It may present changes very early in the disease. Thus Oppenheim has reported instances in which the alcoholic neuritis occurred simultaneously with delirium tremens.

About the toxic form of neuritis, which is not uncommonly found as a consequence of the occupation, we shall speak in the chapter on intoxications.

It has long been known that neuritis may be produced artificially, and that it, for example, often occurs as a consequence of subcutaneous injections of ether; but this has only been carefully studied of late years. Cases of this kind impress upon us the necessity of being cautious in giving the injections for therapeutic purposes and of avoiding especially a too deep insertion of the needle where we should be liable to strike branches of the musculo-spiral or other nerves. Paralysis of the extensors of the fingers has been relatively often observed. References bearing upon this subject will be found on page

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B. Diseases of the Trophic and Vaso-motor Nerves.

In spite of the epoch-making labors of Samuel (cf. lit.), who, after Romberg, was the first to postulate the existence of definite " trophic " nerve fibres for the regulation of the nutrition of the tissues, we are to-day still unable to demonstrate such fibres, nor do we know whether there exist purely trophic centres, or whether the trophic influence is exerted by some centres already well known—viz., by the motor, sensory, or vaso-motor. On the other hand, the existence of such a direct trophic influence of the nervous system upon the tissues can not be called in question. Again, we can not as yet decide whether or not this influence, upon which the nutrition of the tissues normally depends, is different for different tissues—as indeed seems quite probable—and whether it has to be regarded as acting by inhibition or rather by an active stimulation.

Occasionally, besides the trophic we meet also with vaso-motor disturbances, which lead us to conclude that not only the vaso-constrictor, but also the less important vaso-dilator nerves may become affected. With their anatomical relations we are better acquainted than with those of the trophic nerves. At any rate we know that in the cerebrum and in the medulla oblongata vaso-motor centres do exist. In their further course the vaso-motor fibres are thought to pass through the lateral columns of the spinal cord and to leave it through the anterior roots, but this is not yet proven, nor can we accept the exist-ence of Goltz' vaso-motor reflex centres in the spinal cord without further investigation.

The vaso-motor disturbances which we sometimes see in acute diseases—for instance, in typhoid fever (Money, Lancet, December 3, 1887)—are phenomena due either to a condition of paralysis or of irritation. In the former case we have red-dening of the skin with elevation of the temperature, a con-dition which may also appear either independently or in the course of functional neuroses. In the latter we have marked pallor and coldness of the skin, associated with formication, subjective sensations of cold, as, for example, in the so-called anæsthesia lavatricum (cf. Hirt, Krankheiten der Arbeiter, 1878, Part II, p. 100). Such disorders need, however, not always lead to trophic changes. It is rather probable, as Kopp thinks, that, if the latter occur, disturbances in the nu-trition of the vessel walls must have preceded them (cf. the work of Thoma, lit.).

Sometimes vaso-motor and trophic disturbances may coex-ist as genuine complications.

Of clinical importance is the fact that trophic changes may occur either by themselves or accompany other, central, cere-bral as well as spinal affections. This may be explained by the fact that in order to bring about alterations in the trophic influ-ences, it is not necessary to have a disease of the ganglionic cells or cell groups, which probably act as centres, but that pathological processes in the peripheral nerves may also have the same effect. Among the central affections, which, how-ever, may remain latent for a long time, so that one might be led to regard the trophic changes as independent affections, we must mention in the first place tabes, which we shall discuss in this connection later, and after this certain cerebral diseases due to changes in the vessels, such as apoplexy with the acute

bedsore, of which we have spoken on page 233, and again diseases of the gray axis of the spinal cord (Jarisch), among others the " paralysie générale spinale antérieure subaiguë " (Pitres et Vaillard, Prog. méd., 1888, 35). To the diseases of the peripheral nerves and the infectious diseases, in the course of which trophic disturbances may occur, we have already alluded.

At present we can form no idea how many diseases, not only of the nerves and of the muscles, but also of other organs, we shall have to call "trophic" when we have once become better acquainted with the position of the trophic centres and fibres than we are now. For the present the term is restricted to a small number of affections, and it will suffice to say a few words about the most important among them, and first about the tropho-neuroses of the skin.

Anomalies of secretion which have to do with the sebaceous as well as the sweat glands are not uncommon. It is well known that seborrhœa, for example, may occur after longstanding menstrual disturbances, chlorosis, anæmia, after overexertion, or as a consequence of too great sexual excitement, masturbation, etc., especially in young individuals, whereas diminished secretion of the sebaceous glands, as found, for instance, in ichthyosis and in senile atrophy of the skin, is comparatively rare. The purely nervous origin of this, as well as of hyperidrosis and anidrosis, can hardly be questioned. Hyperidrosis is seen on one side alone or on both sides in central diseases—for instance, in some diseases of the medulla oblongata (Traube), of the spinal cord (spinal apoplexy, myelitis), and of the entire nervous system (tabes, hysteria). It also occurs reflexly (Raymond). The anidrosis appears in peripheral facial paralysis, in dementia paralytica, and in certain skin affections, such as psoriasis, lichen, and ichthyosis.

Among the skin affections associated with exudation we have erythema nodosum, urticaria, and a disease probably akin to it, the angio-neurotic œdema (Quincke), which appears sometimes quite suddenly on different parts of the body, the patient feeling otherwise perfectly well. Accompanying the menses cutaneous swellings of nervous origin have been described (E. Boerner, Volkmann'sche Sammlung klinischer Vorträge, 1888, xi, No. 312). Again, we have certain forms of eczema, prurigo, herpes zoster, and others, although the nervous origin of them is not established beyond doubt. As every one of these affections presents in its development, in its clinical significance and

in its treatment, so much that is by no means clear we deem
ourselves hardly called upon to enter into a detailed description
of them here.   Some, as, for instance, the herpes zoster in the
course of facial paralysis, have been mentioned above (cf. page
89).   Equally obscure is the origin of cutaneous hæmorrhages
—we only wish to recall the ecchymoses which occur in tabes
after severe attacks of pain—of the pigment hypertrophies
(e. g., in lepra), of the anomalies of cornification (keratosis and
ichthyosis), of the nævus, which is said to be due to intra-
uterine disease of the spinal ganglia, of the atrophic conditions
of the skin (striæ and maculæ atrophicæ), of the so-called glossy
skin (glossy fingers), of the pigment atrophies (vitiligo), of the
atrophy of the hair, and the atrophies or deformities of the
nails, changes which we meet with in the most varied nervous
affections and under the most varied circumstances.

An interesting angio-neurosis is the so-called night palsy,
which has been described by Ormerod, Bernhardt, and others.
It consists in numbness, pain, and a feeling of weakness occur-
ring at night in the upper extremities.   Distinct anæsthesia and
actual paralysis are not present.   Women are affected more
frequently than men, and seem to be particularly prone to it at
the menopause.

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The so-called symmetrical gangrene of the fingers and toes
(sclerodactyly) which was first described in 1882 by Raynaud,

and which has, after him, been called Raynaud's disease, comes on with the following symptoms: The fingers appear at times as if dead ("*doigts de mort*"), at another time they turn a dark-red color and burn violently. Gradually disturbances in nutrition, at first only transitory, later permanent, develop and blebs form, which open, leaving a sore, which heals with loss of substance. The nails fall out and are not replaced, whole parts die, the necrosis being symmetrical on both sides, and none of the usual causes of gangrene—such as disease of the heart or of the blood-vessels, septicæmia, traumatism, etc.—are present. The disease is, however, very rarely met with in its full development, while lighter grades, in which we have only to deal with a transient spasm (or paralysis) of the vessels, especially in the hand, are not uncommon. In such instances the hands become bluish and icy cold, and we have a condition known as local asphyxia. Raynaud's disease may be confounded with peripheral neuritis, ergotism, diabetes, and senile gangrene. It should, however, not be difficult to avoid such a mistake if we take into consideration the characteristic course of the disease and the absence of any of the ætiological factors before mentioned. In the treatment repeatedly, favorable results have been obtained by bathing the hands in warm water and the application of alcoholic menthol solution with a camel's-hair brush.

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Undoubtedly a close relation exists between Raynaud's disease and scleroderma.   In this latter very rare affection, which also depends upon trophic disturbances, the skin, after having presented œdematous swellings in the first stage, becomes later hard and immovable, so that it is impossible to pick up a fold of it between the fingers.   The affected parts, more particularly the face, neck, and the upper portion of the chest, where frequently a diffuse increase in the pigment is noticeable, are impeded in their movements, the play of the features is lost, the mouth can not be completely opened, the eyes can not be closed, and rotation of the head becomes impossible, etc.   The patient feels a sensation of discomfort; the coldness of the skin, which reminds one of that of a corpse, is most distressing, and a slight fall in the outside temperature is sufficient to bring about cyanosis.   Quite gradually the atrophic, the terminal stage comes on, in which the skin gets as thin as paper, remaining, however, firmly fastened to the underlying tissues, so that it is still impossible to pick up a fold.   With these changes is associated an atrophy of the muscles, which has to be regarded partly as a tropho-neurosis, partly as an atrophy due to inactivity, and the patient becomes helpless and unfit for work. After the disease has lasted for several years, if convalescence has not set in in the second stage, a general marasmus develops which leads to a fatal issue.   An effectual treatment is not known.   Warm baths, simple ointments, the constant current, internally tonics, iron, cod-liver oil, etc., may be tried, but we are not justified in placing any confidence in them.

Very closely related to scleroderma is a disease which fifteen years ago was for the first time described by the Englishmen Gull and Ord under the name of myxœdema, and later by Charcot under the name of *cachexie pachydermique*, an affection which only in quite recent years has attracted attention in Germany.

The beginning of the disease shows itself by a peculiar swelling of the whole face, the skin, especially of the eyelids and the cheeks, appearing œdematous.   The lips are not completely closed and the saliva dribbles from the corners of the mouth.   The face becomes, owing to the stumpy thick nose

and half-opened eyes, somewhat uncouth and common, then expressionless and cretin-like (cf. Fig. 137). The patients, especially if they are females, grow to look so much alike that they appear as if they all belonged to the same family. The color of the face is pale, the skin is wax-like, but does not pit on pressure. The œdema of the rest of the body has the same character as that of the face. The skin of the neck forms folds, the hands are thickened. On the hard wrinkled skin circumscribed thickenings can be seen, hair and nails fall out, the

Fig. 137.—CASE OF MYXŒDEMA. (After CHARCOT.)

teeth become carious, the secretions diminish and dry up. Amenorrhœa is common. Lungs, heart, and the large vessels present no abnormities. On examination the urine is found to be negative, while the temperature is subnormal. Among the concomitant symptoms must be mentioned sensory and motor disturbances, uncertainty in the gait, and general lassitude. Such patients get easily fatigued, their mental faculties suffer, and a condition of beginning imbecility develops.

The pathogenesis and the anatomical seat of the disease are quite obscure. It is generally accepted that the swelling is not

an œdema, but is due to the formation of a myxomatous new growth containing mucin. Mucin has been found in great quantities in the skin, the connective tissue, the blood, and the saliva of these patients. We seem to be justified in assuming that the thyroid gland has a good deal to do with myxœdema, since in most instances after the extirpation of this gland symptoms of myxœdema appear, and where this was not the case it is possible that either accessory thyroid glands existed, or that the removal was not complete. The operations on men and monkeys (Horsley) which have so far been performed seem to give no support to the idea that the true cause of this is to be sought for in injury of the trachea, of the recurrent laryngeal nerve, or of the cervical sympathetic. In the cases in which operative measures are not concerned we have to deal with a degeneration of the thyroid gland, the normal glandular tissue becoming replaced by a delicate fibrous tissue. The cause of such a degeneration is entirely obscure. The few instances in which just before the onset the patient had gotten a thorough drenching in the course of his daily work afford no sufficient grounds for looking upon this as an ætiological factor.

The researches recently published by Munk (cf. lit.) indicate that after all we are justified in being doubtful whether the symptoms appearing after extirpation of the thyroid are actually due to the loss of the gland, or whether they have to be regarded as a consequence of the operation itself. According to him, it is not as yet settled that the thyroid gland itself possesses important vital functions which are of great significance to the central nervous system.

We are not likely to mistake the affection for any other; still attention has been called to the fact (Lassar) that certain chronic erysipelatoid swellings may give to the face a similar expression to that seen in myxœdema. Among those affected with the disease observed up to the present time the majority were women and middle-aged. We are not yet acquainted with any treatment, nor has any been tried so far as we know.

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The next affection to which we shall call attention is as remarkable as it is rare. According to our present ideas, it has also to be ranked among the tropho-neuroses. We are referring to a very gradually developing atrophy of the face (sometimes ushered in by pain and paræsthesias), which may appear on one or both sides, and generally embraces equally the skin, the subcutaneous tissue, the muscles, and the bones. The beginning is usually as follows: Whitish spots appear on the skin of the face, which sink in more and more and are accompanied by a diminution of the fatty tissue below; gradually the atrophy increases in extent, and nothing escapes with the exception of the musculature, and this only occasionally and for a certain time. The affected side is sunken in and the skin assumes a whitish-brown discoloration. The bones, especially the upper jaw, and with it the teeth, atrophy; the latter fall out as well as the hair, which often appears of a light color or distinctly gray. The bone atrophy is the more marked the younger the patient at the onset of the disease (Virchow). If the disease is confined to one side only—hemiatrophia facialis—the median line forms a sharply defined border and the diagnosis is very plain. If both sides are affected, as happened in Eulenburg's case after measles (Lehrb. der Nervenkrankh., 1878, ii, p. 620), it may be more difficult to recognize the affection. The grooves and furrows which are found in the face greatly disfigure it (Fig. 138). The corresponding half of the tongue becomes small and often presents gap-like retractions, such as we described in hemiatrophy of the tongue (Fig. 119). Among the

muscles, not only those supplied by the facial, but also those supplied by the trigeminus, are implicated. The extension of the atrophic process to the neighboring shoulder and even to the upper arm is not unusual. Sensibility is not altered in the affected region.

Most of the few cases observed clinically have never come to autopsy. Of great interest, therefore, was the result of an examination, which Mendel was enabled to relate before the Berlin Medical Society, on such a patient who had died of

Fig. 138.—HEMIATROPHIA FACIALIS (personal observation).

phthisis, and who had previously been examined by Romberg and later on by Virchow (Deutscher Med.-Ztg., 1888, xxxiii, p. 407). On examining the origin of the trigeminus all the other roots were found to be normal; only in the descending root could atrophic changes be recognized, a fact which would indicate that the trophic fibres must be contained therein.

Other observations (Ruhemann) also point to a very intimate connection between facial atrophy and the trigeminus; however, more post-mortem observations confirmatory of those of Mendel are needed to clear up the pathology of the disease.

Of the ætiology little that is positive is known. Age and sex seem to have little influence. The disease has been known to occur at all times of life and also to be congenital; it has been observed in both sexes. According to Lewin, the frequency with which the two sexes are attacked is in the ratio of six males to nine females. Hereditary predisposition is certainly not a *conditio sine quâ non*, because there are cases—among others, the one I have reported myself—in which the patients belonged to quite healthy families. Sometimes it has happened that the atrophy was preceded by other nervous affections—trigeminal neuralgia, migraine, epilepsy, etc.—but this is by no means the rule. In a few cases the disease has followed injuries about the face or of the cervical sympathetic (Seeligmüller); more frequently no cause whatever could be demonstrated, and it was impossible to make any conjecture as to the ætiology. The outlook for recovery is absolutely bad, and therapeutics, so far as our knowledge goes, is powerless.

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Under the name of acromegalia (ἄκρον, extremity) Marie described, in 1886, a peculiar non-congenital hypertrophy of the hands, feet, and head, to which affection attention had been drawn before him by Fritsche and Klebs (cf. lit.). The extremities appear increased in length as well as in breadth. The bones of the face, especially those of the cheeks and the lower jaw, present considerable enlargement (cf. Figs. 139 and 140), and the measurements of the skull are above normal. In the same way the lips, ears, nose, and tongue are found enlarged, whereas all the muscles are feeble. The skin appears yellowish and pale, but is otherwise normal. The thyroid gland was almost always atrophic in the cases observed up to the present

time. In spite of their gigantic appearance the patients are feeble and without strength. The sexual functions are lost early and completely (Freund, cf. lit.).

The onset of the disease dates back to early childhood,

Fig. 139.—CASE OF ACROMEGALY. (After P. MARIE.)

and it has to be regarded as an abnormity in development (Freund) " which, probably beginning as early as the cutting of the second teeth, certainly sets in energetically at the period of puberty, and consists in a rapidly developing enlargement of the facial part of the skull, which by far exceeds the physiological limits of growth. This increase is especially marked in the lower jaw and also in the extremities, with their girdle attachments, while the rest of the skull and the trunk are only secondarily altered."

Only three cases have come to autopsy since Marie's work appeared. In one published by Fräntzel (cf. lit.), congenital smallness of the aorta, with atrophy of the right ventricle, was noted. The second is reported by Broca (cf. lit.), who in his account chiefly occupies himself with a description of the hypertrophy of the ends of the bones; and a third by Saundby (cf. lit.).

About the cause of acromegalia just as little is known as

about its treatment. According to Goldscheider, who estab-
lished the fact that the giant growth is not confined to the dis-
tribution of any one nerve—being found, for instance, in the
hand, in that of the musculo-spiral and median, more rarely

Fig. 140.—CASE OF ACROMEGALY. (After P. MARIE.)

in that of the ulnar—the ætiological influence of the trophic
nerve-fibres is still a matter of doubt.

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Appendix.—Diseases of the Muscles—Primary Myopathies.

Diseases of the muscles, which consist in alterations in their volume (which is generally diminished, less frequently increased), and consequent disturbances of function, may occur under the most varied conditions. They may be produced by cerebral affections, as we have pointed out on page 233, where we spoke of the possibility of an affection of trophic centres in the cortex ; they may be the result of spinal diseases, as we shall later find out in discussing syringomyelia and progressive muscular atrophy, but they may also—and this is what interests us here more especially—occur independently of any central disease as primary myopathies.

Considered from an anatomical standpoint, this disturbance in the nutrition of the muscles, the dystrophia muscularis progressiva of Erb, the myopathia progressiva primitiva of Charcot, consists either in a diminution, a wasting of the muscular tissue, owing to which the volume of the part affected becomes smaller ; or during the pathological process there may come about an increase in volume, which is either due to an actual increase and abnormal growth of the muscles—a genuine muscular hypertrophy—or to a growth of the interstitial fatty tissue, in which latter case we speak of a pseudo-hypertrophy of the muscles. Sometimes both conditions are found in one and the same individual, so that certain muscles appear atrophied, while others, in consequence of the simultaneous development of fat, appear strikingly large and hypertrophied.   The microscopical examination (Charcot, F. Schultze, Strümpell) shows considerable increase in the interstitial connective tissue between the individual fibres, which latter have retained their transverse striation.   The genuine hypertrophy which is seen in places must be regarded, according to Strümpell, as compensatory.

About the ætiology of primary myopathies we know very little.   It should, however, be mentioned that, according to all the observations made up to the present time, they belong entirely to early life, developing as they do before the twentieth

year. Heredity plays frequently a *rôle* in the disease, since not uncommonly several cases occur in the same family. Lesage has shown that they also may follow certain other diseases, as, for example, typhoid fever (cf. lit.). In such instances we have to deal with a secondary lipomatosis, developing in circumscribed areas of the body, as the result of certain arterial changes.

In our present state of knowledge we seem justified in assuming that these myopathies occur regularly in certain groups of muscles, so that different "types" can be distinguished, and that on the whole the upper half of the body, more particularly the upper extremities, are more often and more severely attacked by the pathological process than the lower parts, and more especially than the legs. It is important always to observe whether the face remains intact or not, as in the former case we are dealing with the hereditary muscular atrophy which Erb has described as the "juvenile form"; in the latter, with the form which Landouzy and Déjerine have described, and which has by them been called "myopathie atrophique progressive."

The so-called "juvenile muscular atrophy" which develops in early youth, more often in boys than in girls, attacks by preference the pectorales, the trapezius, the latissimus dorsi, the serratus magnus, the rhomboidei, the sacro-lumbalis, and the longissimus dorsi, while the majority of the muscles of the forearm, the sterno-cleido-mastoideus, the levator anguli scapulæ, the coraco-brachialis, the teretes, the deltoid, the supraspinatus and infraspinatus, remain, as a rule, intact. The small muscles of the hand, which in spinal atrophy become affected so early and in such a typical manner, are here not implicated (Fig. 141). It is hardly necessary to enter into a description of the disturbances of function which necessarily must result from disease of so many muscles. If we remember how much impaired are the movements of the arm, which can not be raised above the horizontal position, etc., we can understand the gravity of the child's affliction. If, as in the long course of the disease not uncommonly happens, the process extends to the lower half of the body, the glutei, the peronei, the quadriceps, and tibialis anticus become implicated and the patients at first walk with an uncertain gait, then waddle in a characteristic manner, and finally lose the use of their legs. The implication of the muscles supplied by the bulbar nerves, which has

been observed by Bernhardt (cf. lit.), of course has a very decisive influence upon the duration and course of the disease.

Fibrillary twitchings in the affected muscles, as we see it so commonly, we may say regularly, in the so-called progressive
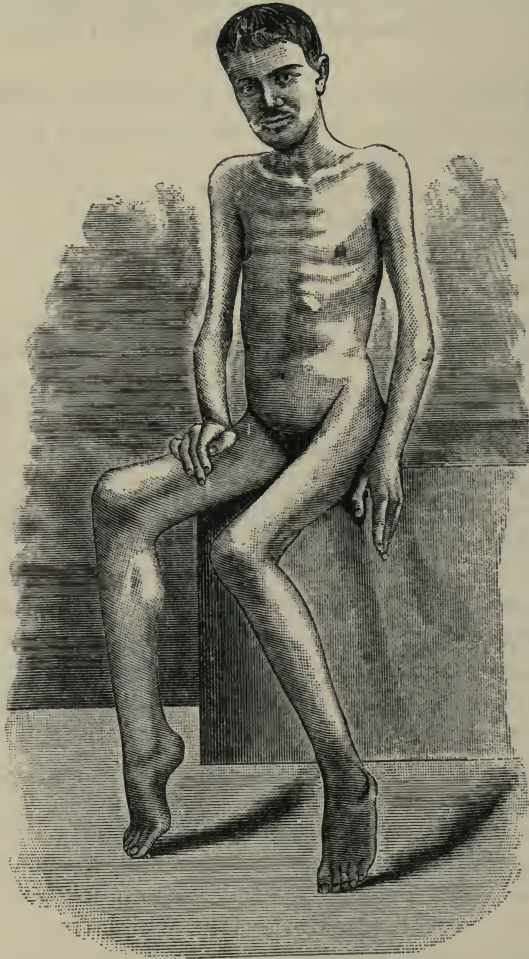
Fig. 141.—ERB'S JUVENILE MUSCULAR ATROPHY. (After MARIE and GUINON.)

muscular atrophy (spinal), is here wanting with the same regularity. Neither can any changes in the electrical excitability be demonstrated, with the exception, of course, of a diminution due to the disappearance of a more or less large number of muscle fibres. The course of the disease is, as we said, eminently chronic. It may extend over a space of twenty or thirty

years, since bulbar symptoms occur but rarely and we can
only look for a fatal issue if the process involves the diaphragm
and respiratory disturbances result. The diagnosis never pre-
sents any difficulties. After careful examination, taking into
account the distribution of the atrophy, the onset of the disease
in early life, with the fact that more than one member of the
family is affected, the long duration of the disease, the absence
of fibrillary twitchings, we can not mistake the myopathic for
the spinal form. The treatment must consist chiefly of good
care and nursing. All attempts to arrest the process by the
application of electricity or the use of massage, baths, or in-
ternal medicines, have proved to be of no avail.

The facio-humero-scapular type of muscular atrophy of
Landouzy and Déjerine, the "progressive atrophic myopathy,"
a form which had, however, already been described by Du-
chenne under the name of "progressive muscular atrophy of
infancy," begins in the muscles of the face, and before any other
symptoms make their appearance our attention is attracted by
the listless, sleepy expression of the face, the smooth forehead,
the faulty movements of the mouth, the inability to whistle and
to keep the lips together. At the same time there is a condi-
tion of lagophthalmus, so that the patient in spite of the great-
est exertion is unable to shut his eyes (cf. Fig. 142). Gradually
the muscles of the upper extremities and the trunk become af-
fected in almost the same distribution as in the juvenile paraly-
sis. The course is the same in both these forms. Here also
there are no fibrillary twitchings and no changes in the elec-
trical excitability, and although the pseudo-hypertrophy of the
muscles, which we shall presently describe, is not uncommon
in the juvenile form and is here never present, there is no ques-
tion but that the two affections are identical and that only in
some cases, from reasons not as yet understood, the interstitial
connective tissue becomes early increased, while in others
nothing else can be demonstrated but simple atrophy, with in-
crease in the number of muscle nuclei and here and there the
formation of vacuoles in the fibres. The diagnosis is so much
facilitated by the "myopathic facies"—that is, the expression
produced by the sinking in of the cheek, the somewhat depend-
ent lower lip, and the inability to close the eyes—that the ex-
perienced diagnostician is frequently able to recognize the
disease at the first glance. Marie and Guinon have called
attention to the possibility of confounding the disease with

lepra anæsthetica, in the course of which also weakness of the facial muscles exists (cf. lit.). It is interesting to note in this connection that sometimes disturbances of function in the facial muscles may constitute a congenital defect which under certain circumstances may be followed by an actual atrophy;

Fig. 142.—Progressive Atrophic Myopathy. Inability to close the eyes completely. (After Marie and Guinon.)

further, that in sisters or brothers of individuals who suffer from this myopathy which we have just described, a certain imperfection in the development of the facial muscles may be found, although the disease never breaks out in them. These are facts which Strümpell especially has pointed out, but the cause remains still wholly unexplained. About the treatment we need add nothing to what we have said with reference to the juvenile form.

The third form of the muscular diseases now under consideration—the so-called pseudo-hypertrophy—is connected with an increase in the interstitial adipose tissue which, in spite of the atrophy of the muscle fibres, leads to an apparent increase in the volume of the affected parts. The disease was known and described by Griesinger in 1864, and again by Duchenne in 1868. It begins generally in the muscles of the trunk and attacks, in contradistinction to the two forms just described, by preference the lower parts of the body, the muscles of the back,

loins, and thighs. Though for a long time the patient can use his arms and hands just as well as usual, the walk, owing to the affection of the erector muscles of the spine, becomes altered in the characteristic manner which we have described on page 363. The condition of the patient may remain unchanged for years before the arms also take part in the process. When this happens it occurs in the same manner as in the juvenile form. The diagnosis is very much facilitated by the appearance of the patient. The enlargement of the calf muscles, the thighs, and the glutei (which are sometimes colossal), give to him the appearance of a giant and suggest a supernatural strength (cf. Fig. 143); but the fact that these great masses feel spongy and soft, and that the electrical excitability is considerably decreased owing to the diminution in the number of the muscle fibres, readily explains why these sturdy-looking persons are feeble and without strength and as good as deprived of the use of their limbs.

In its onset the disease resembles closely the other forms. Here also only children become affected, more especially those between the ages of four and nine. Here also the disease may occur in several members of the same family, so that we must undoubtedly assume a hereditary predisposition, and here also the fibrillary twitchings are not met with.

Fig. 143. — PSEUDO-HYPERTROPHY OF THE MUSCLES OF THE LEGS WITH ATROPHY OF THE MUSCLES OF THE BACK. (After DUCHENNE.)

Duration and treatment are the same as in the juvenile atrophy.

Congenital atrophy of the muscles may be found in cases of malformation of the arms and hands. Fig. 144 represents a boy aged thirteen in whom the forearms are absent; some of the fingers are grown together and some deformed. A similar case has been reported by Wilkin (Lancet, page 1265, December 14, 1887), where there was atrophy of the biceps and the brachialis anticus.

Absence of certain individual muscles is rarely observed. Erb has recently reported a case in which there was an almost entire absence of both trapezii (Neurolog. Centralbl., i, 1889). Among earlier instances the pectorales (Ziemssen), the biceps (McAllister), the deltoid, and gastrocnemius (Gruber), were wanting. These cases possess no clinical interest.

The sensory disturbances which are peculiar to the muscles, but about the exact anatomical nature of which we know
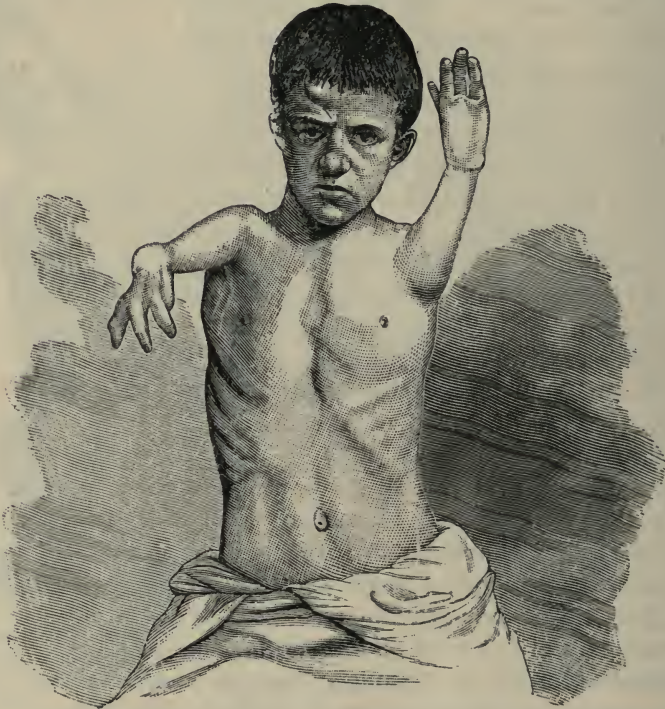
Fig. 144.—Absence of the Forearms. The fingers are partly grown together. Atrophy of the muscles of the upper arms (personal observation).

nothing, are called myalgias or muscular rheumatisms. Ætiologically, overexertion, strains (possibly rupture of certain muscle fibres which may happen during gymnastic exercises or other violent bodily exertion), must be mentioned in this connection. Sometimes we are unable to find any such cause, and we have to attribute the trouble to the influence of cold. There are persons who for years or tens of years suffer from myalgic pains which come and go and may disappear for

certain periods of time completely, and it is just possible that chronic intoxications—e. g., alcoholism, perhaps also circulatory disturbances—have a predisposing influence. Among such myalgias, which may be very painful, even sufficiently so as to interfere with the occupation of the patient for a longer or shorter period of time, we have, for instance, the torticollis rheumatica, in which the muscles of the neck, the myalgia lumbalis (lumbago), in which the muscles of the loins, the myalgia intercostalis, in which the intercostal muscles are attacked. The shoulder muscles may also be affected, and the myalgia in this region may become very obstinate without any implication of the brachial or cervical plexuses being demonstrable. In the diagnosis we must think of the possibility of an implication of the nerves and endeavor to exclude neuralgia. We must further remember that central diseases may give rise to muscular pains. The inexperienced may mistake the lancinating pains of tabes for chronic muscular rheumatism, and thus obscure the correct diagnosis for years. It will hardly be difficult to avoid confounding muscular rheumatism with articular rheumatism if we take into consideration the general condition of the patient, the appearance of the joints, the temperature, pulse, etc., which in the former affection remain normal.

In the treatment we should, first of all, endeavor to detect any underlying cause, and, if such exists, remove it. In recent cases, besides subcutaneous injections of morphine, salicylic acid may be tried internally; yet we should not spend much time with it if we perceive no effect, but should rather prefer local applications—irritants to the skin, poultices, mustard plasters, liniments, also massage and electricity—especially if the affection remains localized. If this is not the case, but if the pains travel round the body and the course assumes a more chronic type, treatment by sweating, steam baths, also mud baths or baths of *Pinus silvestris*, the non-medicated hot springs (Gastein, Johannisbad, Teplitz) or the sulphur springs, among others Pistyán, in Hungary, will be recommended. As a last resort, we may advise the patient to go to a well-conducted hydrotherapeutic establishment (Gräfenberg, Kaltenleutgeben, Nassau, etc.).

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PART III.

DISEASES OF THE SUBSTANCE OF THE SPINAL CORD.

DISEASES confined to the substance of the spinal cord are rarer than those of the brain substance. The cause of this may lie in the fact that not only are the vessels of the spinal cord actually less frequently the seat of disease than those of the brain, but also that when they become diseased the consequences entailed are generally not of so grave a nature as those resulting from lesions of the cerebral vessels.

As in cerebral diseases, here also two questions must ever be kept in view by the physician: (1) Where is the spinal lesion situated? (2) What is its nature? As we shall see later, it is especially the second which is of importance for the prognosis and choice of treatment. Both, however, are of equal weight for the proper recognition and conception of a given case. As in the study of the brain lesions, the topical and pathological diagnosis should here no less go hand in hand.

I. CONSIDERATION OF SPINAL DISEASES WITH REFERENCE TO THEIR SEAT—TOPICAL DIAGNOSIS.

As a thorough acquaintance with the anatomy of the parts is of the highest importance in making a topical diagnosis, some remarks on these points may in this place not be unwarranted.

Without being separated by any sharp line of demarcation from the medulla oblongata, the spinal cord extends from the upper margin of the arch of the atlas to the first lumbar vertebra, where it ends in the conus medullaris. From this point it is seen as a long filiform continuation—the filum terminale. The cauda equina consists of the longitudinal nerve bundles which accompany the filum terminale, and corresponds to the lumbar and sacral part of the vertebral column. As it is apparent that the different pairs of nerves

do not leave the spinal cord at the level of the vertebræ after which they are named, but that they must necessarily do so higher up, it is important to know to what nerves certain parts of the vertebral column correspond. Thus we must remember that the first three cervical vertebræ correspond to the origin of the third, fourth, and fifth cervical nerves, and that the seventh cervical vertebra corresponds to the first dorsal nerve. The spinous process of the fifth dorsal vertebra corresponds to the origin of the seventh, that of the tenth to the twelfth pair of dorsal nerves. Opposite the eleventh dorsal vertebra originates the first, between the eleventh and twelfth the second, opposite the twelfth the third and fourth lumbar nerves. Between the twelfth dorsal and first lumbar vertebra the fifth lumbar and first sacral nerves take their origin, the other sacral nerves opposite the first lumbar vertebra. The cervical enlargement corresponds, therefore, to the spinous processes of the cervical vertebræ, the lumbar enlargement to the spinous processes of the last dorsal vertebræ. All these relations, and, moreover, the fact that the spinous processes, which alone can be our guides, are not always on the same level as their corresponding vertebræ, are demonstrated in Fig. 145.

The relation between the white matter and the gray which it incloses becomes apparent in a transverse section of the spinal cord. Here we see also that an anterior and a posterior fissure divide the spinal cord into two halves. These fissures, however, do not meet, but are separated from each other by the so-called " commissures " which connect the two halves of the cord. The anterior part of the gray matter, the so-called "anterior horn," does not present the same diameter and form throughout, and in the cervical and lumbar en-
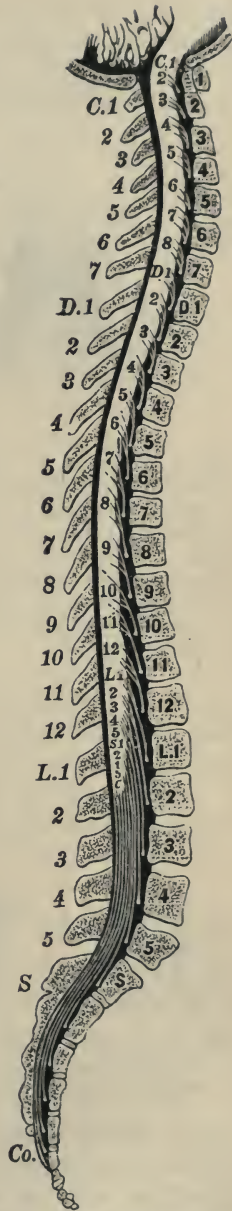
Fig. 145.—THE RELATIONS OF THE ORIGIN OF THE NERVES TO THE BODIES OF THE VERTEBRÆ AND THE SPINOUS PROCESSES. (After GOWERS.)

27

largement is larger than in the dorsal part of the cord (cf. Fig. 147). From this anterior horn proceed the anterior nerve roots and pass through the white matter which lies externally. The posterior horn is much smaller and extends almost to the entrance of the posterior roots, which reach it after passing through the external part of the posterior columns ("root zone" of Charcot). The arrangement of the white substance and its subdivision into columns and tracts is determined (1) by the existence of the above-mentioned fissures, (2) by the entrance of the nerve roots, (3) by the shape of the gray matter. We distinguish roughly an "antero-lateral column" and a "posterior column" on each side. The former contain (a) the crossed lateral or pyramidal tracts (cf. page 188), (b) the direct cerebellar tracts, (c) the anterior direct pyramidal tracts, also called columns of Türck or uncrossed anterior

Fig. 146.—Scheme of the Conducting Paths in the Spinal Cord at the Level of Fifth Dorsal Nerve. (After Flechsig.) *vw*, anterior, *hw*, posterior root. *a*, direct, *g*, crossed pyramidal tracts. *b*, anterior column ground bundle. *c*, Goll's column. *d*, Burdach's columns. *e* and *f*, mixed lateral paths. *h*, direct cerebellar tracts.

columns. The posterior columns consist of the columns of Goll (at the inner side) and the columns of Burdach, which latter have also received the name "root zone" (cf. Fig. 146).

Physiologically, the spinal cord is primarily important as a great conducting system, and next as the seat of numerous centres. The
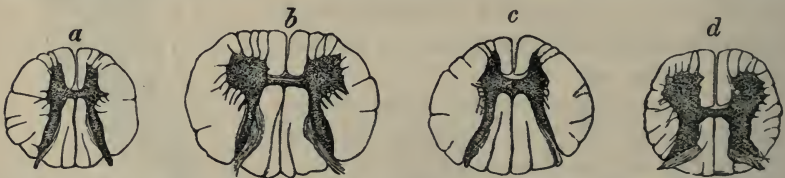
Fig. 147.—Cross-section through the Spinal Cord at Different Levels. *a*, level of the second. *b*, level of the seventh cervical vertebra. *c*, level of the second. *d*, level of the third lumbar vertebra. (After Quain.)

motor impulses originate in the brain (cf. pages 172 and 182), and travel down along the antero-lateral column chiefly in the crossed pyramidal tract of the opposite side, the decussation, as has been repeatedly pointed out, taking place for the most part in the medulla oblongata. Through the large ganglionic cells of the anterior horns these crossed pyramidal tracts are continued into the anterior nerve roots and leave as such the spinal cord. The sensory impressions are

transmitted through the posterior roots, hence (some passing through the postero-lateral columns) they reach the posterior horns, where they at once cross over to the opposite side of the spinal cord. The further course of the sensory fibres as they pass to the brain is not clearly understood; especially imperfect is our knowledge with regard to those for the different qualities of sensation—e. g., the sense of touch. It seems, however, that the central gray substance must be looked upon as the path for impressions of pain (cf. the investigations of Edinger about the continuation of the posterior spinal roots up to the brain, Anatom. Anzeiger, 1889, iv, 4).

We know that reflexes originate by the stimulation of a sensory nerve. By this an impulse is conducted to a centre, and hence is transferred to a motor nerve—reflex arc (Fig. 148). Among such
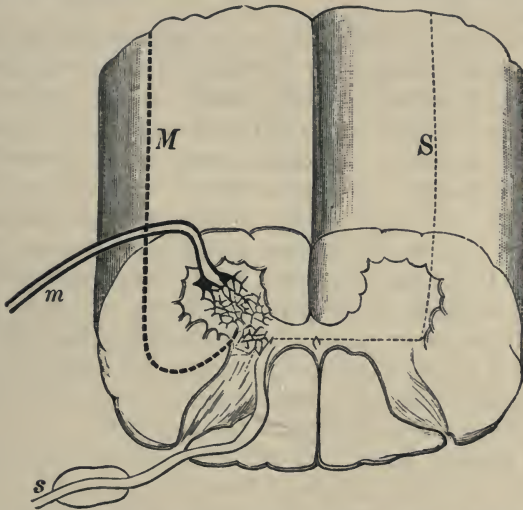
Fig. 148.—Reflex Arc. *M*, motor path. *S*, sensory path. *m*, motor (anterior), *s*, sensory (posterior) nerve root.

reflex movements we distinguish (1) skin reflexes caused by irritation of the skin, (2) tendon reflexes, which are produced by tapping on a tendon. To the former belong the plantar reflex, the centre for which is situated in the lower part of the lumbar enlargement, the gluteal, the cremasteric, and the abdominal reflexes, which are obtained by irritating the skin of the buttocks, the inside of the thigh, and the abdomen respectively. If we find these present in a patient we may assume the centres, which are situated in the lumbar and the dorsal cord respectively, to be intact.

One of the diagnostically most important signs is the condition of the so-called patellar reflex. When the tendon of the quadriceps

femoris is tapped, a reflex contraction of this muscle ensues by which the leg is jerked forward with more or less vigor. This is found in most healthy persons. It has been called by Erb "patellar tendon reflex "; by Westphal, who doubted its reflex nature, "knee phenomenon "; by Gowers, "knee jerk."

To a certain extent the mode of tapping this tendon and the position of the patient is a matter of indifference. The only points to remember are these : The lower leg should be held perfectly loose and no superfluous clothing should prevent the proper striking of the tendon. The simplest way is to place the patient on the edge of a table, remove all clothing from his legs, then, while conversing with him about indifferent matters so as to distract his attention from what is going on, to observe the effect of the percussion of the patellar tendon.

If we find the reflex present, we may at once conclude that the spinal cord at a certain place—that is, from the second to the fourth lumbar or first sacral nerves, according to Westphal—is intact.

If, on the other hand, the reflex is not obtained on the first and after repeated examinations, the patient ought to be directed to interlock his bent fingers and pull strongly (Jendrassik), and only if the knee jerk does not occur after repeated trials in the way described should we assume its absence (Jendrassik, Neurol. Centralblatt, 1885, 18). It has for some time been Jendrassik's experience that the tendon reflexes, more particularly the patellar reflex, is much enforced if the other muscles of the body are put into strong action (Deutsch. Arch. f. klin. Med., xxxiii). This method of Jendrassik is an excellent and indispensable means in doubtful cases for establishing the presence or absence of the knee jerk.

Besides the patellar reflex, the Achilles tendon reflex, and the ankle clonus, the foot phenomenon of Westphal, must be mentioned. The latter consists of a succession of clonic contractions of the tendo Achillis which occur on a sharp dorsal flexion of the foot. To the violent shaking movements of the whole leg, which occasionally occur under these conditions, the very inappropriate name of spinal epilepsy has been given.

If the reflex excitability is much increased, a simple tapping on the front of the lower leg is sufficient to produce a contraction of the calf muscles. This is what the English writers call the " front tap."

Whether all these so-called tendon reflexes are really of reflex nature, or whether they are not rather phenomena due to a direct stimulation of the muscles (Westphal), is still an unsettled question.

The same uncertainty exists about a symptom which has by West-

phal been termed " paradoxical contraction," and which consists in a muscle remaining in tetanic contraction for quite a time after it has been passively shortened. For instance, if we flex the foot of a patient lying in bed, the tibialis anticus may under certain conditions remain for some time in a state of contraction; its tendon becomes prominent, and only gradually relaxes and allows the foot to return to its normal position of rest. Only rarely has this phenomenon been observed in other muscles.

Further reflex centres are found in the lumbar region of the spinal cord for the emptying of the bladder and rectum, for the erection of the penis and the ejaculation of the semen—reflexes which are concerned with the sexual functions. The mechanism of these processes is physiologically by no means understood. Finally, we have vaso-motor centres in the cord, of which sufficient mention was, however, made on page 394.

With regard to the localization of the spinal cord lesion, two questions arise: (1) Which portion of the cord is diseased? Is it the cervical, dorsal, or lumbar? (2) Which part of the cross section of the cord? Is it the gray or the white matter or both? The first question can be answered without difficulty in cases where the vertebral column is diseased; we only need to examine the latter by pressing upon the vertebræ or by applying a hot sponge, etc., over them. Those spots at which tenderness is elicited by the application are the seat of the disease. The occurrence of spontaneous pain is rarer in diseases of the cord. It should, above all, be remembered that lesions of the spinal cord, as such, wherever they may be, almost never produce pain in the back, but that this is in a majority of cases due to trouble in the muscles or their nerves. It is a characteristic feature of these pains that they become especially marked after prolonged standing and stooping, and that they are very bad on rising in the morning. They may occur sometimes after a quick movement, in which case some muscle bundles have been overstretched or even torn. Pains in the back which persist for months and years unaffected by any therapeutic measures justify a suspicion of the existence of an aortic aneurism which may be pressing against the vertebral column or of enlarged carcinomatous abdominal glands (Johnson, British Medical Journal, February 12, 1881). In disease of the vertebral column, especially if it be cancerous, pain in the back is a prominent symptom, as we have said.

But, leaving out the tenderness on pressure, there are other

symptoms which may help us to decide what segment of the cord is diseased in a given case.

Diseases of the cervical cord generally produce symptoms of motor or sensory irritation or of paralysis in the upper extremities, pains, paræsthesias, feelings of weakness, jerkings, and the like in arms, hands, and fingers, to which may be added also trophic disturbances. Muscular atrophies and loss of reflexes in the upper extremities are often observed. The lower extremities, however, remain intact, and the patellar reflex is present and sometimes increased. Repeatedly a very decided slowing of the pulse (as low as thirty-two beats to the minute in a case of Lebrun, Bull. de l'Acad. de méd. de Belgique, i, 1887, i) has been met with in lesions of the cervical cord and has been attributed to a chronic state of irritation of the vagus due to compression or some similar influence.

Affections of the dorsal cord are mostly accompanied by sensory disturbances, paræsthesias in the back, intercostal neuralgias, aching, boring pains, which sometimes radiate into the lower extremities. Anæsthesias, though they are not the rule, may be found. If a distinctly circumscribed zone of anæsthesia is made out, it corresponds exactly to the place where the lesion in the spinal cord is situated (cf. what will be said about lesions of one half of the cord on page 450).

Lesions of the lumbar cord entail symptoms in the lower extremities, giving rise to weakness and paralysis, sometimes also jerkings and stiffness; furthermore, to pains, numbness, anæsthesias of the legs and feet. The reflexes are lost and vesical and rectal symptoms are present, the former consisting of retention or dribbling of the urine, pains, strangury, etc. Of course, the symptoms may greatly vary according as the whole transverse section or only some or even one system of fibres alone is affected in the given level of the cord.

An answer to the second question demands a thorough acquaintance with the symptoms produced by lesions of the different portions of the cross section. These we will, therefore, now consider.

I. LESIONS OF THE GRAY MATTER—" POLIOMYELITIS."

In giving the name poliomyelitis (πολιός, gray) to all spinal affections confined to the gray matter, we must at once insist that these lesions are almost entirely limited to the anterior portion of the gray matter, the anterior horns, and more espe-

cially to the large ganglionic cells in them. Other portions have only rarely been found affected, and then only in connection with the just-mentioned lesion. The diseases of the gray substance proper which have come under observation were confined to the groups of ganglionic cells of which we have just spoken. Clinically, there are two such diseases to be distinguished, namely, poliomyelitis anterior acuta, or spinal paralysis of children (infantile spinal paralysis), and progressive muscular atrophy.

POLIOMYELITIS ANTERIOR ACUTA—INFANTILE SPINAL PARALYSIS.

INFANTILE paralysis, first accurately described by Jacob von Heine in 1840, is one of the best-known diseases of the spinal cord, both as regards its anatomical seat and its clinical course. As has been demonstrated beyond doubt by Charcot, Prévost, and Joffroy, it is an acute inflammation of the anterior horns, or rather, as is usually the case, of one of them. This leads to an atrophy and sclerosis, so that a dense tissue remains, containing the dilated vessels and small remains of ganglionic cells, which are not rarely found to be calcified (Friedländer, cf. Fig. 149). The seat of the process is usually either in the

Fig. 149.—TRANSVERSE SECTION FROM THE CERVICAL PORTION OF THE SPINAL CORD. Atrophy and sclerosis of the right anterior horn. (After CHARCOT.)

cervical or the lumbar enlargement. In the former case the paralysis affects the upper, in the latter the lower extremity. The secondary degeneration, which ensues as a consequence of the atrophy of the ganglionic cells, extends to the anterior

nerve roots, the motor nerves, and the muscles supplied by them. It is a genuine degenerative atrophy, just as much as the one described as coming on after peripheral paralyses.

Symptoms.—The clinical picture of the disease is very characteristic. The onset bears a striking resemblance to that of cerebral infantile paralysis described on page 271. In the midst of perfect health the child is suddenly seized with headache, vague pains in the limbs, and fever, the temperature reaching 104° F. or even more. He becomes stupid and somnolent, and soon, while complete unconsciousness is developed, general convulsions set in, which last usually from one to three days and then disappear. The patient's condition becomes better, consciousness is fully regained, he becomes bright and talkative, and the relations think that the malady has already spent itself, when unfortunately a more careful examination reveals that the movements of the child are impaired, that one, more rarely both, upper or lower extremities are paralyzed. The paralysis, which usually affects one arm or one leg, has developed rapidly and reached a considerable extent, which, however, we would remark, is rarely ever maintained. On the contrary, as a rule, it partially recedes and confines itself to certain muscles, which then remain permanently paralyzed. According to Beevor (cf. lit.), the affection sometimes embraces groups of muscles corresponding to those which Ferrier in his experiments on monkeys saw contract after stimulation of the different cervical nerve roots. In the majority of cases the paralysis takes in one leg. The paralyzed muscles rapidly atrophy and the electrical excitability undergoes quantitative as well as qualitative changes—reaction of degeneration. The whole extremity is stunted in its growth, and even the bones may be found several centimetres shorter than those of the other leg. The appearance of such an extremity in which at first all passive motions are possible is quite characteristic. The skin is pale, cyanotic, and feels cold, but retains its sensibility completely. Skin and tendon reflexes are lost, but there are no vesical symptoms. Later on secondary contractures develop, among which the so-called "paralytic clubfoot" is the best known. In consequence of the paralysis of the peroneal muscles, their antagonists, the calf muscles, become permanently contracted and cause the point of the foot to hang down. In the arms analogous conditions may be found, the

non-paralyzed antagonists always assisting in the production of the contractures.

Roughly speaking, this is the course in most cases, only occasionally the initial fever may be slight enough to be overlooked and the paralysis develop without the child ever having taken to his bed. In rare cases the convulsions, instead of lasting days, continue for weeks. In others, again, several months may pass before the onset of the actual paralysis ; but all these are the exceptions which need not confuse us in making a diagnosis. The further general development (with the exception of that of the paralyzed extremity) is perfectly normal, and neither, as happens in the cerebral infantile paralysis, does the mind become in any way impaired nor do the initial convulsions ever recur. The child grows up in good health, but always remains, especially if one leg is affected, a cripple. If, as often happens, contractures or a spontaneous paralytic luxation of the hip develop, the patient has for years to be under the care of the surgeon and needs braces and the like. If an arm is affected, the capability of the patient for making his living is naturally considerably and permanently interfered with.

Diagnosis.—It is not difficult to avoid mistaking the disease for any other if we bear in mind the characteristic onset, the localization, the behavior of the paralysis itself, the flaccid condition of the muscles, the absence of the reflexes, and the cold and cyanotic skin. Where we find a hemiplegia—i. e., where the arm and leg of the same side are paralyzed—we should in children always first think of infantile spastic hemiplegia (page 272), as it is one of the rarest exceptions for the spinal paralysis to take on this distribution. Confusion with the syphilitic pseudo-paralysis, also known under the name of Parrot's disease, is avoided by remembering the fact that in this disease the paralysis makes its appearance immediately or at least within a few days after birth (Dreyfouss, Revue de méd., août 1885, v), while Heine's paralysis of children does not occur at such an early age.

Prognosis.—The prognosis, as soon as the initial acute symptoms have passed, is, as far as life is concerned, absolutely favorable ; as far as the recovery of function in the affected extremity is concerned, equally unfavorable. Any notable improvement is very rare, complete cure out of the question. These points should be carefully considered before inducing a

poor and struggling father to let his child undergo year after year an expensive and useless course of treatment.

Ætiology.—Of the ætiology of the disease we know nothing. It is doubtful whether cold is ever a causative factor. It is possible that infectious influences, the action of certain micro-organisms, will at some time be proved to be the cause of the disease. For the present, however, this is nothing more than a hypothesis which has not gained any firmer ground from the report of Cordier of an epidemic of the disease (Lyon méd., 1888, I, 2). In a small village thirteen children were inside of two months taken ill with anterior poliomyelitis and four died. According to Cordier, the appearance of the disease in summer, the sudden onset, the similarity in course, speak for an infectious origin, the infection, as he supposes, taking place through the air passages.

Treatment.—Little more is known about the treatment than about the ætiology. All measures to cure or even merely to improve this rapidly developed paralysis are more or less useless. Electrical treatment with the faradic or galvanic current, systematic massage, gymnastic exercises, together with rubbing with all sorts of salves—all these have been tried without any noteworthy success. In a few cases I have seen the methodical use of heat, in the form of hot sand baths, warm packs, etc., bring about at least a perceptible improvement; but even here this was out of proportion to the care and trouble which had been taken. Certain it is that the influence of the different baths has been greatly overrated, whether it be the brine baths of Kreuznach, Reichenhall, Kolberg, or the chalybeate springs of Pyrmont, Flinsberg, Schwalbach, or the sodium waters of Rehme, Soden, or finally the non-medicated hot springs of Gastein, Johannisbad, and many others, each of which has its advocates. The most appropriate appear to be those last mentioned, but in most cases we shall even then find that while perhaps the child's general condition is improved and it becomes strong owing to the good hygiene and fresh air, the paralysis, for the sake of which all has been undertaken, remains absolutely unchanged and presents no improvement.

In view of these unsatisfactory results, the interesting but still scanty communications, according to which the growth of bone can artificially be increased, deserve our deep interest. In 1887 Helferich proposed to tie round the affected (paralyzed, atrophic) limb elastic rubber tubing so as to produce an arti-

ficial engorgement, and through this a more active nutrition of all the tissues, including the bones. Schüller also has reported before the Berlin Medical Society, November 28, 1888 (Deutsch. Med.-Ztg., 1888, 99, page 1182), several cases which were thus treated and which showed decided improvement. To judge from his communication, this treatment undoubtedly should be tried in all suitable cases. It is, however, a procedure which, as well as the orthopædic treatment so important for the prevention of deformities, should not be undertaken without consulting a surgeon.

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Though the lesions of the gray anterior horns when occurring in children are well understood, both in their anatomical and their clinical aspect, yet when the same process takes place in adults our knowledge becomes very limited. Here the material at our disposal is still so small that only in rare exceptions can we say definitely whether we are dealing really with an anterior poliomyelitis and not rather with a peripheral disease, a multiple neuritis. Clinically, the differential diagnosis between the two can only be made in the initial stage, as the peripheral disease is accompanied with pains and sensory disturbances which are absent in the central affection.

A patient is taken ill with grave general disturbances—

fever, somnolence, convulsions, delirium, etc.—and within a short time, perhaps in one or two weeks, a widespread paralysis in all four extremities is developed. The paralyzed muscles become flaccid and atrophy, the tendon reflexes disappear ; sensation, however, as well as bladder and sexual functions, present no abnormity. With a history like this we must think of a lesion of the anterior gray horns. This idea becomes more than a conjecture if on examination the affected muscles are found to be such as are supplied from ganglionic cells, which most probably lie in close proximity to one another in the spinal cord. In such cases, as Remak has shown so beautifully, certain types of paralysis are observed—the forearm type (paralysis of all the extensors without the supinator longus) and the upper-arm type (paralysis of the biceps brachialis anticus, deltoid, and the supinator longus)—but unfortunately such instances are rare, and therefore even quite an experienced physician may feel uncertain about the diagnosis.

The difficulty becomes greater if the paresis or paralysis is not extensive and does not develop rapidly, but slowly and by fits and starts. In these cases not rarely a temporary improvement may be noted and arouse hopes of complete recovery, unfortunately never justified. These are the instances in which we find not complete, but partial reaction of degeneration in the paralyzed muscles—intermediate form of chronic anterior poliomyelitis (Erb). It goes without saying that we must have the other symptoms, especially the loss of reflexes, even to justify a conjectural diagnosis. Moreover, it is necessary that there should be absolutely no sensory changes and that bladder and sexual functions should be normal. Of the points of difference between anterior poliomyelitis and tabes we shall speak later.

We can hardly expect much from any treatment. Electricity, however, should be tried, if for no other reason than that something is done. Duckworth recommended, besides, belladonna, iron, quinine, and cod-liver oil, and claims to have cured cases with these remedies.

With reference to the ætiology, nothing certain is known. Whether traumatism can ever cause anterior poliomyelitis remains doubtful, notwithstanding the report of Gibbons (Med. Times and Gazette, September 5, 1885). He had among his patients a boy nine years of age who after a fall on his knees developed the symptoms of an anterior poliomyelitis (and re-

covered completely !).   In cases of chronic anterior poliomyelitis which came to autopsy, sometimes atrophy in the ganglionic cells of the anterior horns through the whole length of the cord, as well as atrophy of the anterior roots, was observed, while the peripheral nerves remained intact (Oppenheim).

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PROGRESSIVE muscular atrophy was first described by Du-
chenne and Aran in 1849 and 1850, and was recognized by
Cruveilhier in 1855 as a spinal affection. Thanks to the work
of Lockhart-Clarke, and especially that of Charcot, the occur-
rence of a pathological process restricted to the gray substance
of the spinal cord, which is accompanied by a muscular atrophy
of typical distribution, is now established beyond the slightest
doubt.

Pathological Anatomy.—The process, which is usually most
pronounced in the cervical cord, consists again of an atrophy
and transformation of the gray anterior horns into a fine fibrous
tissue containing spider cells. The large ganglionic cells are
partly or wholly destroyed, or at any rate are diminished in
number and perceptibly smaller. Here, too, the lesion extends
to the anterior nerve roots and the corresponding fibres of the
motor nerves. On microscopical examination we find that the
muscles supplied by them retain their transverse striation, but
the fibres are decidedly diminished in size. Some fibres also
show the so-called degenerative atrophy—that is, a fatty, wax-
like degeneration, with increase of the interstitial connective
tissue and multiplication of the muscle nuclei. Which of the
described processes has to be regarded as the primary one,
or, in other words, whether the disease actually does start in
the gray matter of the cord and not perhaps in the peripheral
nerve endings, or whether this differs in different cases, we
are unable at present to decide. One thing we ought to keep
in mind with reference to the anatomical seat of the affection,
namely, that in the cord no other parts than the ones men-
tioned are affected, and more especially that the pyramidal
tracts always remain intact.

Symptoms.—The onset of the disease is in many cases very characteristic. The patient begins to complain of weakness in the arms, sometimes more in the right than in the left, which soon interferes to some extent with his ordinary actions. Sensory changes and pains are absent, a point which is of vast diagnostic importance. Not many weeks after these symptoms have appeared the competent observer will notice a peculiar flatness, a sunken-in condition of the ball of the thumb, while at the same time the thumb is more than usually approximated to the second metacarpal bone ("ape hand," Fig. 150). The in-

Fig. 150.   Fig. 151.

Figs. 150, 151.—PROGRESSIVE MUSCULAR ATROPHY. (After EICHHORST.) Fig. 150. Ape-
    hand. Fig. 151. Sunken-in interosseal spaces on the back of the hand.

terosseal spaces on the back of the hand are sunken in and the terminal phalanges of the fingers are in incomplete extension (Fig. 151). The hollow of the hand seems flattened (atrophy of the lumbricales) and the atrophy of the muscles of the thenar and hypothenar becomes more and more apparent. As the function of the interossei becomes disturbed to a greater extent, the same claw-like position of the fingers develops which has been described on page 349 as occurring in affections of the ulnar nerve ("claw hand," "*main en griffe*").

After this condition has thus for weeks or months undergone no marked change, the disease begins to attack either the muscles of the forearm, or, passing over these, implicates the muscles of the shoulders and with special preference the deltoid. In the former case the extensors are attacked earlier and more seriously than the flexors. The muscles of the trunk and

legs are either later or never affected, but if the diaphragm and other respiratory muscles are invaded this may prove fatal as well as an extension of the process from the cord to the medulla oblongata, in which case the symptoms of progressive bulbar paralysis are superadded (page 154). If this does not take place and the respiratory muscles are spared, the disease may last for years and tens of years and death is only caused by an intercurrent acute malady.

Apart from the characteristic onset, the following signs help to make the diagnosis certain: (1) Fibrillary twitchings in the affected muscles, which can at times be produced by tapping the muscles, but which often are seen to appear of their own accord and continue without interruption. (2) The condition of the electrical excitability, which depends directly on the number of muscle fibres left. If the greater number of the fibres are wasted, then the excitability for both currents is equally decreased. If all the fibres of a muscle have disappeared and only fat and connective tissue remain, the excitability of the muscle is completely lost. It is only exceptionally that the excitability also undergoes qualitative changes and we find reaction of degeneration. (3) The loss of the tendon reflexes, which is sufficiently explained by the disappearance of the ganglionic cells, a part of the reflex arc. It is only because the lower extremities are rarely affected that the patellar reflexes are usually retained. (4) Sensibility remains everywhere and for any kind of impressions intact (touch, pressure, pain, temperature). The coldness and blueness of the hands is to be attributed to the disuse of the muscles. True trophic disturbances of the skin, as well as bladder and rectal symptoms, are usually absent.

Diagnosis.—Remembering, then, the different points just alluded to, the diagnosis should be easy, and it will not be difficult to avoid confounding the disease with myelitis, neuritis, or syringomyelia. The flaccid paralysis, the absence of all symptoms of motor irritation and sensory disturbances, is especially of moment in differentiating this disease from myelitis. More particularly characteristic is the commencement, the onset of the disease in the small muscles of the hands. If this has been well pronounced an error in diagnosis is unpardonable.

Ætiology.—With reference to the ætiology a little more is known about this disease than about spinal infantile paralysis; for certain cases at least it has been shown that overexertion

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of the muscles, as happens sometimes to those who work with the sewing machine, has a causative influence, or at any rate the disease has been preceded by some overexertion of the muscles, to which we are then justified in attributing an ætiological importance. The conditions, however, under which paralysis and fatigue of the muscles lead to atrophy—why, for instance, the serratus magnus (Chvostek) is in some cases at first thus affected—we are wholly ignorant of, just as we do not know the conditions under which the genuine hypertrophy develops which we often find in the biceps of blacksmiths. Recently Bernhardt has again drawn attention to the fact that the disease may be hereditary (Virchow's Archiv, 1889, 115, 2).

Little need be said about the therapeutics; there is no effectual treatment, and all measures that have been tried have not been efficient in hindering the progress of the disease.

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(Cf. also pages 414 and 415.)

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II. LESIONS OF THE WHITE MATTER OF THE SPINAL CORD— "LEUCOMYELITIS."

While, as we have said on page 422, the lesions affecting the gray matter (poliomyelitis) are almost entirely confined to one portion of it—namely, the anterior horns—we shall soon see that this is different with the lesions of the white matter, to which the general name leucomyelitis may be given (λευκος, white). Here different parts can be attacked, either alone or in conjunction with others, and it is of great importance to differentiate between the clinical symptoms which occur in the diseases of the different columns or "systems" (Flechsig), hence called "system diseases."

The affection is either a primary one, when it is often impossible to ascertain any ætiological factor, or it occurs secondarily and as a consequence of certain affections of the brain (cf. page 229) and the spinal cord itself, such as traumatic inflammations and compression. We shall consider both separately.

A. Primary Lesions of the White Columns.

Regarded from an anatomical standpoint, the primary tract-degenerations of the white substance consist in a destruction of the nerve fibres and a simultaneous increase of the neuroglia. The medullary sheaths are the first to disappear; the axis cylinders, which are more resistant, do not degenerate till later. Compound granular corpuscles, which remove the detritus from the diseased regions (Ziegler), accumulate in the lymph sheaths of the vessels. The increasing neuroglia crowds in and displaces the empty nerve tubes, a process which, in conjunction with the thickening of the walls of the vessels, which develops at the same time, is described under the name of sclerosis, or gray degeneration.

An affection confined to one nerve tract or system has up to this time only been observed in the antero-lateral, but not in the posterior columns. In the former, the anatomical arrangement of which has been described on page 418, we meet especially frequently with sclerosis of the so-called crossed pyramidal tracts, but the lesion does not necessarily extend over the whole length of the tract, but may be only partial (Westphal). Most of the cases which have come under observation

were, however, not pure instances, but presented other ana-
tomical changes as well, and there is only one case reported,
by Dreschfeld, in 1881, which, viewed from an anatomical
standpoint, can pass for a pure lateral sclerosis.

The primary sclerosis of the lateral columns—spastic spinal
paralysis, *tabes dorsale spasmodique*—was first described by Erb
and Charcot in 1875, and characterized by them as a motor
paralysis with remarkable increase in the tendon reflexes ; and,
indeed, if we examine such patients, all we find is that they
have lost to a greater or lesser extent the use of their legs ;
they are unable to walk, the feet are glued, as it were, to the
floor, and the patient can only shuffle along, the inner margin
of the foot never leaving the ground.   At the same time the
muscles feel firm and hard, the legs are in extension, and any
attempt at flexion is difficult.   If such a patient is made to sit
on the edge of a table the legs do not hang down flaccidly, as
might be expected, but are thrown into a state of tetanic
tremor, produced by contractions of the quadriceps extensor.
There is an enormous exaggeration of the patellar reflexes, and
the ankle clonus is obtained without the slightest difficulty.
With the exception of the inability to walk, the patient has no
subjective complaints ; neither sensation nor the functions of
the bladder, rectum, or the sexual apparatus show any abnor-
mity.   An implication of but one of these would at once
exclude the diagnosis of lateral sclerosis, as would also (and
this should be especially remembered) the existence of any
muscular atrophy.   The very characteristic spastic or spastic-
paretic walk of the patient, the traces which his feet leave on
a gravel path, for instance, and which can be followed up as
distinct continuous streaks, the shuffling noise which accom-
panies every step when he attempts to walk about the room,
these are of great diagnostic value ; the examination of the
soles of the patient's shoes, which appear thinner and more
worn down on the inner side, will be of interest and value.

The disease begins in youth or in middle life, attacking
first the one then the other leg, without, as a rule, extending to
the arms or trunk, yet the upper extremities are said to be
occasionally affected (Strümpell).   The disease may last years
or tens of years without presenting any decided change for the
worse.   Death is brought about by intercurrent diseases.   It
is not known whether, as in progressive muscular atrophy,
overexertion is of ætiological importance ; instances, however,

where acrobats (Donkin) and hod-carriers (Munter) were attacked seem to suggest this. Morgan pointed out that exposure to cold, such as long standing in water, may be the immediate cause of the disease (Lancet, January 19, 1881).

Much more frequent than a lesion confined to the crossed pyramidal tracts is one which implicates not only these, but with them the posterior columns and the direct cerebellar tracts, in which, although not always, Clarke's columns take part. The anatomical character of this "combined system disease" which results from these lesions has been repeatedly described (Westphal, Gowers, Strümpell). The symptoms vary according to the distribution of the lesion; thus, if the disease of the lateral columns extends low down, while the posterior columns in the dorsal and lumbar region present no changes, rigidity of the muscles and increase of the reflexes will be found. If, on the other hand, the disease in the posterior columns extends further downward, these symptoms will be absent, the lesion in the lateral being neutralized as it were by that in the posterior columns (Westphal).

Not rarely the affection seems to depend on faulty development, a condition which we may meet with in more than one member of the same family, and which may be hereditary. In these cases the disease appears in early childhood, and, as we said, sometimes in several children of the same family. It has been called, after the author who first described it, Friedreich's "hereditary ataxia." The motor disturbances in the children begin in the feet, the walk becomes awkward, they stumble, and in passing over small obstacles have to look at their feet to keep from falling, etc. The patellar reflexes disappear; the arms are not affected until later, and, indeed, they are by no means always implicated. The second motor disturbance establishes itself in the muscles of the tongue and the lips which are necessary for speaking, and this produces a very characteristic defect of speech of motor origin. Finally, the muscles of the eyes become implicated, and there results a distinct nystagmus. The combination of these three symptoms is pathognomonic for this rare disease. It has no connection with tabes and sensory changes, and bladder symptoms, manifestations which are probably never wanting in cases of tabes, are never met with in the disease under consideration. Nor can it be mistaken for multiple sclerosis, as vertigo and "scan-

ning speech" are never associated with it. The course is tedious, the prognosis always unfavorable, the muscles undergo atrophy in consequence of inactivity, and contractures occur in the joints.

Similar symptoms are observed in adults in cases of combined lateral and posterior sclerosis; yet there are certain peculiarities to which Gowers especially has drawn attention. The disease was named by him "ataxic paraplegia." The lower extremities are ataxic and paretic, which gives rise to an uncertain, swaying walk; but this is associated with paræsthesias, weakness of the sphincters, and decrease of the sexual power. The patellar reflexes are at first increased, and only at times become lost later in the disease. Such an increase is never known in hereditary ataxia. During the period of increase, rigidity of the muscles, spasm, and ankle clonus are present. It is evident that cases of this kind may be mistaken for tabes, especially if the patellar reflexes are absent, as exceptionally occurs. Then the history may be of use to us, as syphilis seems to possess no ætiological importance whatever in the combined sclerosis, while exposure to cold and overexertion seem to be of considerable moment.

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B. Secondary Lesions of the White Columns.

Lesions of the motor centres of the brain cortex, or lesions of the path between these centres and the motor ganglia of the spinal cord, the so-called cortico-muscular tract or pyramidal tract, give rise to a descending degeneration of the motor fibres on the same side as the brain lesion. This secondary degeneration is in the cord continued in the crossed pyramidal tract of the opposite side, while the direct pyramidal tract only presents traces of it. About the causes of this sclerosis we possess just as little definite knowledge as about the clinical symptoms by which it manifests itself. The former is sought in the cutting off of the parts from their trophic centres, and with regard

to the latter it is generally supposed that the gradual developing rigidity of the muscles, the increase of the reflexes, and the later contractures depend on this degeneration. The cases, however, in which at the autopsy an extensive degeneration was found, while during life not a trace of such symptoms was present, do not speak much in favor of this view.

Lesions of the whole transverse section of the cord also produce secondary degeneration, which, however, not only extends downward (in the pyramidal tracts), but also upward—(1) in the inner segment of the posterior columns (Goll, cf. Fig. 152), and (2) in the direct cerebellar tracts (Flechsig), which are in connection with Clarke's columns (cf. Fig. 153). While this ascending degeneration is physiologically extremely interesting, as it indicates that the trophic centres of these two tracts must be situated more peripherally (as, for instance, in Clarke's columns), we are not as yet able to attribute any clinical importance to it.

Fig. 152.—Ascending and Descending Degeneration in the Spinal Cord. *A*, primary area of degeneration (lesion). *B*, degeneration of Goll's columns (ascending). *C*, degeneration of the crossed pyramidal tract (descending). (After Gowers.)

Fig. 153.—Secondary Ascending and Descending Degeneration in a Transverse Affection of the Upper Dorsal Cord. The ascending degeneration occurring in Goll's columns and the direct cerebellar tracts, the descending degeneration in the crossed pyramidal tracts. (After Strümpell.)

III. LESIONS OF THE GRAY AND WHITE MATTER OF THE SPINAL CORD.

Charcot and Joffroy were the first to show that the large ganglionic cells of the gray anterior horns and the pyramidal tracts can be affected simultaneously by a disease which produces characteristic clinical symptoms, but it was not until Flechsig announced his discovery of the system of conducting

fibres that these clinical observations became fully understood. Now we know that the disease which the French authors, following Charcot, have termed *sclérose latérale-amyotrophique*, amyotrophic (more properly myo-atrophic) lateral sclerosis, consists of a lesion of the cortico-muscular tract which begins as a degenerative atrophy in the lumbar cord and which, as Charcot and Marie have lately pointed out, can be traced as far as the motor ganglionic cells of the central convolutions. Attention has already been called to the fact that, just as the ganglionic cells of the anterior horns, in the same way the motor nuclei of the medulla oblongata may be implicated, and thus the clinical picture of progressive bulbar paralysis develop. The two diseases are therefore analogous, and akin to them is a third—namely, the progressive spinal muscular atrophy—in which affection also the large ganglionic cells are diseased, as we have already pointed out on page 431. From the ganglionic cells the atrophy spreads toward the periphery to the anterior nerve roots and the muscles supplied by them.

That the clinical manifestations are strictly motor and trophic, and that no sensory changes can occur, we can well understand from the anatomical distribution of the lesion. The patients at first complain of weakness in the arms and the hands, which soon interferes with their occupation. This loss of strength increases fairly rapidly, and the atrophy in the muscles of the hand—the thenar, the antithenar, and interossei —becomes more and more apparent.

The muscles of the arms also waste, more especially those of the extensor side, and the former roundness of the shoulder is soon lost owing to the atrophy of the deltoid. The triceps and other muscles also then take part in the lesion, and the helplessness of the patient, who has but little use of his upper extremities, rapidly increases. At the same time the tendon reflexes are increased and tapping of the bones of the forearm elicits lively contractions of the muscles ("periosteal reflex").

That the so-called "jaw-jerk," which has been described by De Watteville, is characteristic of the disease I am very much inclined to doubt, since in a number of perfectly healthy persons I found it in some present, in some absent. It certainly does not possess any diagnostic value. This jerk may be produced by pressing down the lower jaw by means of a broad paper-cutter and tapping the latter with a percussion hammer

near the teeth.   The lower jaw will then respond with a contraction of the muscles of mastication.

In a relatively short time the paralysis of the upper extremities has become so complete that not even the slightest motion is possible, and gradually contractures develop (by preference in the wrist and elbow joint).   In the lower extremities the same changes may be noted, but they make their appearance later and do not reach such a high degree.   Here, too, we have first weakness, difficulty in walking, and general awkwardness in making movements, then rigidity and stiffness of the muscles, enormously increased patellar reflexes and ankle clonus, later on total immobility and contractures in hip, knee, and ankle joints.

A case in one of my wards, a woman thirty-four years of age, has been for two years without power of motion, and is so entirely deprived of the use of her four extremities that without assistance she is unable to make even the slightest motion with either fingers, hands, arms, toes, feet, or legs.   The disease goes on to invade the motor nuclei of the medulla oblongata, and hence is produced difficulty in swallowing, which ultimately amounts to a total inability to get food down, and the patient dies of starvation.   At other times a disturbance of the respiratory apparatus may bring about a fatal issue.   It is exceptional that the whole course of the disease comprises a period of more than two or three years.   The diagnosis is, as a rule, easy enough, and it is not difficult to differentiate the disease from progressive muscular atrophy if its duration and the condition of the reflexes are well borne in mind.   Of the cause of the disease as well as of effectual means wherewith to combat it we are equally ignorant.

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While the diseases of the cord which we have studied so far were confined to certain systems of fibres—in other words, were "system diseases"—the affection now to be considered does not present this peculiarity, but the process which affects the gray as well as the white matter is more or less widely extended over the cross-section of the cord, forming a small number of large or numerous small foci. In other words, the disease is what we call "asystematic" or diffuse. It is an inflammation of the cord, which according to its course is called an acute or chronic myelitis, and to which the name transverse myelitis has also been given.

Pathological Anatomy.—Anatomical changes may in such cases be scarcely demonstrable even though the severest paralytic symptoms may have existed during life. This is more especially true in cases of spinal paralysis due to pressure, occurring in consequence of disease of the vertebræ. Here we must assume that even moderate pressure is capable of bringing about a break in conduction without any destruction of nerve elements. Usually in cases where changes can be demonstrated we find a diminution in the size and an atrophy of the nerve fibres. The axis cylinders appear swollen and have lost their myeline sheath. The ganglionic cells, which are not affected until later, become shrunken and lose their processes. While thus the nerve tissue undergoes disintegration the connective tissue increases, the meshes of the neuroglia become broader, and in it are seen the connective-tissue cells first described by Deiters, which, owing to their numerous processes, have also been called spider cells. In the meshes of the neuroglia reticulum, compound granular corpuscles are found which have taken up the fat and disintegrated nerve substance. These are leucocytes and in turn undergo, sooner or later, destruction. The vessels are dilated and changes are seen in their walls, consisting of thickening or hyaline degeneration. In cases where this process has run its course in a comparatively short time, the cord is found at the autopsy to be soft and of a grayish-red color, whereas if the process has been slow the cord appears, in consequence of the connective-tissue increase, hardened, or, as we say, "sclerosed."

Macroscopically, little is to be seen. At the most some portions may, when the cord is put into Müller's fluid for the purpose of hardening it, look light yellow, while others are dark green. The former are the diseased parts, which can not be-

come stained because the myeline sheaths, which are turned green by chromium, are absent. With this exception all information about the pathological changes must be derived from the microscopical examination of fresh as well as of hardened sections.

According to the location of the process we distinguish a dorsal myelitis, the most common ; a lumbar myelitis, the rarest; and a cervical myelitis, a relatively frequent form. In the first and second the upper extremities are entirely intact, while they are implicated if the process is situated in the cervical cord.

Symptoms.—It is very natural that the clinical manifestations of myelitis must, on the whole, very much resemble those which we have learned to recognize in the "system-diseases," and, as a matter of fact, almost all that will be described has already been said. Here, as there, we have to do with motor, sensory, and trophic disturbances, with changes in the reflexes and symptoms referable to the bladder and rectum. The motor disturbances may consist of symptoms of paralysis and irritation. The former are usually the more prominent of the two, and weakness of the legs, which sooner or later amounts to complete palsy, is one of the chief symptoms of a myelitis. As a rule, both legs are about equally affected—paraplegia ; sometimes one retains its strength longer than the other, according to the extent to which the pyramidal tracts are diseased. If not the legs but the arms are paralyzed, the lesion is situated in the cervical cord. The symptoms of irritation consist of twitchings, which occur sometimes spontaneously, sometimes as the result of slight stimulation of the skin. In many instances the removal of the bedclothes and the change of temperature resulting therefrom are sufficient to cause quite protracted clonic spasms of one or both legs. This and similar phenomena seem to be of reflex origin.

The sensory changes are less regularly met with and are of less importance than the motor disturbances. There are indeed cases where they are almost entirely absent or where they at least do not annoy the patient or do not become marked until relatively late in the course of the disease. They consist mostly of paræsthesias, numbness, formication, also of decrease in sensibility, which may amount to a complete anæsthesia, varying in extent and situation. Actual pains, which are sufficient from their duration and intensity to cause much suffering to the

patient, and which are so commonly seen, as we shall learn, in tabes, belong in this disease to the exceptions. In fact, we may say that they are usually absent or, at any rate, not at all severe. If we are able to detect sensory changes on the trunk itself, the level up to which these extend gives us valuable indications as to the seat of the myelitis. If it is in the lumbar cord, sensibility is intact above the navel; if in the lower dorsal, above the middle of the sternum. Sensory changes in the neck and upper extremities indicate the seat to be in the cervical cord. The more prominent the sensory disturbances and the pains, the greater is the extent to which the gray matter of the posterior horns and the posterior columns participates in the inflammation or degeneration.

Trophic disturbances appear when the trophic centres—that is, the ganglia of the anterior gray horns—are diseased. Thus, if we are able to demonstrate atrophy, with reaction of degeneration in the legs, this denotes a lesion of the gray anterior horns in the lumbar cord, while the same condition in the arms indicates a disease of the anterior horns in the cervical cord. The electrical examination should never be omitted in such cases, because it may happen that the legs present a certain degree of atrophy without the presence of any reaction of degeneration. This atrophy is, then, purely the result of disuse—the atrophy of inactivity. Other trophic disturbances or vaso-motor changes in the skin are not the rule. Herpes and urticarial eruptions, slight œdema and changes in the sweat secretion occur, but possess neither diagnostic nor prognostic value.

One symptom remains still to be mentioned, because it is rarely wanting, but rather plays an important *rôle* in myelitis, and causes endless annoyance and discomfort to the patient—namely, the bed-sores which occur in the sacral region, and become the more extensive the less the care exercised in the nursing and for the cleanliness of the patient. This is one of the most important trophic disturbances, and one which, even with the most careful attention, can not in all cases be avoided.

The condition of the skin as well as the tendon reflexes depends (1) on the state of the reflex arc in the spinal cord, (2) on the state of the fibres coming from the brain, which have probably an inhibitory function. If the reflex arc is normal, but the conduction of the inhibitory fibres interrupted, then

the corresponding reflex is increased, while if the reflex arc is diseased the reflex is lost, no matter whether the conduction of the inhibitory impulses be intact or not. This holds for the skin as well as tendon reflexes. Therefore, in cases of lumbar myelitis not only the skin but also the tendon reflexes are diminished or lost in the lower extremities. Those concerned are the patellar reflex, the reflex arc of which corresponds to the cord between the second and fourth lumbar nerves; the tendo-Achillis reflex, the arc of which corresponds to the first sacral nerve; the cremasteric and abdominal reflexes, which have their arc at the level of exit of the first lumbar and a portion of the cord between the fourth and seventh dorsal nerves respectively. On the other hand, in a dorsal or cervical myelitis a marked increase of the tendon and skin reflexes of the lower extremities takes place, because the (supposed) inhibitory influences are cut off.

A symptom which, perhaps, causes the patient himself more annoyance than any other is the disturbance in the functions of the bladder, which in a myelitis is hardly ever totally absent. At first there is some difficulty in micturition, which may end in complete retention, so that the patient can not void his urine, but requires to be catheterized. In the later stages of the disease, however, the urine is passed involuntarily, there being either a constant dribbling—incontinentia urinæ—or from time to time an involuntary evacuation of the bladder. In either case the patients can not dispense with a portable urinal. Occasionally there is a painful burning sensation when the urine is passed—ischuria—so that the patient dreads every evacuation of the bladder. As might be expected, cystitis frequently develops in these cases, partly owing to the length of time that the urine remains in the bladder, partly owing to the frequent use of the catheter. The rectal symptoms consist either of a most obstinate constipation, or, if the sphincter ani becomes paralyzed, of incontinence of fæces (incontinentia alvi), which aggravates to a very serious extent any bed-sore that may be present. For the localization of the myelitic process neither bladder nor rectal symptoms can be used. They are always present at whatever level the lesion may be.

Ætiology.—Of the ætiology of myelitis little is known. It seems justifiable, however, to divide the causes into those which act chemically and those which act mechanically, the former being either of an infectious or of a toxic nature. That

infectious diseases may produce myelitis is shown by the fact that it occurs occasionally after diphtheria, more frequently after small-pox, and also during the course of syphilis, and that the influence of poisons may at least favor the development of myelitis has been upheld since the action of arsenic, of mercury, and of lead, and the symptomatology of the resulting intoxications have been more accurately studied. We shall later return to this. Among the mechanical causes the most important is pressure, which can be exerted upon the cord by structures surrounding it, as happens, for instance, in spinal meningitis and meningeal tumors. Of greater importance in this connection is the chronic caries of the vertebræ (malum Pottii), spondylarthrocace, the tubercular spondylitis, and carcinoma of the vertebræ (cf. Figs. 154 and 155), in which either the dis-
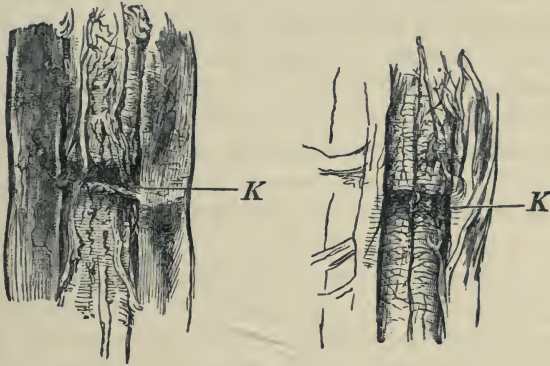
Fig. 154.   Fig. 155.

COMPLETE INTERRUPTION OF CONDUCTION OF THE SPINAL CORD DURING LIFE. Fig. 154, anterior, Fig. 155, posterior aspect of the spinal cord. The dura mater is divided and folded back. Circular compression and narrowing of the spinal cord at *K* in consequence of carcinoma of the vertebræ in a woman thirty-four years of age. Natural size. The drawing is made from a fresh preparation. (After EICHHORST.)

located (diseased) vertebræ themselves or the caseous and inflammatory products which are found between the dura and the bone may exert a compressing influence. That there are still other causes which may give rise to myelitis we do not deny ; we would only mention bodily fatigue and exposure to cold, but these are infinitely rarer. On the other hand there exists not the smallest ground for the assertion that sexual excesses ever produce it.

Course.—The course in general is the following : After the patient has for weeks and months managed with difficulty to

get around, his legs becoming weaker and weaker, he has to take to bed or to the rolling chair, where he spends one, two, even four years, harassed by various afflictions, among which the bladder symptoms and the motor disturbances are especially prominent. Recovery, if it occurs at all, is only very exceptional, and the prognosis must, therefore, always be very unfavorable. Death occurs in consequence of bed-sores, which are seldom absent, or is at least precipitated by them. Some patients die from intercurrent diseases, others from the cystitis. The course of the so-called pressure myelitis and its resulting pressure paralysis, the symptoms of which have before been alluded to on page 314, is so far characteristic that we can here distinguish a prodromal stage, a stage of irritation, and a stage of paralysis. The prominent features of the first are rigidity of the vertebral column, dull, vague pains in the back, and the first signs of a commencing deformity. In the second stage we have severe neuralgic pains, hyperæsthesias, paræsthesias, and girdle sensations. In the third, finally, paralytic symptoms, increased reflexes, vaso-motor and trophic disturbances (herpetic eruptions, muscular atrophies, bed-sores, etc.). This distinction, however, is only possible in isolated cases. Bladder and rectal symptoms are absent in no case of pressure paralysis.

In our prognosis we must not leave out of consideration the possibility that the inflammatory new formations in the vertebræ may disappear, and thus, the cause which produced the break in the conduction ceasing to act, it may be possible for the spinal cord to recover completely all its normal functions, provided, of course, that none of the nerve elements have been destroyed.

Treatment.—The treatment of any case of myelitis necessitates much patience on the part of the sufferer, because weeks and months may pass before any sign of improvement can be perceived, and much circumspection on the part of the physician, because we are never able to say beforehand how certain measures are going to be borne by the patient, and because what often helps one is harmful to another; hence one must proceed carefully and systematically, and, as it is likely that the course of the disease is going to extend over years, one should always have something new and as yet untried in reserve. If the diagnosis has once been made with certainty, it is our duty to inform the patient in a delicate way of the true

state of affairs, and how seriously his capacity for following his occupation will be interfered with; further, to see that he is properly fed on a nourishing diet and obtain for him as far as possible mental and bodily rest. It is a gross error to recommend such patients, who are easily fatigued and who on the slightest provocation are attacked by all sorts of pains, to take as much exercise as possible, or even to prescribe gymnastics for them.

The electrical treatment is indicated and ought to be begun early. The constant current should be applied near the seat of the lesion (the anode being placed on the tender parts of the spinal column if there be such), the faradic to the peripheral parts, especially the lower legs. Definite rules can not be laid down. It is best to seek information from a reliable text-book —e. g., Erb—and to try which mode of treatment is best borne by the patient, and by which most is accomplished. Tepid baths—84° to 88° Fahr.—three or four times a week for from fifteen to thirty minutes, best taken in the forenoon, usually have a favorable influence, and are, if not of lasting benefit to the patient, frequently productive of at least a transient feeling of comfort. The addition of rock-salt, sea-salt, or lye (one or two quarts) should only be ordered if the patient himself seems to lay much stress on it, as we can not expect any especial effect from them. Neither should we raise our expectations too high when we recommend warm brine baths containing carbonic acid gas, or non-medicated warm baths, or mud baths and the like. Of course every patient, rich or poor, expects us to send him in summer to the springs, but he will gradually find out that the success attained does not compensate for the expense and the trouble which the yearly course at such places entails, and that it is wiser to remain in his comfortable home or to betake himself into the country and enjoy the mountain or forest air somewhere where he can live in peace. The life in modern watering-places is not adapted for a patient with myelitis. Mild cold-water treatment in an intelligently conducted sanitarium (Graefenberg, Nassau, Elgersburg, and others) may well be recommended. All internal medicines (strychnine, silver, ergotine, iodide of potassium, etc.) are of no avail. The treatment of the retention of the urine and the consequent cystitis must be carried out according to strict surgical principles. In the treatment of a compression myelitis we must not forget the necessary extension apparatus, braces,

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etc., for the vertebral column. These means, however, belong to the domain of orthopædic surgery.

Sometimes the effect of a unilateral section of the spinal cord, where we consequently again have a lesion of the gray as well as the white matter, can be observed in those rare instances in which traumatism, a tumor, or the like has rendered the half of the cord incapable of performing its functions. The clinical picture resulting from such a lesion is much more rarely observed than we should be led to suppose from the accounts in the text-books. The disease is called Brown-Séquard's spinal paralysis. It, in short, manifests itself as a motor paralysis on the side of the lesion, and a sensory paralysis on the opposite side. This is explained by the distribution of the fibres, inasmuch as the sensory fibres cross over to the other side soon after their entrance into the cord, while the motor fibres pass upward to the medulla oblongata without crossing (cf. Fig. 156); thus, if, for instance, the lesion be in the right half of the lumbar cord, a paresis of

Fig. 156.—Schema of the Course of the Nerve-fibres in the Spinal Cord. *v*, uncrossed motor fibres. *v′*, uncrossed vaso-motor fibres. *sm*, uncrossed fibres for the muscular sense. *s*, decussating sensory fibres. (After Brown-Séquard.)

the right leg ensues, while the left is anæsthetic; if the lesion is high up in the right half of the cervical cord, the right arm and right leg are paralyzed ("spinal hemiplegia"), and the other half of the body is anæsthetic. The fact that on the side on which there is motor paralysis there is often a hyperæsthesia to be found for certain qualities of sensation—with the exception of the muscular sense, which appears diminished—is explained, according to Brown-Séquard, by the fact that the fibres for the muscle sensibility do not cross over as the other sensory fibres. Above the hyperæsthetic there is an anæsthetic zone, due to

the destruction of the posterior nerve roots. Further, there is an increase of the reflexes on the side affected with motor paralysis, owing to the cutting off of the inhibitory influence, as well as a vasomotor paralysis, manifesting itself by an elevation of temperature. On the anæsthetic side the reflexes are normal; a narrow hyperæsthetic zone (on the trunk) is here also noticeable above the area of anæsthesia.

On the whole, the descriptions which we possess of unilateral cord lesions are of no great practical use, because, as has been stated, the clinical picture just described is but rarely distinct and complete, and may present all kinds of variations (cf. Hoffmann, Deutsch. Arch. f. klin. Med., 1886, 38, 6, where three cases of this class which occurred in Erb's clinic are described).

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II. Spinal Lesions regarded from their Pathological Aspect—Pathological Diagnosis.

I. AFFECTIONS OF THE SPINAL CORD DUE TO DISEASES OF THE BLOOD-VESSELS.

A. Diseases of the Arteries of the Spinal Cord and their Consequences.

The vertebral arteries which arise from the subclavian, and which unite to form the single basilar artery, give off, after having entered the skull, an anterior spinal and a posterior spinal artery by which the spinal cord is supplied with blood. The anterior spinal arteries of both sides unite to form a vessel which runs along the spinal cord in the anterior spinal fissure, while the posterior spinal arteries anastomose freely with each other without, however, completely uniting; the horizontal branches run along the septa. White and gray matter are nourished in the same way, but the capillary network of the latter is much denser than that of the white substance.

The venous blood is collected into two fairly large veins, which are called the central veins of the spinal cord. They anastomose freely among themselves, and are connected with the anterior and posterior spinal veins. From them the venous blood passes into the vertebrals, which empty into the innominate or the subclavian vein. About the diseases of the spinal veins up to the present nothing is known.

1. Spinal Hæmorrhage—Hæmorrhagia (or Apoplexia) Medullæ Spinalis—Hæmatomyelia.

While, as we have shown above, a primary hæmorrhage from the cerebral vessels is one of the most common causes of lesions of the brain, spontaneous hæmorrhages from the spinal arteries are exceedingly rare, and indeed it seems hardly possible that a hæmorrhage could take place into the substance of the cord, so firmly held together as it is by the tough pia mater, without the previous existence of alterations in its consistence; besides, the anatomical conditions of the arteries are such that the blood pressure is decidedly lowered before the blood wave reaches the spinal cord; furthermore, and this is, perhaps, the most important reason for the rare occurrence of hæmorrhage into the cord, miliary aneurisms, which in the brain are the most frequent source of hæmorrhage, are never found here. For these reasons the existence of primary spontaneous spinal hæmorrhages has been absolutely denied, and it has been assumed that in every case changes in the consistence of the cord substance must have preceded. We fully agree with those who believe in their extreme rarity, but, nevertheless, we are of the opinion that under certain conditions primary hæmorrhages actually do occur. Such conditions are: (1) in old persons the coexistence of cerebral hæmorrhages in consequence of arterial disease; (2) the presence of such ætiological factors as excessive muscular exertion (heavy lifting, cutting wood, etc.); (3) the sudden and violent suppression of hæmorrhages in other places (the menses, hæmorrhoids, etc.); (4) the exposure to a sudden marked diminution of atmospheric pressure, as happens to those who follow certain occupations, as, for instance, workers in compressed air in building bridges or winning amber (cf. Hirt, Gewerbekrankheiten im Handbuch der spec. Pathologie und Therapie, vol. 1, 3d edition, reprint, pp. 83 *et seq.*).

The pathological condition is either one of capillary hæmorrhages or of a hæmorrhagic infiltration in which the escaped

blood extends between the nerve fibres along the course of the vessels, or finally we have hæmorrhagic foci, in which the blood coming from the vessels in larger quantities presses the nerve tissue apart and forms a sort of cavity. The focus usually extends in the longitudinal direction of the cord. Hæmorrhage may occur at any level of the spinal cord, and in any portion of the cross-section, and may produce the same changes in its substance as cerebral hæmorrhage produces in the brain, —changes with which we have become familiar in a previous chapter.

Clinically, spinal apoplexy is characterized by paralysis with a sudden onset, sometimes attacking the patient without any premonition and while he is apparently in the best of health; he suddenly sinks to the ground without losing consciousness, and is deprived of the use of his limbs; occasionally prodromata, such as tearing pains, formication in the limbs, may precede for hours or days. The extent and the degree of the paralysis depend entirely on the seat of the hæmorrhage; it may be confined to one half of the body or to both legs or to both arms, or it may take in all four extremities simultaneously. It develops extremely rapidly, and reaches its fullest extent within twenty-four hours. If this is not the case it is not a spinal hæmorrhage with which we are dealing. Pains and rigidity of the back and clonic muscular twitchings are equally constant, as are the bladder symptoms, which are probably never absent in hæmatomyelia. With regard to sensation and the reflexes no general rule can be given, yet an increase of the reflexes immediately after the catastrophe is not exactly rare. Death may occur within a few hours, an event which is especially likely to take place if the hæmorrhage is situated high up. In other cases the patient lives for days and weeks and finally dies from the effects of bed-sores, of a cystitis, etc. Finally, at least relative recovery is not excluded; the patient may either get over the effects of the lesion, or he may be left with motor or sensory disturbances of the most varied kinds. The differential diagnosis between hæmatomyelia and hæmatorrhachis (meningeal apoplexy) has been discussed above. For the treatment we may try the application of ice to the spinal column and the internal administration of ergotine. The success of these measures is always very doubtful, and a careful attention to the nutrition and the cleanliness of the patient should in all cases be considered the thing of most importance.

2. *Embolism and Thrombosis of the Spinal Arteries and Myelomalacia.*

Embolism of the spinal cord, the development of which has been studied experimentally by Panum, is extremely rare in man, probably owing to the smallness of the spinal arteries and the fact that they arise at right angles. The symptoms by which emboli manifest themselves are not definitely known; possibly there is a connection between embolic processes and the so-called choreic movements, but this is still hypothetical.

It is about the same with arterial thrombosis, the independent existence of which is, to say the least, doubtful, but since, as Leyden has pointed out (Rückenmarkskrankheiten, ii, 41), disease of the spinal vessels is extremely common, the occurrence of arterial thrombosis is very easily possible. Not only the inflammatory processes in the spinal cord, which are accompanied by arterial disease, but also the senile changes, which consist in fatty degeneration and thickening of the vessel walls, predispose to it. The necrosis which occurs in the substance of the spinal cord in consequence of arterial obstruction is similar to that described on page 247 as occurring in the brain substance. The condition of softening is called myelomalacia.

3. *Endarteritis (syphilitica).*

That the spinal arteries participate in the process which Heubner has shown to occur in the cerebral arteries (page 252), according to competent observers, does not seem to admit of doubt. It is equally certain that this process plays here a relatively smaller *rôle* than in the brain. Heubner himself, Knapp, Leyden, and others have reported interesting observations bearing on this, and it seems that an endarteritis obliterans in the spinal cord leads either to a myelitis or a multiple sclerosis. Rumpf, in his excellent treatise on The Syphilitic Diseases of the Nervous System (page 349), has published in full a very interesting case of syphilitic disease of the spinal arteries, which was followed by a similar report by Knapp (Neurol. Centralblatt, 1885, 21), and another by Graeff (Arch. f. Psych. und Nervenkr., 1882, xii, 3). There are, however, only comparatively few cases to be found in the literature, and, in almost all, syphilis of the brain coexisted with syphilis of the spinal cord, and endarteritis obliterans was almost always

demonstrable in the brain as well. Lately two interesting cases of Schmaus have appeared (Deutsch. Arch. f. klin. Med., 1889, vol. xliv, 2, 3, p. 244). In one of them the syphilitic affection took the form of an arterial disease, running a subacute course with hyaline fibrous thickening of the intima and simultaneous inflammatory infiltration of the whole vessel wall, which was followed by an irregular disseminated patchy sclerosis of the white matter, a marginal sclerosis, and a degeneration of Goll's columns in the cervical cord. That the degeneration of the nerve parenchyma was attributable to the low state of nutrition in consequence of diminution in the blood supply seemed beyond doubt. As for the symptoms, sensory disturbances (pains, paræsthesias, hyperæsthetic zones) and motor disturbances (at first fatigue and finally complete paraplegia), furthermore incontinence of the urine and fæces, constituted the clinical picture. In the second case a syphilitic degeneration of the vessel walls combined with a poliomyelitis was found. With our present knowledge we must content ourselves with diagnosticating a diffuse affection of the spinal cord, a transverse myelitis, a tumor, and the like. The diagnosis of a syphilitic disease of the arteries must be made with reservation during life, and must only be assumed when the luetic history is certain.

4. Dilatation of the Spinal Arteries.

We know very little about aneurisms of the spinal arteries. Besides the case reported by Liouville, which is also quoted by Leyden (*loc. cit.*, 2, p. 42), none can be found in the literature. The question, therefore, whether syphilis may give rise to aneurisms here can not be answered. It is possible that bodily exertion has a predisposing action. A symptomatology and a therapy do not exist for aneurisms of the spinal cord.

5. Neuroses of the Spinal Arteries.

The vaso-motor nerves of the spinal arteries behave just like those that supply the cerebral vessels, and upon whether they are in a state of irritation or in one of paralysis the amount of blood in the spinal cord depends. But easy as it is to demonstrate hyperæmia and anæmia of the cord in the cadaver, it is difficult, on the other hand, to say in what way changes in the amount of blood in the spinal cord influence the health of the patient, and whether a greater or lesser fullness of the ves-

sels, or frequent fluctuations between the two, are attended
with any marked symptoms. All views on this subject are
entirely hypothetical. The pathological changes in the spinal
cord, due to an artificial transient anæmia produced by liga-
tion of the abdominal aorta, Spronek (Arch. de physiol. norm.
et pathol., September 1, 1888, xx), following out the early re-
searches of Brieger and Ehrlich, has lately demonstrated, with-
out, however, throwing any further light upon the clinical
symptoms caused by spinal anæmia.

Since the time of Peter Franck (1791) there has been a wide-
spread opinion that hyperæmia of the spinal cord can give
rise to a number of symptoms of irritation, some of which
being motor, some sensory, together make up the clinical pic-
ture of what has been described as spinal irritation. Although
we are at present not able always to sharply identify "spinal
irritation" as a clinical entity, and although so much ignorance
has undoubtedly been cloaked by this term, especially of late
years, still we can not dispense with it where we wish to de-
scribe a certain combination of symptoms. The patients, who
as a rule are females belonging to the best classes of society,
complain of an occasional feeling of fatigue and of pains in
the back, which are intensified by the erect posture. Walking
becomes difficult, and the gait is that of an old person; they
walk with a bent back and take each step with care. Painful
sensations, paræsthesias, formication, and numbness in the lower
extremities are complained of. The functions of the bladder
are more or less disturbed; often there exists a uterine catarrh.
The patient is low-spirited, and has a tendency to hypochon-
driacal notions. On examination, we find the reflexes either
normal or exaggerated; sensibility is somewhat affected, and
disseminated anæsthetic plaques are demonstrable. A certain
tenderness over the vertebræ is almost always noted; it is
usually more pronounced in the lumbar and dorsal than in the
cervical region. The course of the disease is eminently chronic;
often months and years pass before, notwithstanding all thera-
peutic measures, any decided improvement occurs, and those
unfavorable cases in which the patient finally becomes bed-
ridden and, after having been for years affected with paresis
or paralysis, falls at last a prey to an intercurrent malady, are
by no means exceptional. A cause is often looked for in vain.
It is true, overexertion or sexual excesses may sometimes in-
directly give rise to the disease; it is true, we are at times

justified in blaming to some extent the immoderate indulgence in tobacco, but more frequently all such factors are wanting, and we are forced to attribute it to a congenital weakness of the nervous system.

The diagnosis is not always easy, and many a myelitis probably has been taken for spinal irritation, or more frequently, perhaps, spinal irritation has been mistaken for myelitis. Only in the cases, which are not rare, in which remissions lasting for months occur, so that the patients begin to have hopes of a speedy and complete recovery, the diagnosis is not doubtful. Sometimes it may not be possible to make it at all.

The treatment should be local and general. The former consists in the early and energetic use of the Paquelin cautery and of the constant current (descending); the latter in the use of tepid baths and tonics. Yet often all measures are fruitless, and it is advisable to be very guarded in giving an opinion with regard to the duration and probable outcome of the disease.

That a chronic anæmia of the substance of the spinal cord may give rise to a paralysis, especially of the lower extremities, which may last for years, seems probable according to the thesis of Meunier (Paris, 1886), yet nothing certain can be said, especially as in the cases in question it may be difficult to exclude hysteria.

Just to what class we must assign those instances of paralysis, described more especially by Russell Reynolds, which depend on the imagination—whether they are due to functional disturbances in the spinal cord, or whether, under the influence of psychical activity in consequence of auto-suggestion, a disease of the whole nervous system develops—is not known.

The various disturbances in the sexual functions—for instance, the impotentia coeundi, which is quite a common manifestation of a functional disturbance of the spinal cord in young and middle-aged men—we shall enlarge upon in the chapter on neurasthenia.

II. INFLAMMATORY PROCESSES IN THE SUBSTANCE OF THE
SPINAL CORD.

1. Purulent Myelitis—Abscess of the Spinal Cord.

While circumscribed pus formations in the brain substance are by no means rare, the formation of an encapsulated pus focus in the spinal cord is one of the greatest exceptions. Although Leyden succeeded in producing such foci experimentally in dogs, the clinical observations in man are so few that it is impossible to formulate from them a definite symptomatology. Pathologically, it is interesting to note that Ollivier and Jaccoud (quoted by Leyden, *loc. cit.*, ii, 205) have seen abscesses which varied in size from that of a bean to that of a hazel-nut and were filled with a greenish-white pus. They were situated some in the cervical, some in the dorsal cord. The symptoms, on the whole, were those of a grave, acute softening. In a recent article of Ullmann (Zeitschr. f. klin. Med., 1889, xvi, 2, page 39) an interesting discussion on spinal abscesses and an exhaustive collection of references will be found.

2. The Non-purulent Myelitis.

Inflammatory processes in the spinal cord are very frequent. In the majority of cases they are of a chronic type and less often acute. With reference to their situation we have already stated that they may implicate the white as well as the gray matter.

A. The Acute Form.

As we said on page 443, we have in acute myelitis a process which is characterized by the death of the nerve elements and a secondary increase of the connective tissue. In the acute stage a change in the consistence of the cord takes place; the parts become softened and appear swollen and infiltrated. Sections of the cord are not so distinct, and the demarcation between the white and gray matter is less sharp. The color may be reddish (hæmorrhagic), yellowish-red, rusty brown, whitish, or of any intermediate shade. The extent of the process of softening varies. It may be spread over the whole or only a part of the cross-section, and may extend longitudinally for a greater or less distance. Sometimes disseminated foci are

found not only in the cord, but can also be demonstrated in the brain. We shall speak about these later.

In exceptional cases, which are difficult to explain, absolutely no changes were found at the autopsy, although the course of the disease seemed in every way to suggest an acute lesion. These patients were, for the most part, young and up to the time of their illness vigorous persons. After a short prodromal stage, in which there was headache and some fever, they were attacked by a flaccid paralysis of both legs, which developed in a few days. To this was added in a very short while paresis of both arms, so that the helplessness of the patients reached an unusual degree. The condition of the reflexes and the electrical excitability varied in the few cases reported up till now. According to the records, the functions of the bladder and rectum as well as sensibility remained normal. The prognosis is very doubtful. Sometimes bulbar symptoms appear and the patient dies within from eight to fourteen days after the onset. Sometimes the course is more protracted and some improvement occurs, which, however, is never complete. The affection, which presents the clinical picture just described, is called Landry's paralysis (1859), *paralysie ascendante aiguë*, acute ascending spinal paralysis, although it is not definitely known whether we actually have to deal with a spinal affection and not rather with a very acute infectious peripheral neuritis. Until we possess the results of a larger number of anatomical examinations it is no use to theorize any more about the nature of the disease (cf. Schultze, Schwarz, Bernhardt, etc.).

With regard to the ætiology of acute myelitis, all that is necessary has been stated on page 447. The influence of certain poisons in causing changes in the spinal cord, more especially the so-called intoxication paralyses, will be discussed in the chapter on the diseases of the general nervous system. It is possible that Landry's paralysis, about which so little is known, can be caused by infectious diseases, for instance by whooping-cough (Möbius). Of great interest is the communication of Curschmann (Verhandl. des fünften Congresses für innere Med., Wiesbaden, 1886, page 469), in which he speaks of a case of acute ascending paralysis where at the autopsy typhoid bacilli were found in the spinal cord. Further, it has been known to follow working in great heat (Prahl, Hosp. Tidende, 1876, 2 R., iii). Often no ætiological factor can be made out.

The symptomatology, diagnosis, and treatment of acute myelitis have been discussed on pages 444 to 449.

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B. The Chronic Form.

Chronic myelitis is much more commonly observed than the acute form. It is characterized by the death of the nerve elements and a consequent increase of the connective tissue, which gives to the tissue a peculiar firm appearance and consistence—sclerosis. That this sclerosis is frequently confined to certain nerve tracts, giving rise to the so-called "system-diseases," we have pointed out above on page 435. On page 444 will be found some account of the sensory, motor, and trophic changes which are found in these affections. It is in all cases of great importance to look to the condition of the reflexes, as this may have a decisive significance for the diagnosis. The disturbances of the bladder and rectum in chronic myelitis and the treatment of the disease have been discussed above.

III. Spinal Tumors.

Pathological Anatomy.—In the spinal cord, just as in the brain, the glioma is relatively the most frequent form of primary neoplasm. What has been said on page 288 about its development holds good here also. The cervical and dorsal part of the cord seem by preference to be the seat of the glioma. Sarcomata, which from the onset present a sarcomatous nature, and gliosarcomata—that is, gliomata with unusually marked proliferation of cells—have been observed, although but rarely as primary tumors. Angiomata, small reddish, probably congenital (Virchow) foci, have been found, and Gan-

guillet has observed a cylindroma in the lowest portion of the spinal cord. Solitary tubercles and syphilomata are much rarer here than in the brain. Carcinomata usually start from the vertebræ and afterward spread to the spinal meninges. The secondary changes are, of course, not nearly so well marked here as those we find in the brain, since the spinal cord is in a position to offer greater resistance to the growth that presses upon it. Only when the tumor has reached some considerable size—e. g., that of a hazel-nut—do symptoms analogous to the so-called "indirect symptoms" in the brain make their appearance.

Ætiology.—The ætiology is absolutely unknown. Though in certain cases traumatism has been made responsible for gliomata in the spinal cord, we are still in complete ignorance about the real cause, as we confessed ourselves to be when treating of their occurrence in the brain. The influence of age and sex here is the same as in tumors of the brain.

Symptoms.—If a patient complains of persistent pains and stiffness in his back, if at the same time there are found sensory disturbances in the form of paræsthesias, circumscribed areas of anæsthesia, and motor disturbances in the form of slowly but steadily progressing paralysis of one or more extremities, the suspicion, that a tumor of the meninges or of the cord itself exists, is justifiable. The likelihood is greater if other spinal affections can be excluded and if occasional remissions in the progress of the disease can be noted. It is true the diagnosis of spinal tumors always remains a very difficult thing, and at times, for instance, we may not be able to definitely differentiate a myelitis from a spinal tumor. This is easily understood if we consider that spinal tumors may give rise to the most varied clinical pictures, according to their position and size and according to the greater or lesser involvement of the white or gray matter. There is no doubt but that a tumor of the spinal cord may give rise to symptoms of a compression myelitis, of tabes, or of a myelitis, and that if it be confined to one side it may produce the symptoms of a Brown-Séquard's paralysis. Roth (cf. lit.) claims that loss of the temperature sense is frequently observed in spinal glioma, and that this, combined with analgesia, paresis, and muscular atrophy, is sufficient to settle the diagnosis. The considerable material which Roth has at his disposal makes his monograph

very valuable. It is only to be expected that vaso-motor as well as trophic symptoms should be found. To interpret these must be left to the physician's skill in diagnosis, upon which so much depends in the recognition of tumors of the cord. Sudden changes in the spinal symptoms, temporary remissions, then again sudden changes for the worse, should all be made to have their proper diagnostic value. In cases of well-marked paraplegia dolorosa, where we have tearing pains in the small of the back, radiating into the extremities, together with atrophy of the muscles of the lower legs, we should always think of one or several tumors of the cauda equina. In these cases contractures of such severity sometimes develop that the heels touch the buttocks (Leyden).

Prognosis.—The prognosis depends upon the nature and the seat of the tumor, although the ultimate outcome is always unfavorable. If the growth be benign and be situated in a relatively indifferent area, the patient may last for years, and even enjoy periods so free from discomfort that he may deem a recovery quite possible.

Treatment.—The treatment can only then be of any avail if surgical interference—that is, excision of the tumor—is possible. A case of this character has been reported by Gowers and Horsley. An oval myxoma which had pressed upon the cord was, after removal of the spinous processes of the third, fourth, and fifth dorsal vertebræ, excised, and the patient recovered completely. Bruce and Mott (cf. lit.) diagnosticated *intra vitam* a tumor which, originating in the fifth left dorsal nerve, pressed upon the middle of the dorsal part of the spinal cord; the patient presented the symptoms of a compression myelitis and died. At the autopsy softening with ascending and descending degeneration was found. The authors regret in their paper not having decided upon an extirpation of the tumor.

All other means are fruitless. If there is any suspicion that the case is one of syphilis, inunctions with mercury ought to be given a trial.

Appendix.—*Parasites in the Spinal Cord.*

About parasites in the spinal cord we may look in vain for information in the text-books, probably because their occurrence is very unusual, and also because, if they are present, they may not give rise to any symptoms. But here we ought

to make at least a short mention of the cysticerci which have been found not only in the brain, but also in the spinal cord. Leyden only devotes a few words to this subject in his Klinik der Rückenmarkskrankheiten (1, 445): "Still more rare (than the cysticerci in the brain), and as yet of no clinical significance whatever, are the cysticerci which may develop . . . in the annexes of the spinal cord, etc." I have shown in a case which came to my notice, and which I have reported (cf. lit.), that cysticerci of the spinal cord—there were fifteen or twenty in the dural sac—may give rise to symptoms simulating those of tabes; some clinical significance has, therefore, to be attributed to them. That the symptoms of spinal irritation, which are associated with such parasites in the cord, are not to be attributed to the increased intraspinal pressure, but that they are of a reflex nature, seems beyond doubt. To diagnosticate *intra vitam* the existence of intraspinal parasites is only possible in exceptional cases—as, for instance, if the patient is a butcher by trade, or if his frequent indulgence in raw meat gives rise to the suspicion of cysticerci; but even in the most favorable cases the diagnosis can not claim to be more than conjectural.

Almost as rarely do we find echinococci in the vertebral canal. A case of this nature, however, which is of a great deal of interest, has been published by Jaenicke (cf. lit.). An echinococcus, which had existed in the subpleural tissue in the region between the ninth and the twelfth dorsal vertebra, penetrated into the vertebral canal, and, owing to the compression thus exerted upon the spinal cord, gave rise to such characteristic symptoms that the diagnosis *intra vitam* was to a certain degree justifiable.

IV. Congenital Diseases—Hydrorrhachis—Spina Bifida.

To a collection of fluid in the skull we have given the name hydrocephalus (page 305); similarly a like collection in the vertebral canal we call hydrorrhachis, and specify two forms of the disease—the hydrorrhachis externa and interna—according as the fluid is situated in the meshes of the pia, or between the meninges, or, on the other hand, in the interior of the spinal cord. In the latter case we find a dilatation of the central canal, which is either uniform throughout or beaded.

At the autopsy we not rarely, instead of the normal central canal, the ordinary diameter of which measures from one

tenth to one millimetre, find a canal with a diameter of two, five, or even ten millimetres (" hydromyelia "), or alongside of the usual canal abnormal cavity formations (" syringomyelia"); during life, on the other hand, such conditions are by no means often correctly recognized. The practical significance of these abnormities is not great, as for one thing the signs during life are so uncertain and changeable that a correct diagnosis has almost to be regarded as accidental, and, secondly, because the disease, even if recognized, is not at all accessible to any treatment. Notwithstanding this, it is of course desirable that the present state of our knowledge of hydromyelia and syringomyelia should be given briefly here, and that attention should be drawn to the symptoms most commonly observed in these affections.

With reference to the origin of hydromyelia it is especially abnormities in development which we have to deal with, and more rarely does the influence of pressure—e. g., a tumor in the posterior fossa of the skull—come in. For the development of syringomyelia central gliosis, with secondary disintegration and cavity formation, is said to play an important part. These cavities may connect with the fourth ventricle, and extend through the medulla oblongata as far as the conus terminalis, and on a cross-section two or more lumina may be seen. They are of variable lengths, and are, as a rule, situated in the lower cervical and in the dorsal cord, and especially in close proximity to the central canal, sometimes also in the posterior horns. Their width varies from a half to ten millimetres; their contents are sometimes watery and thin, sometimes milky and viscid. The relation of the central canal to these cavities varies so much that no rule can be given on this point. In certain instances it remains intact in its whole length.

Among the clinical signs there are, more especially, three which should arouse a suspicion of syringomyelia—namely, (1) localized muscular atrophies, more especially in the upper extremities, be it in one or in both; (2) a widespread, non-typical hemianæsthesia (more especially analgesia); and (3) trophic disturbances of the skin and the deeper parts (whitlow, phlegmon), and also of the bones, which easily break (Schultze, cf. lit.). The muscular atrophy is always associated with a more or less pronounced paralysis, as we might expect in lesions of the gray anterior horns. In such instances amyotrophic lateral sclerosis or peripheral neuritis may suggest

itself as a diagnosis. The sensory changes we shall understand if we remember that the posterior commissure, Goll's columns, and the posterior horns are preferably the seat of the affection. In a case of Schüppel (Arch. d. Heilk., 1874, xv, p. 44) general anæsthesia was found. On the other hand, we ought not to omit to state that in many instances all sensory changes are absent, so that even this symptom is far from being pathognomonic. The condition of the reflexes varies much, as does also the appearance of trophic and vaso-motor disturbances under the form of exanthematous eruptions, vesicles, ulcerations, erysipelatous swellings, etc., which are sometimes present, sometimes absent. All the cases observed up to 1887, and two new ones that had come under her own observation, have been collected and analyzed by A. Bäumler (cf. lit.).

Somewhat related to these dilatations of the central canal are those congenital cystic tumors which, penetrating through the walls of the vertebral column, make their appearance below the skin on the back. If the cyst, the size of which may vary from that of a walnut to that of a man's fist, is situated in the middle line over the sacrum, it is called a sacro-lumbar myelo-meningocele, or spina bifida. The skin over the tumor is normal; below it are found the bulging dura and arachnoid. The contents of the sac, which has sometimes smooth, sometimes rough walls, are as clear as water and identical with the cerebro-spinal fluid. The spinal cord is attached to the inner wall of the sac by a broad base, or at its point of entrance divides into several strands which pass directly into the wall of the cyst. The coexistence of a hydromyelus with a spina bifida, the former causing an atrophy of the substance of the spinal cord and a communication between the central canal and the cavity of the spina bifida, is a rarity.

In a child born with spina bifida we find, as we stated, in the middle of the back, in the region of the sacrum, a soft, doughy, elastic, not rarely fluctuating, tumor, which can be made smaller by pressure. The position of the child influences the condition of the sac. It is tense in the erect posture; when the child lies down it becomes flaccid and soft, a fact which must be referred to the communication usually existing between it and the cranial cavity.

Although the child thus affected may at first develop fairly normally, his life is endangered from the first moment. Not

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only does the pressure exerted upon the spinal cord by the increasing tumor lead to motor and sensory changes, as well as bladder symptoms, but there exists a constant menace to life which the rupture of the sac would entail, an accident which is favored by the gradual thinning of the overstretched skin. Such a rupture is almost always followed immediately by convulsions and death.

The ætiology is not known. Possibly we have to do with a developmental anomaly, possibly, as Virchow believes, with an early formation of partial hygromata (hydromeningocele).

The treatment of spina bifida belongs to the domain of the surgeon. We may either endeavor to get rid of it by repeated puncture and subsequent injections of a solution of iodine in glycerine (Morton), or we may content ourselves with methodical compression. The whole treatment, however, should, owing to the danger of a meningitis, always be undertaken with the greatest caution.

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DISEASES OF THE GENERAL NERVOUS SYSTEM.

IN diseases of the general nervous system, the brain and spinal cord and the nerves which come off from them all share in the morbid process, yet the extent to which the different parts are implicated varies very much in different cases. Sometimes, in so far as clinical symptoms would lead us to conclude, the trouble lies mostly in the brain, sometimes in the spinal cord. In the latter case, again, we may have a more marked implication of the substance of the cord itself, lesions of certain tracts, or perhaps the lesions of the peripheral spinal nerves may come more into the foreground. Between such extremes there exist manifold intermediate forms, but to say much about the course of these diseases which would be applicable to all becomes all the less possible because a second point has to be taken into consideration, namely, whether, and if so how far, the whole organism shares in the disease of the nervous system. This participation varies in many ways, and there are diseases of the general nervous system which can exist for years without any serious implication of the general organism; while there are others, and these are far more numerous, in which sooner or later the nervous disease grows, as it were, into a general disease, in which the organs which have to do with digestion, circulation, secretion, excretion, sometimes even respiration, are affected more or less seriously. That the course of the disease and the prognosis must sometimes be materially influenced by this we need not say, and one rule is forcibly impressed upon us by such cases, a rule which must never be lost sight of by the physician, viz., never in a case of disease of the general nervous system to content ourselves with an examination of the nervous system, but to remember

that the same care must be devoted to all organs without exception. This rule, self-evident enough to the conscientious observer, we have dared to emphasize again because it is more especially in nervous diseases that it has been allowed to fall into abeyance. As to the pathology of the diseases which we are about to consider, our knowledge is unfortunately in many respects very scanty, and in many of them no lesions at all have been found after death, although the assumption, that in the majority of cases some anatomical changes, macroscopical or microscopical, must have been present, would appear to be justifiable. Only for certain of the diseases in this category, as tabes, dementia paralytica, multiple sclerosis, and certain chronic intoxications, have anatomical changes been demonstrated, and even here we are not always clear about their significance. Again and again it has happened that after an accurate analysis of the clinical symptoms a diagnosis has been made *intra vitam* and this and that anatomical change has been reckoned upon with certainty, and then at the autopsy the whole nervous system was found to be absolutely intact. Among such cases we may mention that of Westphal, where a multiple sclerosis was diagnosticated ; that of Killian, a supposed chronic myelitis ; and a certain case of ophthalmoplegia externa progressiva of Eisenlohr. Instead of the pathological condition expected, the brain, spinal cord, and their nerves were found to be absolutely normal. On the other hand, it has happened that where hysteria, epilepsy or chorea had been diagnosticated and one had prophesied most confidently that the condition of the central nervous system would be found normal, the autopsy has shown extensive changes—multiple foci in the spinal cord or in the brain cortex, recent or old areas of softening, etc. To such errors even the most reliable observer is exposed, and it is just the man who has observed accurately the greatest number of cases and assisted at the post-mortem examination of them who will be most cautious in his diagnosis and in his prophecies as to what will probably be found at the autopsy.

Uncertain then as is the condition of our pathological knowledge in these cases, still, if we decide to treat of diseases of the general nervous system not simply one after the other, but to adopt some arrangement into groups, it is best to base this in a general way on the conditions which we find after death, and to distinguish two classes, the first consisting of

nervous diseases in which up to the present time no anatomical changes have been demonstrated at the autopsy, diseases which we therefore call functional neuroses; the second, those diseases which are always associated with known anatomical changes.

PART I.

DISEASES OF THE GENERAL NERVOUS SYSTEM WITHOUT ANY RECOGNIZABLE ANATOMICAL BASIS.

"FUNCTIONAL NEUROSES."

THE affections coming under this head are much more numerous than those for which an anatomical basis is known. Their number justifies the desire to divide these, again, into groups, an undertaking, however, which presents the greatest difficulties, because any classification must always appear to a certain extent forced. Inasmuch as no pathological anatomy enters into the question, we have here to help us only the ætiology and the symptomatology. From the former we can expect nothing, since, as we shall see, the cause is sometimes utterly unknown, at other times so uncertain and variable that it does not warrant us in assigning a particular disease to any particular group.

As for the second, the symptoms also present so many variations that it is difficult to classify them; and even if we take into consideration the fact that in some diseases the symptoms belonging to the cerebrum, in others those belonging to the spinal cord predominate, a distinction can not always be arrived at, since some symptoms, such as certain motor and sensory disturbances, may be of cerebral just as well as of spinal origin, and in a given case it may be absolutely impossible to determine with which kind we are dealing. It only remains, then, if we are not willing to renounce all attempts at classification, to come back to the question as to what influence the neurosis exercises upon the general condition of the patient and to determine whether, and if so, to what extent, the general organism shares in the nervous disease. Thus we shall find that some of them (though these are the few) disappear after running a shorter or longer course without leaving behind them

any bad effects, or again last for years, during which time the patient feels fairly well, or, at all events, shows no serious general symptoms; while there are others that are characterized not only by their long duration, their obstinate resistance to treatment, and their tendency to recur, but also by the baneful influence which they exert on the general system. The former, for the sake of brevity, we shall designate as mild, the latter as grave neuroses, although we do not mean to exclude the possibility that now and again among the ones we call mild we may not encounter a serious disease running a tedious course, while among the grave forms we may have cases by no means as severe as usual. We would also especially state that we are in no wise satisfied with our classification, and look upon it only as a temporary makeshift, to be superseded as soon as some better method has been discovered.

FIRST GROUP.

NEUROSES WHICH ARE WONT TO RUN THEIR COURSE WITH-
OUT ANY ESSENTIAL IMPLICATION OF THE
GENERAL ORGANISM.

CHAPTER I.

CHOREA—CHOREA ST. VITI—ST. VITUS' DANCE—BALLISMUS—MELAN-
CHOLIA SALTANS—SYDENHAM'S DISEASE.

By the term chorea in general we mean the occurrence of
peculiar irregular movements entirely beyond the control of
the patient. They appear in the upper extremities and in the
face, as well as, though to a lesser extent, in the lower extrem-
ities and in the trunk. They only attack the voluntary mus-
cles and may persist for days, weeks, and even months unin-
terruptedly, except during sleep. If these movements, as is
frequently the case, are confined to one side only, to one half
of the face, to one arm and the corresponding leg, we speak
of a hemichorea. The distinction which is made in some of
the older books between chorea major and chorea minor has
become superfluous, since the symptoms which were formerly
described as constituting the clinical picture of chorea major
do not represent an independent disease, but belong to the
domain of hysteria. Hence we can also dispense with the
designation " chorea minor."

The " choreic " movements may appear independently
where it is impossible to find any coexisting symptoms of
another disease, or they may be no more than symptoms of
another affection, be it of the brain or spinal cord. Our exam-
ination will have to decide between these two possibilities.
We shall first call attention to the idiopathic, genuine chorea,
and we need hardly say that only this form is to be regarded
as a mild neurosis in the sense pointed out above.

Symptoms.—To describe the choreic movements in detail

is not easy, because they present very many varieties in degree and extent. In the relatively severe cases all the muscles participate, the head is thrown about and shaken, the neck is twisted, the forehead is wrinkled and smoothed, the eyelids closed and opened, and the eyeballs rolled around. The facial muscles, including those of the lips and the mouth, take part in the movements, thus giving rise to the most varied expressions —e. g., those of terror, anxiety, or joy—according to the particular muscles most strongly affected. Hasse states that the tip of the nose may be moved, though I myself have never seen this. Very conspicuous are the movements of the tongue muscles, since they interfere with speaking, chewing, swallowing, and with the protrusion of the tongue, which in the worst cases become entirely impossible. The implication of the muscles of the trunk gives rise to rotatory and other involuntary movements of the body ; the patient rises and falls down again, and may work himself into the most peculiar and marvelous positions ("*folie des muscles*").

In the great majority of cases the movements do not take place in the way we have described, except, perhaps, the twitchings of the face, but are confined to the upper extremities, or are at any rate most marked here. Shoulders, arms, hands, and fingers are constantly in motion, the affected muscles twitch, the arms are extended and flexed, the fingers spread apart, and so forth. A similar restlessness is observed in the muscles of the thigh and calf, the feet are alternately lifted, the toes moved, etc., although the lower extremities are generally attacked to a lesser extent. Sometimes the movements are gone through with lightning quickness, in which rare instances the name chorea electrica is justifiable. In milder cases the patients may at times be able to remain perfectly quiet, and only slight twitchings in the arms, the fingers, perhaps also in the facial muscles, will betray the existence of the disease. It is a characteristic feature of idiopathic chorea that all movements entirely cease when the patient is asleep, although going to sleep may be rendered somewhat difficult. Once asleep, however, such patients rest quietly, and are not disturbed by any muscular unrest.

That the intended movements are influenced by the pathological ones goes without saying, and it is quite possible that at a time when the disease is still at its beginning and has not yet been recognized, but is already exerting its influence upon

the voluntary movements, the patient may be simply regarded
as awkward and clumsy. If this happens to children who have
to write in school, or recruits who have to drill and learn the
different manipulations, much unpleasantness for the patient
may arise from this condition, which might have been avoided
by a careful examination by a physician. Generally the vol-
untary movement is normal in its first phase, but soon the
muscles begin to be seized by the spasms and the patient is
not able to carry out the movement intended. This is noticed
in dressing or eating, or in other ordinary actions of daily
life, but most of all is it seen in writing, playing the piano, or
in the performance of other movements requiring a high de-
gree of co-ordination, and can even be marked if we ask the
patient to put out his tongue.

His apparent awkwardness excites the patient very much,
and the more he tries to execute the intended movement, the
more he tries to govern his unmanageable muscles in the usual
manner, the less he succeeds and the more he is annoyed by
the involuntary movements. Only a few particularly well
disciplined patients are, at the height of the disease, able to
keep their muscles for a few moments at absolute rest. The
reflex and automatic movements are not interfered with.
Protective movements are performed as by healthy persons;
coughing and sneezing are done normally; neither do the car-
diac or respiratory movements suffer.

The sensibility is in no way interfered with. Tenderness
over the spine may be present, although not regularly. Other-
wise nothing abnormal can be noticed in the domain of the
sensory nerves. It is remarkable to note that there is no sense
of fatigue, which we certainly should expect after such excess-
ive muscular action. The body temperature and the urine
remain normal throughout the disease if no complications are
superadded. On the other hand, the psychical condition of
the patients, especially if they be young people, undergoes
more or less marked changes, which constitute a prominent
feature of the disease if the course be prolonged; children who
have up to this time been kind, obedient, diligent, and willing,
become willful, peevish, and spiteful; although learning nicely
and without difficulty and making good progress in school be-
fore they had any symptoms of chorea, they become slow at
grasping and understanding what they are taught; the easiest
things must be repeated and impressed upon them, and often

enough they are forgotten again in a few hours. If, and this is not rarely the case, an impediment in speech is added in consequence of the choreic movements of the tongue, the children become wholly unfit for school. It is at this time no longer necessary to advise keeping the child at home, since the teachers themselves will no longer permit it to attend. The influence of the disease upon the psychical functions is generally much less marked in adults.

In the idiopathic uncomplicated chorea the described manifestations persist usually for several weeks with varying intensity. From the onset to the cessation of the disease from sixty to ninety days may elapse (sixty-nine days, Sée; eighty days, Jürgensen; eighty-nine days, Riecke), yet, as we shall show later, the treatment is not without influence upon its duration. By far the most frequent issue is recovery, although the possibility of a relapse is by no means excluded, and in giving a prognosis this feature should be taken into account. Death from chorea is a very uncommon event, and only may occur in very weakly children or when complications arise; otherwise we may in most cases give with a clear conscience a good prognosis.

Complications.—But it is just these complications and the relation that chorea bears to other diseases which deserve so much attention, more particularly as this relation is to a great extent still obscure. In the first place articular rheumatism must here be mentioned, the connection of which with chorea everybody knows, but which, however, is not interpreted by all authors in the same manner. While the French writers especially, among them Sée and Roger, regard rheumatism as an almost regular precursor of chorea, in Germany there is much diversity of opinion on this point. Several authorities (Lebert, Eichhorst, Strümpell) only state that the two affections are relatively frequently found together; others, with Brieger, draw attention to the alternating appearance of the two (Berliner klin. Wochenschrift, 1886, xxiii, 10); again others (Henoch, Litten) look upon rheumatism as " the most important and best founded cause of chorea "; still others, in contradistinction to the rest, deny the existence of any connection between the two affections (Romberg, v. Niemeyer, Prior). However obscure this association may be, to deny it absolutely would be to set facts at defiance. According to our own opinion, we have to deal with a common noxious agent, an infection which,

if chiefly localized in the brain, gives rise to choreic move-
ments, while if it affects the joints it causes acute rheumatism
in them. Most probably, we may almost say unquestionably,
it is the same infectious material which, if affecting the heart,
produces endocarditis and myocarditis, for chorea is as fre-
quently connected with valvular disease of the heart as with
articular rheumatism, though the one relationship is as obscure
as the other.

If chorea, or, we had better say, if certain forms of chorea
are actually to be traced to an infection, we can not be sur-
prised if choreic movements are found to appear after other
infectious diseases—e. g., whooping-cough, typhoid fever, diph-
theria, or cholera.

The possibility that chorea has some connection with epi-
lepsy can not *a priori* be thrown aside. I have twice had occa-
sion to observe children who up to the age of puberty had re-
peatedly suffered, as it seemed, from genuine chorea, and who
afterward became subject to epileptic attacks. It is true the
tongue was not bitten in these paroxysms, but otherwise all
the signs of a classical epilepsy were present, not excluding the
aura. A later communication of Marie (Progr. méd., 1886, xiv,
page 39), in which the occurrence of ovarian hyperæsthesia in
the course of chorea is mentioned, led us to the idea that pos-
sibly the above-mentioned attacks were of a hysterical nature
and to question whether there may not be certain forms of St.
Vitus' dance which could be designated as hysterical.

Lastly, those very rare cases of tropho-neurotic disturb-
ances in chorea are of interest; thus, bald spots on the skull
(Escherich, Mitth. aus der med. Klinik zu Würzburg, 1886, ii),
or in places absence of pigment in the hair or the skin, were
noted (Möbius, Schmidt's Jahrb., 1886, vol. ccix, page 251).
How these are brought about we are utterly unable to explain.

Diagnosis.—It is usually not difficult to recognize chorea if
we remember that young patients of the female sex, who are
often also anæmic, form the largest contingent of the cases,
that the twitchings chiefly affect the upper extremities and the
face, and that they are entirely independent of the will of the
patient. Their disappearance also during sleep is an important
point, and this fact by itself would distinguish them from the
athetoid movements. These latter, possibly the twitchings of
the tic convulsif, the tremor of paralysis agitans, the shaking
movements of the intention tremor of multiple sclerosis, finally,

certain muscular spasms, which Leclerc and Royer (cf. lit.) have designated as pseudo-choreas, must more especially be taken into consideration, but they ought never to render the diagnosis really difficult.

Pathology.—Our knowledge of the pathology of idiopathic, uncomplicated chorea is very imperfect. Repeatedly capillary emboli have been found at the autopsy in the thalamus and the corpus striatum, often they could not be demonstrated. The experiments of Money on guinea-pigs and dogs (Lancet, 1885, 1, page 985) would indicate very decidedly that chorea can be caused by capillary emboli. Their mode of action, however, remains unexplained. The objections which Litten has raised against the embolic theory, that the demonstration of embolic processes in ordinary cases of chorea is not proved, and that in spite of the diversity of the localization of the foci of softening in the brain the clinical picture is always the same, can not be regarded as convincing.

The communication of Flechsig, who in the two inner anterior segments of the lenticular nucleus, but nowhere else, found small bodies in the lymph sheaths of the vessels, some of which were larger, some smaller than blood-corpuscles, has since been neither confirmed nor overthrown. "Their arrangement resembled that of glandular structures; they were strongly refractive, very firm, and almost like chalk, although they contained no lime. In alkalies they slowly swelled." Though their chemical nature is unknown, they resemble in the main that material which von Recklinghausen has termed "hyaline." This observation has not as yet been interpreted, and Flechsig himself declines to give a decided opinion as to whether the bodies have been formed in the blood or lymph vessels or whether they have to be regarded as products of degeneration from ganglionic cells and nerve fibres. Although we have to admit that lesions in the lenticular nucleus may cause choreic movements, we can as yet make little use of these bodies as an anatomical cause for the disease. Earlier observations of conditions which were considered as significant for chorea—that is, hyperæmia of the brain and the spinal cord, lesions of the corpora quadrigemina, tubercles in the cerebellar peduncles, inflammatory conditions in the vertebræ, and spinal irritation resulting therefrom—possess only historical interest.

Although we are then still unable to say anything definite

about the nature of the disease, the assumption that we have before us an affection of the entire nervous system, in which, to be sure, the brain takes the most prominent part, seems the most probable. Whether certain portions of the brain are particularly qualified to produce choreic movements—whether, besides being produced by irritation of the cortical motor centres, they may also be brought about by lesions of the basal ganglia; further, whether this irritation can ever be attributed to infectious material, microbes, or the like, or whether it can ever be connected with fungous growths, such as, for example, Naunyn has found in the pia belonging to the species of the cladothrix or leptothrix—all these remain open questions, and we must also leave undecided whether or not perhaps the alteration of the blood depending upon the so-called rheumatic diathesis is sufficient for the development of the disease.

Ætiology.—Among the causes of chorea heredity plays an important *rôle*, as it does in all diseases of the general nervous system. This factor is more important, since heredity can here not only be called an indirect predisposing circumstance, owing to which an individual is more prone to one or the other nervous disease, but because there exists actually a hereditary form of chorea which is handed down from generation to generation and which for a great many years may remain in the family. This chorea hereditaria, or, as it is also called, Huntington's chorea, does not set in as the common chorea does, in childhood, but usually not until the age of thirty or forty. It attains the highest degree of those above described, and not rarely leads to pronounced mental deterioration. It is incurable. There are "chorea families" in which a whole generation never remains free from the disease and only certain members are exempt. On the other hand, there exists also a chorea congenita (Rau, Inaug. Dissert., Berlin, 1887), which has to be attributed to an affection of the mother caused by fright, etc., during pregnancy (Fox, Richter, Möbius, Oppenheim). It has long been known that pregnancy itself may to a certain extent predispose to chorea, as is shown by the so-called chorea gravidarum. Age and sex play a certain *rôle* among predisposing causes, inasmuch as the young and the female sex are especially prone to it. Among 439 cases, 322 (that is, seventy-three per cent) were girls, and 340 (that is, seventy-four per cent) were between the ages of five and fifteen; 411 (that is, ninety-one per cent) were between the ages of five and twenty

(Mackenzie). In rare cases old people become subject to chorea (chorea senilis). The oldest of my patients was eighty-one, the oldest of Mackenzie's patients even eighty-six.

Among the exciting causes there are two kinds which are particularly important—the one, psychical excitement, particularly fright and anxiety; the second, frequent contact with individuals suffering from chorea, which awakens an impulse to imitate the pathological movements and gives rise to what we then call chorea imitatoria. The latter is far less important than the former, as the imitation choreas are much more uncommon than those depending upon psychical emotions. In Mackenzie's report the cases of chorea caused by fright amounted to sixteen per cent, which seems to be a rather large proportion. The time which elapses between the reception of the noxious influence and the development of the disease usually comprises from five to seven days, sometimes only one day. Sometimes, again, the effect follows the cause immediately, this being so in ten per cent of all cases caused by fright. Besides fright, bodily or mental overexertion, particularly the latter, may become the cause of the disease. According to Mackenzie's report, sixteen per cent of all cases observed are attributable to this cause.

Treatment.—Cases of uncomplicated chorea get well without any interference on the part of the physician, but the results of wide and varied experience have taught us that with certain measures we are able to cut short the duration of the disease to a no inconsiderable extent. With reference to the internal treatment it is interesting to follow up the different phases and changes through which this has passed in the last half century. When the spinal cord was supposed to be the seat of the disease much was thought of strychnine, which had been recommended by Trousseau and which was administered in the form of a sirup. Later, when to the rheumatic basis of chorea a prominent place was given, colchicum and quinine were preferred. Again, camphor, potassium iodide, and hydrocyanic-acid preparations were prescribed when irritation of the sexual organs was held to be the starting point of the disease. Venesection, leeches, cups to the head and along the vertebral column, were applied for a time on the authority of Sydenham. All these measures have now more or less fallen into oblivion, and even the zincum oxidum album, once so warmly recommended by Hufeland, has had to give way to

other remedies. Among those still valued, arsenic, which was introduced by Romberg, stands first. It is best given in the form of Fowler's solution, in doses of from three to five drops three times a day, the dose being gradually increased to twenty or thirty drops a day. The medicine ought to be well diluted with water. Instead of Fowler's solution we might prescribe the waters of the Roncegno or Levico springs in doses of a teaspoonful to a tablespoonful three times a day. At the same time we must be on the lookout for intoxication, which has been known to be produced even by small quantities of the drug, as was proved by a case of my own. The arsenic treatment is to be continued until either the symptoms abate or digestive disturbances make their appearance, which would contra-indicate its continuance. We usually attain our end in from fifty to sixty days.

Next to arsenic we prefer the salicylate of physostigmine (eserine), which, in the form recommended by Riess (Berliner klinischer Wochenschrift, 1887, 22), may be injected hypodermically twice a day in the dose of one milligramme ($\frac{1}{60}$ gr.). Excellent results may be obtained with this mode of treatment, and the duration of the disease may be reduced to thirty or forty days. We need hardly insist that this drug must be administered most cautiously, because eserine poisoning has been observed (Lodderstädt, Berliner klin. Wochenschr., 1888, 17). As soon as any bad effects begin to show themselves, such as nausea, vomiting, etc., it is advisable to discontinue the medicine at once for a considerable time. About antipyrine, which has been recommended by Legroux and others, we can not speak so favorably. The results obtained with this drug are uncertain and transient. We were never able to note cures within from six to twenty-seven days with this remedy, such as Legroux has reported. If these medicines leave us in the lurch we may with caution prescribe chloral, morphine, opium, under the influence of which the movements may temporarily abate.

Among other measures we may mention the use of cold water and electricity, which, although only of secondary importance, may not be without good effects. We have in different places spoken of the cold-water treatment, and wish again to repeat here that extremely low temperatures are unnecessary, but rather that hip baths of 84° F., with cold affusions to the back (81° to 75° F.) and wet packs seem sufficient.

In the electrical treatment chiefly the constant current is to be used, which is made to act alternately upon the brain and the spinal cord (Erb, *loc. cit.*, p. 587).

Sometimes all these means of treatment which we have just described are ineffectual. The patients take medicine, undergo the cold-water treatment, etc., and no improvement is noticeable. In such instances a change of climate is to be recommended; the patient may be advised to travel, and be kept away from his family for some time; excitable individuals especially, in whom psychical influences increase the motor irritation, are to be secluded as much as possible. Visits of friends or members of the family should be interdicted. Children should be kept away from school, and should be spared any mental exertion. Even at home they should not be made to work; they should be encouraged to suppress the movements as much as possible, and a small reward should be promised if they succeed. In this manner often a good deal is attained. Only in exceptional cases need the patient be in bed for any length of time—namely, if the twitchings are very violent and likely to lead to bodily injury. In such instances the use of narcotics, as suggested above, becomes more especially warrantable.

The symptomatic chorea, or rather the cerebral and spinal diseases in which choreiform movements occur as symptoms, we need not here describe, as we have already called attention to them at the appropriate places. It will hardly ever be difficult to decide in a given case the question whether the movements before us are due to a functional neurosis—that is, whether the case is one of an uncomplicated idiopathic chorea—or secondary only to cerebral or spinal lesions.

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CHAPTER II.

THE name tetany (Corvisart) has been given to a neurosis which is characterized by paroxysmal tonic muscular spasms, during which consciousness remains undisturbed. The spasms are often confined to the flexors of the fingers and of the wrist joint, and only rarely attack the muscles of the lower extremities; they are always bilateral. The fingers are drawn together and the hand assumes, to use Trousseau's comparison, the shape which the obstetrician gives it when introducing it into the vagina. With these spasms, which are of great intensity, so that the affected muscles feel tense and hard as boards, are associated slight flexion at the elbow joint and a moderate adduction of the upper arm. Hérard claims that the pressure of the thumb upon the other fingers may be so strong as to lead to pressure gangrene, but this is unquestionably very rare. If the lower extremities are affected the feet assume a position of plantar flexion, and the big toe is drawn under the second or third. Sensory disturbances are usually entirely absent, only the contracted muscles are painful on pressure and the skin over them is covered with a copious sweat.

These attacks, which vary a good deal in frequency as well as in duration, may be produced by pressure upon the larger nerve trunks or the larger arteries of the upper extremities, as Trousseau has found accidentally, by applying a venesection bandage; thus, by pressure upon the median nerve or the brachial artery, a spasm may be produced of exactly the same nature as the spontaneous ones. This is called Trousseau's sign, and is considered to be of great diagnostic importance.

The attacks rarely ever occur suddenly and unexpectedly. Generally they are preceded by prodromal symptoms, which last for a few minutes and consist in a painful drawing sensa-

tion in the hands and arms. Previous to the first attack such
sensations, together with formication, feelings of coldness, etc.,
may have existed for weeks. The attacks last rarely more
than five or ten, usually they are over in one or two, minutes,
and it is only in exceptional instances that they go on for sev-
eral hours. Their frequency also varies as we have said.
Some patients—just as now and then happens in epilepsy—
have not more than one all their life, some have several a
day, and in others again weeks, months, or years pass between
the individual attacks, and the whole disease may extend over
twenty or thirty years. In all cases, however, provided there
exist no complications—such as joint affections—the outcome
is favorable, and in no case can any lasting bad effects upon the
organism in general be noticed. In the intervals the patient
does not complain of anything and feels in perfect health.
Only an objective sign is demonstrable, which betrays that
everything is not going on normally—namely, an increase, not
only of the electrical, but also of the mechanical excitability of
the nerves—a condition to which Erb has called attention.
Even a weak current produces a marked effect, and by simply
stroking the face with the finger it is possible to elicit lively
contractions of the muscles supplied by the facial nerve. Al-
though this sign is not constant, since it has in cases of tetany
been looked for in vain, even after the most careful examina-
tions, and although we must not forget that it occurs not in
tetany alone, but also in organic diseases of the spinal cord—
e. g., in glioma—it remains, nevertheless, very valuable, and
must certainly be taken into account in the diagnosis.

The anatomical seat of the disease is still obscure. It has
been referred to the most varied parts of the nervous system,
to the cerebrum, the cerebellum, the spinal cord, the periph-
eral nerves, even to the sympathetic, which seems anyhow to
be the part of the nervous system which is blamed for affec-
tions we can not locate. All these, one after the other, have
been suspected of playing a *rôle* in the pathogenesis of tetany,
but proofs have never been brought forward for the correct-
ness of any of these views.

The least probable theory seems to be the one which as-
sumes the disease to be of a peripheral nature. This can hardly
be brought into accord with the fact that the affection has been
known to follow psychical influences, for, just as we have seen
to be the case in chorea, and as we shall soon learn for epi-

lepsy, so this disease can be brought about by imitation, and indeed there have been instances recorded where in this manner even small epidemics of tetany appeared in schools (Magnan, Gaz. de Paris, 1876, 50, and Gaz. des hôp., 1876, 141). The disease has further been observed in women who are suckling infants, in young mothers and wet nurses; and so frequently has this been the case that Trousseau felt himself justified in terming tetany " la contracture des nourrices." Quite inexplicable are those cases occurring after extirpation of goitres (N. Weiss, Falkson, and others) and after infectious diseases, especially scarlet and typhoid fevers. All this speaks, however, in favor of the central nature of the disease, as does also the fact that the occupation may have some causative influence, inasmuch as people who have to use their arms, hands, and fingers a great deal—telegraph operators, seamstresses (Mader, Hirt)—are relatively frequently subject to it. According to our opinion, the cortical nature of tetany is as probable as the cortical nature of writer's cramp.

The great rarity with which the affection occurs makes it practically of little importance. If we add to this that the cases, which we see, run without exception a favorable course, one can understand why but little is to be said of the treatment. If any interference be necessary or desirable we may avail ourselves of the galvanic current, placing the anode over the affected parts and the cathode in some indifferent place. This may be repeated two or three times a week, each time a moderate current being allowed to pass for from three to five minutes. During the attack this procedure is sometimes quite beneficial, whereas upon the course of the disease it has as little influence as the well-known nervines. We have used tepid baths with success, inasmuch as the patients felt very comfortable in them and claimed to be able to notice a diminution in the frequency of the attacks. It is our opinion, however, that even the baths can be dispensed with and that it is best not to subject the patient to any therapeutic measures at all.

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Thomsen's Disease.—Under the name of Thomsen's disease an affection has been described which is characterized by "tonic spasms in the muscles during voluntary movements." When any muscle is moved voluntarily, as it contracts, a tonic, painless spasm comes on which either greatly impedes the intended movement or completely frustrates it. If the patient wishes to perform certain motions a sensation of fatigue is felt in the part and a resistance, which he has first to overcome before the intended movement can be executed. Objects which he is holding in his hands he can not let go at once and put down. Running, dancing, gymnastics, the manipulations of the military drill, are absolutely impossible, and any such attempts distress him very much and bring him into the most annoying situations. If the musculature of the tongue is implicated a motor speech disturbance is added. Sensory disorders are not found, and in general the patients are perfectly well if they do not attempt to move. Objectively may be noted, besides the increased excitability to the galvanic current, an unusually strong development of the musculature and an increased power which seem almost to belie the complaints of the patients that they are embarrassed in their movements.

Heredity stands for a great deal in the disease, which was evident from the first from the description which Thomsen himself gave in 1876. He reported that in his own family in five generations more than twenty persons had suffered from it. Often it is congenital, hence Strümpell has proposed the name myotonia congenita.

The nature of the malady is still a matter of conjecture. The fact that on galvanic stimulation of the muscles the contractures are slow and very prolonged, lasting even as much as thirty seconds—myotonic reaction of Erb—the observation of Erb that on microscopical examination the muscular fibres are seen to be broader, the nuclei multiplied, and the interstitial connective tissue increased, are not points sufficient to warrant a definite decision about the seat of the disease Still, the possibility that we are actually dealing with an affection of the muscles is by no means excluded.

The disease, which interferes greatly with the occupation, is wont to last throughout the entire life. The patients learn to accommo-

date themselves to a certain extent to the inconvenience, and by allowing for it are able in a measure to hide their awkwardness. In countries where military service is compulsory any one suffering from myotonia is exempt. No treatment has as yet been promulgated for this rarest of affections.

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AMONG the diseases of which we are treating in this part paralysis agitans is the gravest, but happily also the rarest, for, according to statistics of my own cases, only about 0.43 per cent, or one in two hundred and twenty-nine, of all affections of the nervous system were instances of paralysis agitans. Within the some sixty years which have passed since Parkinson's description appeared, certain symptoms of the disease have, it is true, been studied more carefully, but our knowledge of the ætiology, the anatomical seat, the treatment, etc., has not improved to any extent, and in fact our progress has been unsatisfactory.

Symptoms.—The first thing observed by the patient is a feeling of weakness in the extremities, followed soon after by a slight tremor, which at first only occurs temporarily. It is more marked in the upper extremities, especially in the right arm, yet it is also noticeable in the legs, and exceptionally in the head. The old idea that the head is always exempt from the tremor of paralysis agitans, and that this exemption is, *cæteris paribus*, characteristic of the affection, is untenable. In rare instances the tremor is confined to one half of the body, whereas the other remains quiet.

The tremor consists of uniform oscillating movements, the oscillations being rather few in number, not more than from four and three quarters to five and a half per second (Cramer), whereas the tremor of Graves's disease, for example, presents from nine to nine and a half oscillations per second (Marie). The lengths of the oscillation waves have been studied by Marie, Cramer, and others, and the handwriting of the patients has generally been utilized for such observations. The oscillations were recorded on paper by means of a Marey's drum or rubber ball,

which the patient was made to hold loosely in his hand. Repeatedly with perfect regularity of the wave lengths a variability in their height could be demonstrated, the physiological cause for which is not entirely clear. It is not infrequently seen that the tremor increases on forced attempts at motion, and passes into a regular "shake," so that the patient, although not entirely helpless, becomes very awkward in feeding himself. It is a fact of considerable diagnostic importance that the

Fig. 157.—SPECIMEN OF HANDWRITING OF PATIENT WITH PARALYSIS AGITANS (personal observation).

movements during rest in bed do not cease, but continue and hinder the patient from getting to sleep, and (in contradistinction to what we find in chorea) do not disappear even during sound slumber. Indeed, the intensity of the tremor may remain undiminished in bed, and I know of instances in which the patients procured for themselves iron bedsteads in order to avoid the annoying creaking of the wooden bed caused by the violent shaking. In other cases the condition improved upon lying quietly in bed, and falling asleep was facilitated by the use of certain artifices. Thus Eichhorst relates of one of his patients that he always carried a little twig between his teeth so as to keep his jaws quiet, and one of my own cases only

could obtain comfort and a certain amount of rest in his fingers
and arms by rolling small objects—for instance, little wooden
balls which he had made for the purpose—between his fingers
and thumb. With the aid of these he also could go to sleep.
If by accident he left these balls at home, he unconsciously
picked up other objects which might be lying before him, such
as matches, or he rolled bread pellets, and so on, and only felt
comfortable when his fingers were occupied with something of
this sort. The change in the handwriting caused by this
tremor is illustrated in Figs. 157 and 158.

In connection with, and probably as a consequence of, the
trembling movements, gradually a condition develops in which

Fig. 158.—Specimen of Handwriting of Patient with Paralysis Agitans (personal observation).

the patient gets easily tired, the muscular strength diminishes,
and the muscles assume a certain rigidity which influences the
position of the body and the extremities when at rest as well
as on voluntary motion. The position of the body is charac-
teristic. Not only the head, which affords a good deal of re-
sistance to passive movements, but also the whole trunk is
somewhat bent over, and it appears as if the patient was at
every moment ready to fall forward. The arms, which are
bent at the elbows, are in close apposition to the trunk, the
thumb rests against the fingers, so that the hand assumes some
such position as it would in writing, the fingers themselves
being flexed in the metacarpal joints (Fig. 159). The knees
are so close together that the trousers are rubbed against each
other by the trembling movements, and walking is not a little
interfered with. The legs are usually slightly flexed at the
knee joints, while nothing remarkable can be noticed about
the joints of the toes. The patient impresses one as being in
a constant state of uncertainty and perplexity, an impression
which is only diminished to a certain degree by the very char-

acteristic facies.   The rigidity being also marked in the muscles supplied by the seventh nerve, the face has an expression of majestic calm, nay, even of sublimity.   The patient seems inaccessible to psychical emotions.   His smile is hardly perceptible, since the lower portions of the face more especially are almost immobile.   Only the wrinkling of the forehead is somewhat more marked.   Sometimes the patients have a peculiar piping voice, such as an actor assumes when playing the part of an old man on the stage.

We have already alluded to the fact that voluntary movements are somewhat impeded.   This is not only due to the tremor, but also to the already-mentioned general weakness.

Fig. 159.—POSITION OF HANDS AND FINGERS IN PARALYSIS AGITANS (as if holding a pen).   (After EICHHORST.)

He is, therefore, helpless, and needs some one to assist him if he wishes to sit up in bed or even to change his position.   If he is in a sitting posture rising is difficult, sometimes impossible.   The act of walking is not normally performed, for besides the bent position, which in walking becomes even more exaggerated than in standing, the patient once started has an irresistible tendency, owing to the displacement of his centre of gravity forward, to hurry ahead: his steps, at first short and tripping, become quicker and longer, and so great may be the force with which he involuntarily rushes forward that if there is no one there to stop him he falls on his face with great vio-

lence. The same phenomenon, which is called "propulsion," may sometimes be artificially produced by pulling the patient forward by the coat while he is walking quietly. He then goes faster and faster, and finally breaks into a run alarming to the bystanders. Much more rarely do we find a similar condition in the backward motion ("retropulsion"), so that the

Fig. 160.—POSITION OF THE BODY IN PARALYSIS AGITANS (personal observation).

patient if pulled from behind walks backward faster and faster, to fall over in a short time. Charcot looks upon these phenomena as forced movements, a view which has, however, never been substantiated. They may possibly be explained on purely physical grounds as being due to the displacement of the centre of gravity of the body (Strümpell).

Trophic changes, with the exception perhaps of the transient appearance of purpuric spots symmetrically on the arms and legs ("senile purpura"), are not met with. Changes in the electrical excitability of the muscles do not occur, or are, at any rate, not the rule. Sensation and reflexes remain entirely

normal, and bladder as well as rectal symptoms are not present. An increase in the body temperature can never be demonstrated objectively, although patients complain at times of subjective feelings of increased heat and a disagreeable tendency to sweat a good deal, which is especially pronounced when lying in bed, so that they often sleep uncovered or with but little over them. If any cerebral or spinal symptoms make their appearance these have to be regarded as complications. They do not belong to the clinical picture of paralysis agitans as we know it now.

Cases in which muscular weakness and rigidity, with all their inconvenient consequences, were present, in which, also, the so-called propulsion was marked, but the tremor was absent, have been reported (Amidon, New York Medical Record, 1883, xxiv, 21), but such are rare.

The nature of the disease is not yet understood. We do not even know whether to refer it to the brain or to the muscles. Much less, of course, do we know where the exact seat should be sought for in the nervous system. Before the labors of Charcot and Ordenstein, paralysis agitans was often confounded with multiple sclerosis, and various anatomical lesions were then described as underlying the paralysis agitans. Later the error was cleared up, and even to the present day we are not acquainted with any anatomical basis for the disease.

Ætiology.—Uncertain is our knowledge also about the causes. Of course here, as in all other nervous diseases, heredity and the importance of a neuropathic family history must be spoken of, yet the rarity with which the affection occurs shows that this factor alone is seldom sufficient to cause the disease. Hence other exciting causes must come into play, but it is a fact difficult to understand why the same factors which so often give rise to chorea so rarely produce a shaking palsy. The causes for all these diseases are always the same, or at least similar, and it is here also in the first place that psychical emotions of fright and anxiety are of moment. The French physicians have at no time seen develop so many cases of paralysis agitans as during the time of the siege in 1871, and for years after the relative frequency of the trouble in the Paris hospitals, particularly in the Salpêtrière, acted as reminders of the terrible hours which the besieged must have gone through. In private practice we also have occasion to find that psychical causes bring about the disease; more frequently, however, at

least in my own experience, no cause at all can be found. The influence of exposure to cold and of overexertion of course has here also been thought to be of ætiological significance without there being any grounds for such an assumption; on the other hand, there is no question but that certain infectious diseases— e. g., intermittent fever, pertussis, typhoid fever—may be followed by a paralysis agitans, a connection, however, which, although certain in its existence, is still obscure in its nature. Nothing definite is known about the influence of age and sex.

Diagnosis.—After what has been said little needs to be added with regard to the diagnosis, which is almost always easy. It is certainly not hard to avoid mistaking paralysis agitans for multiple sclerosis or chorea, and chronic alcoholism is easily excluded if we take into account the characteristics of the tremor, its continuance during sleep, and the whole course of the disease. It may be sometimes difficult to differentiate a shaking palsy from the ordinary tremor senilis if the latter occurs as early as the forties, at a time of life during which paralysis agitans is not rare, and it is the more necessary to be careful since the number of the oscillations in both affections is about the same, that is, ranges between four and six per second. The muscular weakness, the peculiar rigidity which accompanies the movements, the characteristic facial expression, the posture, the "propulsion," etc., will in most cases be sufficient to clear up the diagnosis.

Treatment.—The treatment is entirely fruitless. We have not as yet seen any results from any of the therapeutic measures employed. Neither with baths nor with massage (Berbez, cf. lit.) nor with galvanism has anything been achieved, and all internal medicines are of no avail. It is impossible to give particular indications for the treatment, and it must therefore remain for the physician in every case to treat alternately with baths, massage, and electricity, according as he sees fit. As long as he does not do the patient any harm it does not matter much which mode of treatment he decides to use. Lately Erb has recommended the muriate of hyoscine injected subcutaneously or taken internally. This is said to have a very good effect upon the tremor, but whether this effect is lasting, and whether the bad after-effects which occasionally appear after a prolonged use of the drug are not a grave objection to its administration, is not as yet decided. My own experiences with it were not favorable.

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CHAPTER I.

NEURASTHENIA—NERVOUS PROSTRATION.

NEURASTHENIA (*a*, privative; σθένος, force) or nervous exhaustion is an affection of the nervous system with which the general practitioner meets very frequently, and is one of those diseases which may give rise to a good deal of error in diagnosis and prognosis. At the same time it makes the most boundless demands upon the forbearance of the physician and upon the patience of the sufferer. The disease is a child of the modern mode of living, of the desire to become rich as soon as possible, and we look for it in vain in the old text-books. Although it may in earlier times have occurred now and then, the neurologists had neither opportunity nor occasion enough to study it intimately. This has only become possible quite recently, and it is certainly a fact of significance that neurasthenia has been "discovered" in that continent, the inhabitants of which have the reputation of working the quickest, of living at the highest pressure, and therefore of being—of course with exceptions—more nervous and aging sooner than those of the Old World, to wit, in America. Beard, to whom we owe so many excellent observations, so many splendid hints for therapeutics, described it first and gave it the name it bears. Whereas the disease prior to Beard's publication was unknown, it soon began to prevail in such a striking manner and to be diagnosticated so frequently that one is almost led to think that this diagnosis is often arrived at in cases where something else exists, some organic affection possibly more difficult to recognize. The disease in question is not organic

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and not associated with any demonstrable anatomical alterations. Nobody has ever succeeded in finding any characteristic anatomical changes in individuals who have suffered for years from the most pronounced neurasthenic manifestations and then have died from some intercurrent disease. A large number of subjective complaints, many of which fit into other clinical pictures, make it intelligible why a diagnosis of neurasthenia is often made, sometimes without any sufficient, careful, consideration of all the factors which ought to be taken into account. It is comfortable and presumes nothing. Its possible incorrectness can frequently not be demonstrated, and it therefore rapidly attained a great popularity among physicians.

Symptoms.—The first traces of the disease develop very gradually and imperceptibly. Sometimes they assume more of a cerebral, sometimes more of a spinal character, so that it has been thought justifiable to distinguish a spinal and a cerebral neurasthenia. For the cases in which the symptoms of derangement of digestion were most prominent the term gastric neurasthenia was coined, under which head we may possibly class certain of the so-called nervous dyspepsias. In the majority of cases the patients complain of getting easily and rapidly fatigued after the bodily exertion which is associated with their ordinary daily doings, whether at home or in their business, after walks, gymnastic exercise, etc. Things which they used to do without the least difficulty tire them greatly. In going distances which were formerly covered with ease they have to rest half way, and require more time to accomplish a given task. Not always are definite pains present. At times there are aches in the back and loins severe enough to be troublesome. Sensory disturbances, paræsthesias, formication in the extremities, or numbness, are rarely absent. These feelings distress the patient and may make him fear he has tabes, and the idea that he is suffering from some spinal trouble is fostered by the circumstance that the sexual power is usually decidedly diminished, be it that the patient is unable to have connection as often as before, be it that the erection of the penis is incomplete or that no ejaculation of semen occurs. For married patients this weakness is a source of great distress and often is a very prominent symptom, and frequently it is this that finally decides them to consult a physician, a step which has been again and again deferred. The more we have to deal with neurasthenias the more frequently shall we make the ob-

servation that the sexual functions are in the majority of cases in some way or other affected, and that the sexual neurasthenia particularly deserves the most careful attention of the physician. To determine whether the complaints of a patient with regard to his disordered sexual functions depend upon organic disease or upon neurasthenia we have, besides a careful examination of the genitals, to examine the urine. It is well known that the urine of neurasthenics not rarely presents a decided increase of urates, oxalates, and phosphates, and that not infrequently spermatic fluid is passed during micturition or during defecation (Beard and Rockwell, cf. lit.). Where the patients complain, as they so commonly do, of impotence, we shall have to determine what form we are dealing with, and whether organic disease, more particularly atrophy of the testicles, is the underlying cause. Sometimes there exists only a decrease in the sexual desire, while the power remains the same ; sometimes a decrease in the power and an increase in the desire, so that the ejaculation of semen occurs too early, sometimes before the insertion of the penis. Again, both sexual desire and power diminish *pari passu*, or finally the *potentia coeundi* is normal but there is absence of spermatozoa (" aspermatism ").

All changes of this kind are noticed by the patient and their significance is ever exaggerated by a fervid imagination. Even in the cases in which in reality there is no disease and in which the impotence depends entirely upon psychical influences, it makes itself disagreeably felt, and we must not forget that such a " psychical " impotence, in spite of all encouragement and all assurances on the part of the physician is sometimes more difficult to cure than one which depends upon organic disease of the sexual apparatus. Every abortive attempt at coitus exerts a depressing influence upon the patient for a considerable time and is quite liable to lead to a second failure, although all other conditions for the normal performance of the act may be favorable (Fürbringer, cf. lit.).

The disturbances of the cerebral functions which appear in the course of neurasthenia are very manifold. First, the patient is down-hearted and worried and sees everything in the blackest colors, and, above all, despairs of recovery. He becomes irritable and impatient, unsociable with his friends, and feared by his family. In his work he is less efficient. Duties which he previously performed without trouble seem hard to him and require twice or three times as long for their accom-

plishment.   Cases in which this is not a prominent feature, but where the working power remains unchanged, are met with, but are exceptions.   The sleep is usually disturbed ; sometimes a protracted insomnia adds to the trouble.   Headache is not the rule, but the patient often complains of a disagreeable pressure in his head, which is accompanied with a slight feeling of dizziness.   All functions share in the disorder, the appetite becomes bad, the bowels sluggish, the action of the heart feeble, and vaso-motor disturbances in the form of persistent coldness of the hands and feet manifest themselves.   The general condition of the patient is very pitiable in the higher grades of neurasthenia, and it is necessary for the physician to make a most careful examination so as not to go astray in the diagnosis.

The objective examination, in contradistinction to what the manifold complaints of the patient might lead us to expect, reveals strikingly little.   Organic changes can not be demonstrated anywhere.   Thoracic and abdominal organs are healthy ; nothing abnormal can be detected in the domain of the cranial nerves or in the fundi of the eyes.   The condition of the pupils varies.   Transient differences in their size—that is, unilateral dilatation, without, however, any abnormity in the pupillary reflex—is certainly met with.   The dilatation may either always be on the same side or change at times to the other eye. The phenomenon is usually marked when the general condition is bad, while it disappears if decided and lasting improvement is once established.   The claim that lasting inequality of the pupils is always a sign of organic disease, as Beard thinks, must certainly be somewhat modified (Pelizaeus).   I have myself seen differences in the pupils persist for eight or ten months and then disappear and the patient get well.

The peripheral nerves as well as the tendon and skin reflexes are normal.   Tenderness over the vertebræ rarely is absent, but is of no significance.

Diagnosis.—When we have once sufficiently informed ourselves about these points the diagnosis will usually present but little difficulty.   At first, it is true, we may be easily led astray and think of organic diseases of the brain, especially progressive paralysis of the insane or a brain tumor, yet the further course of the disease will soon clear the matter up.   The suspicion of tabes which may arise on account of the cerebral and particularly of the spinal symptoms, the disturbances of

the sexual functions, and so forth, will be discarded, owing to the persistence of the patellar reflexes, the absence of actual bladder symptoms and pronounced sensory disturbances, anæsthesias and hyperæsthesias, as well as of actual motor weakness. Fibrillary twitchings, such as are observed in progressive muscular atrophy, may here also be met with, but they are seen rarely and their occurrence varies a good deal. From hysteria neurasthenia is distinguished by the fact that the constant change of the symptoms which is so characteristic of hysteria, besides the circumscribed neuralgias, the contractures, the spasms, etc., is here not observed. Still, to make a diagnosis, repeated and careful examinations are needed, to which the patients do not submit as willingly as hysterical men and women.

Ætiology.—In every case in which hereditary influences can be excluded the prime cause of neurasthenia is unquestionably to be looked for in an overtax of the nervous system. This is brought about in many ways, by excessive mental work or by habitual bodily overexertion. It may be attributable to repeated emotions or to sexual excesses. Under the latter head we may put masturbation, which is a widespread evil among the young of both sexes and the practice of which not only may begin very early, but may be continued much longer than the physician himself might suspect. One may say that there are but few neurasthenics who have not during their youth been addicted to this habit for a longer or shorter period of time. "Sexual perversion" (Spitzka) and the various kinds of "psychical masturbation" may also become of ætiological significance. Even in married life, where the satisfaction of the sexual desire is otherwise well regulated, the coitus interruptus sive reservatus, which is practiced to avoid too great an increase in the family, may afford a cause for neurasthenia. In my experience very few men have been able to practice with impunity for years this coitus interruptus, and it is the bounden duty of the physician to inquire with much tact but still with perseverance into this question.

In some cases the abuse of tobacco may lead to neurasthenia, so that the latter has to be looked upon directly as a nicotine poisoning, and must, of course, be treated accordingly. Persons whose occupation necessitates work not only energetic, but also associated with the emotions (artists, students, financiers, speculators, etc.), also those whose occupation entails at

the same time bodily as well as mental strain, are all more or less neurasthenics. Not rarely repeated losses of a considerable quantity of blood produce neurasthenia by causing a general anæmia, yet we must definitely state that the neurasthenia may occur very well in such cases without the anæmia. Traumatism also may cause neurasthenic conditions. About these, which are usually intermediate forms between this disease and hysteria, we shall have to speak later, under the head of " traumatic neuroses." Finally, neurasthenia has been known to occur after infectious diseases, typhoid fever, cholera, variola. In these cases the bad state of nutrition and the faulty condition of the blood have to be held responsible.

Treatment.—The treatment of neurasthenia is one of the most troublesome tasks which the physician encounters. It is, of course, not sufficient to give the patient a prescription and let him go. We must frequently examine him, not only on our own account, but for his own sake as well, because he is comforted by the attention and solicitude of the physician, although the examination itself is usually disagreeable to him. There are neurasthenics who are actually relieved by repeated examinations, although nothing is ever prescribed. They gain therefrom the quieting conviction that somebody is looking out for them, and this gives them hope. But here also the direct psychical treatment, such as we usually find to be of value in hysteria, is of the greatest importance. The patient must again and again be encouraged and told that all his organs are healthy, that it is only a nervous overstrain which he is suffering from, a deficit in his nerve capital which it is somewhat difficult to replace. To exert a mental influence upon the patient in this manner time is necessary, and those physicians who can not afford sufficient time for the purpose should not take charge of a grave case of neurasthenia at all.

If hypochondriacal notions are prominent features, so that the patient is beyond the reach of consoling and encouraging words, the question whether or not he should be removed to an institution must come under consideration. In addition to the fact that change of air and scene exerts in itself a favorable influence, it is advisable to place a neurasthenic after a certain time among different surroundings, so that he has to meet with different people and has something fresh to occupy his mind with, and care should be taken to keep him constantly under the guidance of a physician. As supplying such requirements

sanitaria for nervous people, in which insane cases are not admitted, are to be highly recommended. Of course the pecuniary condition of the family must, before deciding upon this, be taken into account, since all establishments of this kind in which patients are well cared for are rather expensive. Sometimes in the more favorable instances a stay of from four to six months is sufficient to bring about a very decided improvement, in which case even families who are not very well off should be able to afford the expense.

There are especially two factors from which much is to be expected in the treatment of neurasthenia, and these are electricity and hydrotherapy, particularly the cold-water treatment. With regard to the former it may well be stated that there is no other nervous affection in which its application is followed by such excellent results as here. Used at the proper time and in proper doses, so to speak, it is most beneficial. The method which is best employed and to which we give by far the preference is the so-called general faradization as recommended by Beard and Rockwell, as well as the general galvanization. The results are especially striking if we make use of the brush, which, in Beard's method, is not only applied to the back—although it is kept here longest—but (with the exception of the head) all over the body. Although the patient may complain of disagreeable and painful sensations for the five or eight minutes during which the sitting lasts, the after effects which soon follow are most gratifying. The patients feel invigorated and leave the physician with a sense of having gained a new lease of life. According to our experience the faradization as advised by Beard is superior in its action to the electrical baths, which are much more circumstantial and have not been as yet sufficiently studied.

With reference to the cold-water treatment, to which we have repeatedly called attention in different places, we must in this more than in any other affection warn against overzealousness and insist upon caution. Low temperatures are borne very badly by these nervous and irritable patients. They become excited and sleepless, and our aim is not only frustrated, but actually more harm than good is done. However, if we cautiously begin with a temperature of from 86° to 78° F., and confine our measures to gentle rubbings, affusions of short duration, cool hip baths, also of short duration, and avoid douches altogether, if care is taken at the same time to insure

proper nourishment and exercise for the patient, the best form
of which, perhaps, is a walk in the woods, the results are en-
couraging and lasting, if neither physician nor client lose their
patience too soon.   A course of treatment of this kind can not,
however, be compressed into the usual four weeks of a summer
vacation, but to do any good six, eight, or ten weeks should be
taken.   Sometimes sea baths will be more useful than the
simple cold-water treatment, but then also care must be taken
in their selection.   For the excitable and nervous who suffer
from insomnia the places on the Baltic will on the whole be
preferable to those on the North Sea, while the latter are
especially adapted for very prostrated patients and individuals
suffering from cerebral anæmia.

A long stay in pure mountain air, at a not too high altitude
and where the barometer is not too low, is usually beneficial
to neurasthenics.   Daily systematic, but not forced, tramps in
the mountains, continued for weeks, do more good sometimes
than all the medicines of the pharmacopœia taken during the
long winter.   The internal medicines are anyhow of not much
avail in the treatment of neurasthenia.   Iron, quinine, arsenic,
the stomachics, all will disappoint us; all will sometimes ac-
complish nothing; they rather tend to derange the digestion,
and with this take away the last remnant of the patient's
courage.   The only drugs necessary will be such as are re-
quired for the proper regulation of the bowels.

Among the above-described symptoms there are two the
treatment of which deserve special mention—first, the sleep-
lessness; secondly, the impotence.   About the former nothing
needs to be added to what has been said on page 162.   To
meet the latter much is to be expected—if, of course, organic
disease, spermatorrhœa, and the like, have been excluded—from
the local application of electricity.   A large electrode, the
anode, is placed over the lumbar cord, while the cathode is
moved from the external inguinal ring down along the sper-
matic cord or applied without being shifted (Erb).   With this
may be combined the application of the faradic brush over
the whole genital region.   One electrode, the cathode, may
also be placed in the rectum, the other upon the sacrum or
perineum (Möbius).   Finally, a bladder electrode, which has
the shape of a catheter, and which is insulated up to its metal-
lic tip, may be introduced into the urethra as far as the fossa
navicularis, while the anode is applied over the lumbar cord,

and at the negative pole the current is made and broken several times. From this method, which has been recommended more especially for paralysis of the bladder and incontinence of urine, we have repeatedly seen good effects in the treatment of impotence.

The feeding system of Weir Mitchell, which has also been recommended in neurasthenia, we shall discuss in the chapter on hysteria.

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HYSTERIA has this in common with neurasthenia, that it does not depend upon any demonstrable anatomical lesions of the nervous system, but it differs from it in the fact that for its development a certain predisposition on the part of the patient is absolutely necessary. Although we are not as yet in a position to say of what nature this predisposition is, we must assume that the whole nervous system of a hysterical patient, central as well as peripheral, is in some points, which we are still unable to determine, different from that of healthy individuals. The increased impressionability, the hyperæsthesia of the central nervous organs, the increased sensitiveness of the peripheral nervous system, the diminished energy with which influences coming from outside as well as from within are met, the lowered general power of resistance and self-control, these are on the whole the traits which characterize hysterical persons, and explain why the symptoms are so manifold and change so rapidly, and why in no other disease of the nervous system can be found a train of manifestations so diverse and so numerous.

Only by unwearied, long-continued study has it been possible to show that even for the apparently arbitrary appearance of the different symptoms there exist certain laws. In a manner which none before or after have been able to rival, hysteria has been studied by Charcot and his pupils, to whom we owe the most interesting observations and investigations of the past two decades.

Symptoms.—For the sake of simplicity, we shall divide in our description of the disease the symptoms into cerebral, spinal, and mixed, that is, pertaining to the entire nervous system.

The cerebral may again be subdivided into psychical and

somatic symptoms. The disposition of the patients is excitable, anxious, often changeable, sometimes passing from the depths of gloominess to the most exalted hilarity. The tendency to speak of nothing else than of their own woes, the constant attempt to greatly exaggerate these, and to excite sympathy in their friends and physicians, the thoughtless demands which they expect to be satisfied at a moment's notice, and the inconsiderate outbreaks of anger if this is not done—all these are characteristic features of the disease with which we meet, not in all indeed, but at any rate in a large majority of cases. The tendency to get easily frightened is very common, and during a state of the highest psychical excitement hallucinations may temporarily exist. In pure cases of hysteria, however, we need never be afraid that these will persist long or lead to any serious outbreak on the part of the patient. Exceptionally an instance of "hysterical sleep" comes under our notice, into which the patient has fallen after certain prodromal symptoms have existed for several hours. The peculiarities of this curious condition, the "léthargie hystérique," which may last for many days, the condition of the organs of circulation and digestion, the characteristic signs by which the hysterical sleep may be discriminated from other states of coma, have recently been described by Gilles de la Tourette in a careful monograph (Arch. de neurol., 1888, 43, 44), to which the reader is referred for further information.

Among the cranial nerves there is not a single one which may not at one time or another in the course of hysteria present symptoms of paralysis or irritation. More than the others the nerves of special sense are interesting for their anæsthesias and hyperæsthesias. The nerves of smell and hearing are those most frequently affected, and both functions may be so much impaired that the patient can smell and hear nothing. They may, on the other hand, become so acute that, if we may believe her own statements, she is able to distinguish any one from a number of perfumes, or to single out an individual by the sound of his voice amid the hubbub of a crowd, or again to recognize people far off by their step, and so forth. These and similar faculties have in Mesmer's time already been spoken of a great deal, and have given rise to much deception and trickery. The opticus is also not rarely affected. Besides the cases where hysterical patients suddenly become blind in one or both eyes without there being any

changes in the disk, there are instances of decrease in the acuteness of vision, contraction of the field of vision, or complete or partial loss of color sense. When the last-named condition occurs the perception of blue and yellow is retained longest, while that of violet and green disappears much earlier. The ocular muscles rarely participate in the disease; hysterical paralysis of them is exceptional, as is also the occurrence of hysterical nystagmus, on which subject I have expressed my opinion elsewhere (cf. lit.).

Among the other nerves of special sense that of taste may occasionally present alterations. The patients lose their taste either completely or only for certain substances (sour, salty), or there may exist such a perversion of this faculty that everything tastes nauseous and disgusting, or that everything tastes of salt or of vinegar, and so forth. Actual hallucinations of the sense of taste, although not so frequent as hallucinations of the sense of smell, are not unheard of.

The trigeminus is generally implicated. Faceache and headache, among others the kind which is confined to a small spot and is known as clavus, are comparatively frequent. The scalp is sometimes so markedly tender that the patients can not stand the slightest pressure, not even the touch of the comb, and in order to avoid the pain they abstain from all care and proper attention to the hair. The pain in the head may also be confined to one side, and resemble in every detail that of hemicrania.

What needs to be said about the facial nerve in this connection has already been treated of in Chapter V, Part II. Tic convulsif, as well as facial paralysis, may be hysterical in nature; however, we must not forget that facial spasm and hysteria may well coexist, and that a tic convulsif occurring in the course of hysteria is not necessarily of hysterical origin. The determination of this question is less important for the diagnosis than for the prognosis. The outlook in non-hysterical tic is very bad, in the hysterical variety relatively favorable (Guinon, Revue de méd., June, 1887). Of much interest are the many forms of vagus neuroses which we meet with in the course of hysteria; they may affect, in the manner described in Chapter VIII of Part II, the organs of respiration, circulation, and digestion. Among the first, not only the larynx, but the lungs also, are sometimes attacked. The laryngeal muscles become the seat of violent spasm, "hysterical spasm of the

glottis," during which the patient is afraid she is choking. The functions of the vocal cords may become so much interfered with that the patient is only able to make herself understood in whispers — to speak out loud is impossible ("hysterical aphonia"). The laryngoscopical examination reveals nothing abnormal, with the exception of some anæsthesia of the mucous membrane of the fauces, which greatly facilitates the examination (cf. page 111). The respiratory muscles may be affected in a peculiar and very striking manner, the acceleration in the number of respirations may attain such a degree that, instead of fifteen or sixteen respirations a minute, we may count from eighty to one hundred. On the other hand, they may be diminished in frequency, and the patient breathe from eight to ten times a minute, but in a labored way, showing signs of a regular dyspnœa, not infrequently with audible wheezing in inspiration and expiration ("hysterical asthma"). A dry and barking cough, which is distressing not only to the patient, but also to all who surround her, is sometimes observed, and paroxysms of yawning, sobbing, laughing, or crying ("hysterical laughing or crying fits"), may persist for hours.

Sometimes following aphonia, sometimes occurring abruptly and unexpectedly without it, in rare instances a complete dumbness sets in; the patient has either actually lost the control of her speaking apparatus or will not make use of it; in a word, she is completely mute, and no amount of admonitions, entreaties, or threats can succeed in eliciting a single word. This condition of "mutismus hystericus" may be of variable duration. In one instance which came under my notice the patient maintained silence from the fifth of September to the twenty-eighth of April of the following year. She found her voice again at once on hearing of the unexpected death of her mother (cf. the articles of Natier, Huysmann, and others).

The circulatory organs, more especially the heart, take relatively the smallest share in the disease. Hysterical tachycardia may occur, but it is rare and never well marked; even in the apparently severest attacks, which we shall describe later, the pulse is quiet. To stenocardia we have referred on page 121.

The digestive tract and the muscles pertaining to it—which, just as the pharyngeal muscles, are innervated at least partly by the glosso-pharyngeal and not by the vagus alone—may be the seat of various hysterical manifestations. The muscles of

the pharynx may present symptoms of paralysis or of irritation. In the former case deglutition is much interfered with, and may, indeed, be impossible ("hysterical deglutition paralysis").

A peculiar affection of the muscles of the œsophagus, which are supplied by the vagus, consists in a spasmodic contraction which gives rise to a very vivid sensation of a ball rising up from the region of the stomach and sticking in the throat. This "globus hystericus" is so frequently met with in hysteria and is usually so well marked that it has been looked upon as pathognomonic for the disease.

The musculature of the stomach and the intestines is liable to disturbances. According to most authors, paralysis of these muscles produces a distention of the bowels and of the whole abdomen which may be simply enormous ("meteorismus hystericus"); this is sometimes associated with colicky pains. A certain amount of the air, which frequently collects in large quantities in the bowels, escapes through the mouth with a loud, sobbing, gurgling noise (singultus, ructus hystericus). Talma (Weekblad van het Nederl. Tijdschr. voor Geneesk., 1886, 9) claims that the cause of hysterical tympanites is to be sought in a spasm of the diaphragm. As evidence in favor of his view he argues that under chloroform narcosis the distention will disappear without the emission of gas, and secondly, that the position of the diaphragm is abnormally low.

Vomiting is one of the most frequent occurrences in hysteria; sometimes it is very profuse and may persist for hours, and be so intractable as to weaken the patient considerably; on the other hand, slight vomiting may occur daily for weeks without affecting the patient's strength. Usually watery masses are thrown up which bear no proportion to the quantity of food ingested. In one of my cases the amount vomited was eight or ten times as large as that taken in.

Affections of the accessorius are not rarely seen in the form of spasmodic torticollis, while affections of the hypoglossus are very exceptional.

One of the most remarkable cerebral affections which may occur in the course of hysteria is an apoplectiform attack with consequent hemiplegia, which in many instances is associated with complete hemianæsthesia. This hemiplegia may develop with symptoms similar to those of the form following arterial

disease, and, as we have already pointed out above, it may be extremely difficult to distinguish a hysterical hemiplegia from one due to organic disease. This is especially the case if there are no other hysterical symptoms to aid us. If the unilateral spasm of the muscles of the cheek described by Charcot, and before him by Brodie (1880), which is said to be characteristic of hysterical hemiplegia, be present, the diagnosis is easier. All the symptoms associated with a cerebral hemiplegia—for instance, tremor, the associated movements, even atrophy of the muscles of the diseased side—may accompany the hysterical variety. The opinion formerly prevalent, that wherever there exists atrophy this must needs depend upon an organic lesion in the brain, spinal cord, or the nerves, has been proved to be erroneous. The hysterical atrophy may not differ from that due to organic disease; it may develop comparatively rapidly, may remain for a long time, and disappear again just as rapidly when motion returns. Fibrillary twitchings in the atrophic muscles and reaction of degeneration are absent. Whether the large ganglionic cells in the anterior horns have anything to do with the occurrence of atrophy, and, if so, what is the nature of the influence, we do not know (Babinski, Arch. de neurol., 1886, xii, 34, 35 ; cf. lit.).

Among the spinal symptoms of hysteria motor and sensory paralyses play the most important *rôle*. With hysterical patients we can not feel certain for a single day or hour that some sort of paralysis will not occur, for it is characteristic, we may say pathognomonic, of hysterical paralyses that they appear quite suddenly, and happily often disappear as quickly, it may even be after persisting for months and years. There is no characteristic distribution of the hysterical motor disturbances; they may take in only one extremity, or may extend to both legs or both arms, so that these are perfectly useless. Examination shows that the paralyses are usually of a flaccid type. We may frequently make the observation that the patients are not completely robbed of the use of the affected limbs, but that they have lost the will to use them. Especially is this apparent when they are asked to perform co-ordinated movements. A patient, though able to move the right arm, may assert that she is unable to write; though she is able to move her legs, any attempt at walking is an utter failure; on rising, her legs give way under her, and she simply

is unable to keep herself on her feet.   In short, the necessary will power is gone.

Symptoms of motor irritation—for instance, isolated muscular spasms—are far less frequent.   Of much interest are the involuntary movements which are now and again observed.   I had a lady under treatment who, without wishing it, but without being able to resist the inclination, would for hours at a stretch keep on raising both arms and letting them fall again without the least feeling of fatigue.

Clonic muscular spasms, in the muscles of the face, as well as in the extremities, which, appearing in paroxysms, usually are symmetrical in their distribution, and are not sufficient to produce movements of the affected limbs, have been described by Friedreich as paramyoclonus multiplex, and by Seeligmüller as myoclonia congenita.   That they are of hysterical origin is more than probable.   The trouble is rare, and has probably always to be regarded as an emotional neurosis.   The strength of the muscles and their electrical excitability remain unaltered, and sensory changes are absent.   Sometimes there are tender points along the spine, which may be best treated by the anode of the constant current.   Other measures are not necessary, especially as recovery seems to be the usual outcome (cf. lit.).

Often combined with paralyses of the extremities are joint contractures, which as a rule appear suddenly, and may persist for months and years.   When occurring in the upper extremities, in the elbow, in the wrist, and in the finger joints, they are usually flexor contractures; in the knee and ankle joints, extensor contractures.   The way in which they disappear under chloroform narcosis is very remarkable.   Individual muscles may also be the seat of contractures, and we have described a case on page 384 in which during the erect posture a contracture in the quadratus lumborum made its appearance, which disappeared when the patient lay down.

Among the sensory disorders the diminution or complete loss of sensibility is the most important; this may be so extensive that the patients can feel nothing on any part of the surface of the body, not excluding the mucous membranes (conjunctivæ, nose, tongue, mouth, vagina, rectum), so that we can touch them with the hot iron (thermo-cautery) or prick them with knives and needles, and they will not make the slightest

sign or attempt to draw away the part; nay, more, there are cases in which the deeper tissues take part in the anæsthesia, so that folds of skin may be transfixed and fine needles thrust into the muscles down to the bone without the knowledge of the patient if she be blindfolded. Besides the general, we may meet with circumscribed spots of anæsthesia, anæsthetic zones, on the back, on the hands, etc. The hemianæsthesia, which is strictly confined to one side, and which implicates the mucous membranes as well as the skin, has already been mentioned. These sensory changes also may appear and disappear suddenly.

Less common are the hyperæsthesias, which probably never take in the whole body and never even one whole half of the body, but are usually confined to circumscribed areas, to certain internal organs, or by preference to certain joints. These circumscribed areas, Charcot's hysterogenic zones, vary in their situation; they may be on the back, on the chest, in the extremities, or elsewhere. Among the internal organs, in women the ovaries, in men and boys the testicles, are the parts that usually suffer. The ovarian hyperæsthesia, which Charcot has studied very carefully, is closely related to the "major attacks" to be described later. That it is actually the ovaries which give rise to the acute pain when pressure is made over them Charcot has proved on pregnant women; during pregnancy the position of the ovaries is changed, and it was found that there was a corresponding change in the position of the tender points. The women who suffer from this hyperæsthesia are in Paris called "ovariennes."

Neuralgiform pains, which often affect the joints and which are very obstinate, are so common in the course of hysteria that whenever we find a joint neuralgia we should think of and search for a hysterical basis. Brodie has subjected them to a very accurate study and has pointed out that it is at times extremely hard to differentiate between a neuralgia and an actual disease of the joint. The hip and knee are most usually attacked. The joint is painful, especially on pressure or on motion; hence such patients are, as a rule, found in bed or lying on the sofa. On closer examination the pain proves not to be confined to one spot, but distributed over more or less large areas of the lower extremity. The patient cries out if pressure is made in the neighborhood of the hip or the knee or lower down over the malleoli. She seems to be especially

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sensitive when watching and following our manipulations; but if the physician is able to divert her attention, pressure over an otherwise painful point will often evoke no complaint. In the course of the disease the glutei may undergo some atrophic changes; now and then transient swellings are noted. On the other hand, there are instances where hysterical joints are the only cause which keeps the patient persistently in bed, where, in spite of an inactivity lasting for years, not a trace of atrophy can be recognized, and where the general health shows no signs of impairment. All these and other spontaneous pains, which we need not dwell upon here, occurring in hysterical individuals are to be regarded as being of psychical origin, and therefore as pain hallucinations (Strümpell, Holst).

Among the abnormities of the secretory organs, those which concern the urine chiefly deserve our attention. Hysterical patients may urinate very little and not without difficulty (ischuria). On the other hand, we find some who urinate frequently and pass almost incredible amounts (polyuria). In the former case the specific gravity is high and the solid constituents of the urine are increased in amount. In the latter the urine resembles almost clear water. It would be erroneous to assume that the small or large amount of urine always depends upon the amount of water ingested. Indeed, patients who drink hardly anything may void very large quantities of urine, while those who drink a good deal may pass only a few drops at a time. Here, again, as with the manifestations of hysteria in general, no hard and fast rule can be given, nor can anything certain or constant be said about the salivary and sweat secretions, since they are equally subject to variations.

The combined hysterical manifestations — that is, those originating in the brain as well as the spinal cord—consist of the so-called "paroxysms" or "attacks," in which consciousness is not lost, as in epilepsy, but which are associated with convulsions. Vague pains, ructus, yawning, the globus hystericus, ischuria, etc., may constitute the premonitory signs, which are immediately followed by violent respiratory movements, regular respiratory spasms, with laughing, screaming, weeping, barking, and finally the climax is reached in muscular spasms and convulsions resembling those of epilepsy. During such paroxysms the whole body may be thrown from side to

side, and it may be impossible to restrain the patients, because they exhibit a strength far greater than that which they ordinarily possess. After the fit, which may last from half an hour to an hour, has spent its force, there follows a condition of general prostration, which usually does not last long and is frequently accompanied by polyuria. It is just these attacks which make the " home treatment " for hysterical patients so very difficult or finally even impossible. It has been found by experience that the sight of such patients—the sight of the various contortions into which their bodies are thrown and the grotesque positions they assume—has an injurious effect on the other members of the family, especially if there are young girls among them. Such a scene and the consequent mental excitement have been known to cause similar attacks in other girls.

Duration and Course.—The duration and course of hysteria are by no means uniform, although this much may be said, that it is always chronic and may last for years and tens of years. There are patients who from the time of puberty until they have passed the prime of life and (if they are women) until after the menopause, are hysterical, and thus never attain to the full enjoyment of life. At the beginning of the trouble there is usually nothing more than a certain tendency to nervousness, a certain proneness to eccentricities, annoying to the patient and still more so to the family. Soon, various pains, which are apt to frequently change their seat and to vary in intensity, make their appearance, and certain respiratory phenomena, perhaps shortness of breath or a barking cough for which physical examination reveals no cause, begin to attract our attention.

Severe motor disturbances are by no means noted in all cases; even contractures are not very common. On the other hand, there are few cases in which the sensibility does not at various times undergo striking changes. Anæsthesias and analgesias alternating with hyperæsthesias, and neuralgias, especially of the joints, and persistent headaches, all help to sour the disposition of the patient. During menstruation the condition is usually aggravated. The patient is still more excitable than usual, and her complaints are louder. In those, in whom the paroxysms are an important feature of the case, this is more especially true, and sometimes the first menstrual period is the

signal for the first attack, which is at regular intervals followed by others. In many cases of hysteria, "attacks" never occur. The patients, indeed, may without any provocation have fits of crying, laughing and screaming, but no convulsions. In old age, at a time when the patients begin to become sexless, the hysterical phenomena fade. As the hair turns gray the disposition becomes calmer and more equable, and even egotistical, exacting, peevish women, who have tormented their families continually and who were extremely hard to manage, become yielding, amiable old ladies after the hysterical manifestations have once left them. Still there are, unfortunately, exceptions where these persist even after the seventieth birthday.

The prognosis may be inferred from our description of the course of the disease. Doubtful as it always is, it is made still more gloomy from the fact that persons who have for years suffered from hysteria are apt to be subject later in life to actual organic diseases, especially of the brain (Feldmann, Inaugural Dissert., Leipzig, 1887).

Hysteria was thought, as the name indicates, to occur exclusively in members of the female sex. That it is more prevalent among them there can be no question, but Charcot and his pupils have shown convincingly that it does occur in men and boys, and that, too, much more frequently than might *a priori* have been supposed. From him we have learned that it occurs frequently among the French soldiers. Further investigations may prove that this would hold good not only for the French, but also for other armies. Age seems to have much less influence than was at first assigned to it. Hysteria in children is by no means rare. The full development of all hysterical manifestations in the young, who are far from being sexually mature, proves that puberty and the sexual organs are of less importance in the causation of this neurosis than has formerly been supposed.

Diagnosis.—The diagnosis of hysteria may at one time be very easy, at another we may encounter no inconsiderable difficulties. When we have an array of ever-varying symptoms occurring apparently without order, when the patients complain to-day of this, to-morrow of that, while the physical signs show no grounds for their troubles, it does not need an expert to suspect and diagnosticate a hysterical condition. If, on the other hand, the disease sets in suddenly without previous dis-

orders of any kind, in one case with a hemiplegia, in another case with a severe hip trouble, it may be by no means easy to say whether and, if so, why the hemiplegia is of hysterical origin, and whether or not the joint affection is to be regarded as a hysterical coxalgia.

The following points will in the majority of cases be found sufficient to clear up any difficulties which the diagnosis presents :

With regard to the cerebral symptoms, and more especially those belonging to the affections of certain of the cranial nerves, we have in previous chapters pointed out some features characteristic of the hysterical varieties. It will be necessary in every separate case to exclude scrupulously anatomical lesions and to determine whether there are in addition to those belonging to the cranial nerves other symptoms which point to a hysterical condition. If such be found, and more especially if our objective examination gives negative results, the diagnosis of hysteria is warrantable.

These rules are particularly applicable where we have to decide whether a hemiplegia is hysterical or due to a lesion in the internal capsule, whether a contracture has to be regarded as hysterical or cortical (page 184), and whether the disturbances of the respiratory organs depend upon diseases of the lungs or the larynx or are to be referred to a neurosis of the vagus or of the recurrent laryngeal nerve.

The recognition of the hysterical nature of spinal manifestations belonging to the motor apparatus may give rise to the greatest difficulties. It is upon the electrical examination that we must rely in deciding whether the paralysis of an extremity depends or not upon a peripheral cause—that is, upon a neuritis. A well-marked reaction of degeneration always points to a chronic inflammatory condition. The age of the patient is of some value. Hysterical paralyses occur between the ages of fifteen and thirty, and more particularly in women. Further, we observe almost always associated with hysterical paralyses grave sensory disturbances which are not necessarily present in the other kinds (cf. Lumbroso, Lo Sperimentale, Firenze, 1887 ; reference, Neurol. Centralb., 1888, 7). The existence of muscular atrophy is not sufficient to determine the organic nature of the paralysis because an atrophy of muscles does not exclude hysteria, as we have pointed out on pages 234 and 511 (Brissaud, Arch. de physiol. norm. et pathol., April, 1887, page 339).

Contractures, if of hysterical origin, set in suddenly, and are almost always accompanied by other hysterical manifestations, meteorism, ovarian hyperæsthesia, and ischuria. Where such symptoms are absent we must be very careful in our examination and take into account the possibility of an anatomical lesion either of central or of peripheral origin (cf. Blocq, Des contractures, Thèse de Paris, 1888; Progr. méd., 1888, xx, page 397).

Hysterical muscular spasms may be taken to be tetany, as the case of Caiger, in the Lancet of August 20, 1887, shows. To the frequent occurrence of rhythmical spasm in certain groups of muscles in hysteria, Pitres has drawn attention in an article in the Gaz. méd. de Paris, 1888, 13.

Trembling and shaking movements, which somewhat resemble those of intention tremor as they become more marked on voluntary motion, have been noted, but are rare. The possibility of mistaking such conditions for multiple sclerosis (or *vice versâ*) should, however, always be kept in mind. In our account of the latter disease we shall come back again to the points for the differential diagnosis between the two conditions.

The sensory changes in hysteria, the anæsthesias, affect, as we said, not only the skin, but also the deeper tissues, so that needles may be inserted down to the bone without being felt. Usually all qualities of sensation take part in the disorder, so that the so-called muscular sense is also lost and the patients are unable after closing their eyes to give any account of the position of their limbs. Pronounced anæsthesia is found during the hysterical paroxysms. An anæsthesia extending over the whole body and taking in all the mucous membranes is almost always hysterical in nature.

Hyperæsthesias and neuralgias occurring in hysteria are typical in that they are very changeable, so that to-day cranial, to-morrow spinal nerves are the seat of the pain. Neuralgias of joints, if organic disease can be ruled out and if they are very obstinate and resist all the ordinary therapeutic measures, may be safely looked upon as hysterical. The "attacks" may be mistaken for epileptic fits. The important point to remember in this connection is that in the hysterical attacks consciousness is never lost as completely as in epilepsy. Biting of the tongue is an exception in the former. The hysterical attacks are, moreover, attended with noisy laughing and crying, etc., while epileptics, with the exception of the initial cry

(which is not constant), pass through the whole convulsive stage quietly and without uttering a sound. It has been claimed that there never occurs an elevation of temperature during the hysterical seizure, while the epileptic fit is accompanied by a slight rise, 1.2° to 1.8° F. This statement can not easily be controlled, and certainly needs further confirmation. Finally, it should be remembered that hysterical attacks may in some instances be produced by pressure upon the ovaries or the testicles, while in epilepsy this is never the case.

Pathogenesis and Ætiology.—About the nature of hysteria we are absolutely in the dark. Not one of the many attempts to explain the disease can be regarded as more than a vague hypothesis. This one fact may be regarded as certain, that the existence of grave anatomical changes is excluded or, at any rate, is highly improbable, otherwise the suddenness with which the symptoms come and go would be absolutely inexplicable. The old idea that the uterus must be held responsible in every case and under all circumstances for the disease, which was consequently called hysteria (ὑστερία), has been shown to be untenable by the number of cases observed in men and young children; and the more cases we see, the clearer it becomes that the hysterias occurring in males and in little children furnish a considerable proportion of the total number, and the more ridiculous becomes the term "hysteria," which sooner or later will be given up completely. The influence of the sexual organs on the disease will be discussed later, but we would state emphatically that the opinion that these are always the starting point of the disease is indefensible.

But how shall we explain the disease? If we agree that all symptoms of hysteria have certain characteristics in common, they may perhaps all together be traced to an increased excitability of the whole nervous system, to the quicker response to stimuli from without and within. Just as we have morbid conditions in which the excitability of the nerves and the muscles to the electrical current is found to be increased, we may imagine also an analogous condition in which all the nerves, including the nerve elements of the central organs of the brain, especially of its cortex, those of the spinal cord, and also of the peripheral nerves, are in a constant state of abnormal or pathological excitability. That in such a state the imagination plays an important *rôle* is self-evident—not, however, in the

sense that all the sufferings of which the patient complains are imaginary and merely depend upon the imagination ; we rather mean that, in the condition described, mental conceptions are formed and disappear more quickly and are constantly changing. Such a quick and unnatural change can not but exert an unfavorable influence, first upon the mind and disposition, and later upon the bodily condition.

In reality it is in the majority of cases a disturbance of the psychical equilibrium which produces the disease. It is not impossible that careful study of the ætiology may do much toward a clearer understanding of the nature of the malady ; hence it seems in place to say a few words about the causes, as far as our limited knowledge will allow. They may be subdivided into direct and indirect. To the former belong a hereditary, physical as well as psychical, predisposition of the individual. There is no doubt that only those persons can become hysterical who are *a priori*—that is, from birth—so predisposed, because they have a nervous system which presents the peculiarities which we have just described. This congenital, because hereditary, predisposition finds favorable conditions for further development in (*a*) sex, (*b*) age, (*c*) education, (*d*) nationality or race of the patient. That the female sex and those just arriving at the age of puberty are prone to the disease we have said before, although the male sex and other periods of life besides that of puberty are by no means exempt. The hysteria which occurs in early childhood, and which has been observed between five and ten years of age, deserves special study (lit., page 530).

Much must be attributed in the causation of hysteria to a faulty education. The brain may be overtasked at the expense of the body, and, in consequence of too little firmness on the part of the parents, capriciousness, inconsiderateness, lack of truthfulness, of energy, and of will power, are fostered in the child, and, finally, when the children have behaved badly, the mysterious threats, especially of injudicious servants, of sending after them wild beasts, ghosts, "the black man," etc., can drive them into such a chronic state of fear that they can not go into a dark room without palpitation and the most intense feeling of terror. All such and many other mistakes in the early education of the child become indirectly causes of hysteria. As to race, the Slavonic (Poles, Russians), the Latin races (Frenchmen, Italians), and, above all, the Semitic, are

more liable to hysteria than the Teutonic. The severest forms of hysteria are seen in French women and in Polish Jewesses. This may depend upon the national characteristics; the lively, impetuous temperament which we find on an average more frequently in the Slavs, etc., than in the Germans, forms a particularly favorable soil for the development of hysteria.

Among the direct causes, disorders of the sexual organs play the most important part, and in both sexes this factor is equally potent. We must not think that the affection, which, especially in women, may, from a gynæcological standpoint, be very insignificant—for instance, a flexion or a change in position of the uterus—has in itself much to do with the matter; it is much rather the idea that the trouble exists, and the anxiety lest it should interfere more or less materially with coitus and parturition, which constitute the direct cause of the depression of spirits. The conjugal obligations—coitus, pregnancy, parturition—play such an important *rôle* in the life of every woman, if she has not missed her calling, that the mere idea that the sexual organs are diseased or incapable of performing their function is sufficient to give a severe shock to her happiness. In a man it is much less the *potentia generandi* than the *potentia coeundi* that causes him anxiety. The above-mentioned psychical impotence, if it exist for a long time, in itself suffices to bring about a hysterical condition, and sexual neurasthenia is not rarely accompanied by pronounced hysterical manifestations, so that we can well speak of a coexistence of the two diseases.

Secondly, traumatism ought to be mentioned as a possible cause of hysteria, and not only injuries which affect the body, but also those which concern the mind and the feelings—psychical traumatism. Above all, injury to certain parts of the body is interesting, inasmuch as such parts frequently become the seat of a hysterical affection; thus we may after a lesion of the hip joint, after the injury has long been recovered from, find a hysterical coxalgia, and so forth. The psychical traumatism may be of such a nature as to have an immediate influence, or may act gradually and insidiously. Among the former we have fright, emotions of anger, rarely of joy; to the latter belong grief, anxiety, wounded self-respect or vanity, etc. Finally, there are traumatisms which, besides the bodily injury, also produce a mental shock; among these may be mentioned injuries by lightning and the effect of accidents

(fire, railroad accidents, etc.). The resulting "traumatic neuroses," which in the case of railroad accidents have also been called railway spine, have in recent times been studied by Strümpell and Oppenheim; they are, without question, hysterical in origin, although they present certain features which do not belong to the clinical picture usual in hysteria. Cases in which, in addition to the purely functional disturbances, definite anatomical changes in the nervous system are developed in consequence of the accident, as, for instance, atrophy of the optic nerve, constitute the intermediate forms between the functional and the organic diseases. Of one thing we may be assured, viz., that a psychical will be more potent in its consequences than a physical traumatism; the morbidly altered mind, by its abnormal reaction to the bodily ailments, constitutes and causes the lasting disease (Oppenheim); through a sort of auto-suggestion the sensory and motor paralyses develop in a similar way as the paralyses, and the like, which Charcot produced in hystero-epileptics by means of suggestion.

The symptoms that appear after a person has been struck by lightning sometimes resemble the array of symptoms observed in traumatic neuroses. Paralyses in the nerves of special sense, and motor and sensory paralyses, appear and last for a shorter or longer time. In the spring of 1889, when thunderstorms were so frequent, I had the opportunity of examining a man who, as a consequence of being struck by lightning, on recovering consciousness after three quarters of an hour, presented loss of the power of sight and smell on the side where the lightning had entered and left the body, while on the same side hearing was diminished, and there was total anæsthesia. These symptoms were associated with an obstinate insomnia. By hypnotism, frequently repeated, we were enabled to lessen this insomnia, and under the use of the galvanic current and the faradic brush the hemianæsthesia disappeared. The nerves of special sense implicated became fully normal after a month's treatment. In this case motor disturbances were never seen. For further symptoms in such cases and for the post-mortem conditions found after death by lightning, we would refer the reader to Schmitz's article in the Deutsche med. Zeitg., 1887, 73, 74, where further references on the subject may be found.

The symptoms of "railway spine" are, as we might con-

jecture from what has been said already, as manifold and as changeable as those of hysteria. Motor disturbances, manifesting themselves usually in a general motor weakness, are associated with abnormities of sensation, pains, especially in the head, paræsthesias, hyperæsthesias and anæsthesias of different parts of the body, not excluding the nerves of special sense; contraction of the field of vision, a certain decrease in the acuteness of sight, photophobia, disorders of the color sense, hyperæsthesias of the nerves of hearing, smell, and taste, have been observed. Cutaneous anæsthesias, mostly on the back over irregular areas, and even pronounced hemianæsthesia hysterica, anæsthesias extending over the head, neck, and upper part of the chest (the so-called "doll's head" form), are often demonstrable, changes which vary with regard to their seat and extent in the same individual, so much so that frequently repeated examinations may give different results. The skin and tendon reflexes (as is the case in hysteria) are variable; in the same way micturition is sometimes normal, sometimes disturbed. In walking, and in general in all movements of the body, the patient avoids as much as possible all motion of the vertebral column. He fixes the trunk and moves forward holding his back stiff, and in every change of position makes as much use as possible of his hands (Oppenheim). Psychical anomalies, although they may be entirely absent, are usually present; they may manifest themselves by feelings of anxiety, depression, abnormal irritability, and hypochondriacal notions, which may eventually go so far as to lead to suicide; sometimes we encounter the so-called phobias, of which agorophobia is one of the most common.

In every traumatic neurosis we must take into account the possibility, if not of complete simulation, at least of a tendency to exaggeration on the part of a patient; this is a difficult question to deal with, and great caution is necessary in view of a possible deception, but it would be quite wrong to go to the other extreme and regard all the patient's complaints and woes as belonging to the realm of fiction, and designate them as falsehoods. The legal bearing of such accident cases makes it here more especially the absolute duty of every physician to make a careful and unprejudiced examination, and it is not sufficient to look the patient over once, but a repeated study of him is necessary before giving a certificate.

Finally, we would add that under certain circumstances the

occupation of the patient may lead to hysteria. Working in certain poisons, especially quicksilver and carbon bisulphide, is said to be especially dangerous. Indeed, French writers speak of a definite mercurial and of a carbon-bisulphide hysteria. We shall return to this subject later.

Treatment.—The treatment of hysteria is always a very tedious matter, and for the physician sometimes the most tiresome and thankless task imaginable and one to which he should only devote himself if he be assured of the implicit confidence of his patient, as far as this is possible in the case of hysterical individuals. This confidence is indispensable, because the treatment of the disease does not consist in the main in the administration of drugs in a routine fashion, valerian, asafœtida, castoreum, and the nervines, but must depend more upon the psychical influence by which we endeavor to diminish the abnormal sensitiveness of the patient to external and internal stimuli, to arouse her energy and to strengthen her will power. This is, we admit, much more easily said than done, and we shall often have to confess that the patient's views about her trouble have not changed in the least, that she is as irritable as ever, that her moodiness and capriciousness are in no way improved in spite of all our lectures—in a word, that we have obtained no positive result after " preaching reason " for hours. Still, we must not allow ourselves to become discouraged, but ever again and again renew our efforts to obtain the desired end.

If we clearly see that these are fruitless, and especially if we are convinced, as is often the case, that the family, far from assisting the physician, are virtually acting against him during his absence, we must impress upon them the necessity of removing the patient to some institution. French physicians lay the greatest stress upon isolation in such cases and are inclined to attribute the relatively favorable results of their treatment to this factor. In this country people are not so easily persuaded to agree to this procedure as in Paris, where in the city itself or in the suburbs there are various admirably conducted institutions which only receive hysterical patients. With us, therefore, home treatment ought first to be tried. In France this is usually discarded from the first. It is a different matter, of course, if we have not to deal with a mild degree of hysteria, but with hystero-epilepsy and major attacks. Then a

transference to an institution, as soon as practicable, ought to be urged.

The bodily treatment may either be general (that is, directed to the nutrition, to the condition of the blood, and the strength of the patient) or symptomatic (that is, intended to relieve the troubles of the patient as they arise).

With reference to the nutrition, it was Weir Mitchell and Playfair who first recommended absolute rest in bed, with massage, electricity, and copious feeding. Their patients were forced to take considerable quantities of milk, meat, bread, etc., and it was found that with the increase of the body weight the hysterical symptoms and attacks diminished. Of late years good results have been obtained from this practice by Binswanger (Allgem. Zeitschr. f. Psych., 1883, xl, 4), and the communications of Leyden (Berl. klin. Wochenschr., 1886, xxiii, 16) and Burkart (ibid., 1886, 16) should encourage us to further trials with this method, although as far as my own experience goes the results have by no means always been brilliant. The cases in which the excessive ingestion of food was badly borne and led to a disagreeable gastric catarrh were by no means uncommon, and even where the food was well assimilated the desired results were not always obtained. That much attention has to be paid to the nutrition there can be no question, and the increase in the body weight usually can be regarded as a favorable indication. To attain this, however, in many cases, not absolute rest, but, on the contrary, systematic muscular exercise is needed. Well-regulated home gymnastics, undertaken according to definite principles (Schreber, Angerstein, and Eckler), are to be preferred and will be often found an excellent means of combating the distressing insomnia.

In certain cases, to be selected, of course, with care, general faradization as recommended by Beard and Rockwell is of great service. The patient for this purpose is placed upon a stool with his bare feet upon a moist large electrode, which is connected with the negative pole of the secondary coil. With the anode, which consists of a large sponge electrode, all parts of the body are treated in succession. Instead of the moist we may avail ourselves of a dry electrode in the form of a soft brush. The pain which is caused by the latter method is, at least with strong currents, quite considerable ; nevertheless, the method deserves warm recommendation in certain hysterical affections and especially in joint neuralgias.

About the influence and the value of static electricity as a therapeutic agent our experience is not sufficient to warrant any definite conclusions. It is not easy to judge of the usefulness of the treatment, as it is usually combined with other measures, the therapeutic significance of which must not be left out of consideration. Whether the action of static electricity differs essentially from that of the faradic and galvanic current, and, if so, in what this difference consists and under what circumstances the one or the other is indicated, we are not as yet in a position to say. The great inconvenience and expense of the apparatus militate greatly against its general introduction.

It is rare that we treat a case of grave hysteria without at one time or another during the course of the disease being obliged to resort to massage; for one thing, because the patient desires as much variety as possible—but at the same time we must not overlook the fact that by its use many of the patient's troubles are considerably relieved. This is not the place to enter into the minute details of this method of treatment. They may be found in the writings of Schreber, Reibmayr, Zabludowski, and others.

The cold-water treatment is indicated where we desire to harden the constitution against external influences, changes of temperature, etc. We should be very careful, however, with low temperatures, and the water with which the patient is sponged or in which hip baths and the like are taken ought to be at least 80° F. For the use of ice-cold douches, in the way recommended by the French, certain facilities are requisite. The pressure of the water should be very great and the duration of the bath should be so short (from ten to fifteen seconds) that the patient has not time to become aware how cold the water really is. I have watched this practice repeatedly in some of the well-known hydrotherapeutic establishments of Paris and have had occasion to notice the immediate beneficial effects following the application. The lasting results, as Charcot and others are quite convinced, are so marked that (in Paris) cold douches are considered to be indispensable in the treatment of hysteria. It would be a very desirable thing if the necessary arrangements for this treatment could be introduced into our hydrotherapeutic institutions. The ordinary shower bath, which comes down upon the patient just about like rain, is, of course, not sufficient. In the treatment of some

of the particularly distressing symptoms it is, of course, in the first place the paroxysms which deserve our attention, because they, more than any other of the hysterical phenomena, are liable to render home treatment almost impossible. We may sometimes be able to cut short an attack by steady pressure with the hand over the ovaries continued for some time, but this can be better accomplished by allowing the patient to inhale a little chloroform. To guard against a repetition of the attacks we have no reliable means, yet cool prolonged baths with affusions of colder water deserve a thorough trial. If these do not seem to be beneficial, and if the patient complains, before the onset of every attack, of pains in the ovarian region, and if we, moreover, can succeed in bringing about an attack by pressure over the (tender) ovaries, the question of oöphorectomy has to be considered. The family relations, especially the sterility which naturally follows the operation, have to be taken into consideration, nor should we forget that the operation has often by no means been followed by the desired effect, although the fact that it frequently exerts a favorable influence, as Hegar and Schröder have seen, can not be questioned. Whether the ovaries are actually diseased or not is altogether of minor importance. It is the presence of pain immediately before or after the attack in the region of these organs which should suggest an operative interference. Cauterization of the clitoris, advised by Friedreich, is a procedure which should only be resorted to in the most exceptional cases. In all instances the sexual organs ought to be carefully examined, and small operations, such as dilatation of the cervical canal, reposition of the uterus when in a position of flexion or version, if indicated, should be undertaken. Vaginismus, if it exists, should also be treated.

The motor and sensory disturbances have to be met in the manner indicated above. In cases where we suspect malingering or willful exaggeration, procedures which are disagreeable or even painful are to be preferred—for instance, the cold baths, the faradic brush, the actual cautery, etc. The more minute details of the treatment must be left to the personal tact of the physician, whose capability of individualization, of treating every case by and for itself, should make it unnecessary for us to enlarge upon all the principal phases of this disease. With regard to the internal medication, let it suffice to warn against the use of narcotics, especially morphine,

which can not be given in a disease of such long duration in effectual doses without creating the habit.

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CHAPTER III.

By epilepsy in the stricter sense of the term we designate a functional neurosis, the seat of which is still unknown, characterized chiefly by attacks of unconsciousness, with or without convulsions. In its wider sense the designation also comprises certain affections which are due to lesions of the brain and more especially of the brain cortex. For these it is less the loss of consciousness than the appearance of convulsions which is characteristic. To the latter kind of epilepsy belongs the so-called Jacksonian, partial, or cortical epilepsy (cf. page 184). A most conscientious examination and a correct answer to the question, whether the attacks constitute alone the disease or whether they are merely a symptom of a primary affection which is attended with still other manifestations, are for the patient as well as for the physician of the greatest importance, as this will be of importance for the treatment as well as for the prognosis. We shall first only give our attention to the so-called " genuine " or idiopathic epilepsy.

The manner in which an epileptic patient can spend his life, the possibility of doing justice to the requirements of his calling and of being a more or less useful member of society, the outlook for improvement or even recovery—all these questions depend in the main upon the "attacks" to which he is subject, on their nature, their duration, their frequency, their after-effects, and so forth. Hence it is our first duty in taking charge of a case of epilepsy to study carefully the attack itself.

Symptomatology. The "Attack." — There are cases in which the attack occurs suddenly and unexpectedly, so that the patient, until now in apparently perfect health, falls to the ground as if struck by lightning. In others—more numerous—it is announced, so to speak, by certain premonitions, which, to maintain Galen's old expression, we call auræ.

In the study even of the aura we can not help being struck with the fact which, on a closer examination of the attack, is still more impressed upon us, that no two cases of epilepsy are alike, that almost every one has its own peculiarities, so that a general description is almost impossible. The premonitions are countless and many attempts have been made to divide them into classes. Even if we have obtained a classification we are far from possessing with it a description of all.

First of all, we may subdivide the auræ into psychical and somatic. In the former case the patient may either become surprisingly quiet and look meditative, or he may present signs of excitement, walk anxiously up and down the room, and seem bewildered. The transition from the aura to the actual pre-epileptic disturbance of consciousness, the pre-epileptic insanity, is not appreciable (Mendel, Eulenberg's Vierteljahrsschrift., N. F., 1885, Bd. 42, Heft 2). This prodromal state may extend over several hours, although it may not last any longer than thirty seconds or a few minutes. In two cases the patients told me that, immediately before the attack, reminiscences of bygone days forced themselves upon their minds, and that portions of their past lives rapidly passed before them. A psychical aura of this kind is rare. Sometimes an irresistible desire in the patient to run constitutes the aura. Just as we shall see in the form of epilepsy called epilepsia procursiva, the patient escapes from his home and runs great distances. While he is running he is seized with the attack. Midway between the cases where there is a psychical and those where there is a somatic aura we shall place those instances in which the patients complain of vertigo, violent headache, and slight disturbances of consciousness, symptoms which also may last but a very short time, and which, indeed, may be of such brief duration that the patient has not time to guard himself against falling. Here, too, belong the hallucinations which occur in the domain of the nerves of special sense, which we are accustomed to call "special sense" auræ. The patient hears, sees, smells, tastes things which either are not there at all or are in reality different from what he deems them. I know instances in which immediately before the fit the patient thinks he is standing in a sea of light; most intense brightness surrounds him, and he is cognizant of wonderful light effects. In other cases again the patient thinks he is standing amid utter darkness, he sees noth-

ing, and the densest obscurity reigns everywhere. To this class belong the instances reported by Heinemann where bilateral amauroses constituted the aura (Virchow's Arch., 102, 3, 1885, page 522). The optic as well as the auditory auræ vary in different patients. Sometimes they hear delightful melodies, sometimes they find themselves amid the wildest tumult of confused noises. Complete loss of hearing, transient deafness, which would be analogous to the transient amaurosis, I have never had an opportunity to note.

Sometimes, not often, the patients imagine they hear distinctly different voices. Then the aura is a genuine hallucination and infringes upon the domain of pre-epileptic insanity. Well-pronounced gustatory and olfactory auræ do occur, but are decidedly less frequent than those just described.

The somatic auræ are either motor, sensory, or vaso-motor. The motor more frequently consist of symptoms of irritation than those of paralysis. There are isolated twitchings in the fingers or toes, in the arms or legs, which progress from the periphery to the centre; contractures in some fingers have also been observed. In addition to or in the place of these there may be twitching movements of the head or neck, twitchings of the facial muscles, or well-marked strabismus. Paretic symptoms, heaviness and fatigue in the extremities, are more rare. Spasm of the glottis, bronchial asthma, palpitation of the heart, retching—all have to be regarded as varieties of motor auræ.

The sensory auræ consist of peculiar paræsthesias in the extremities, formication, numbness in the fingers, the patient feeling as if these were working their way up to the head or to the heart. Not uncommonly they are associated with a pronounced feeling of anxiety and oppression. The sensations which appear in the extremities, sometimes in the fingers, sometimes in the toes, are extremely variable, from a pleasant slight tingling to a painful burning and stinging, which, as we have said, proceeds from the periphery to the centre.

In vaso-motor auræ the hands become cold and pale, the cutaneous veins look less full than normally, and the patient complains that he is getting cold. A general feeling of chilliness, associated with chattering of the teeth, has also been noted (Douty, Lancet, March 20, 1886). In other instances, possibly on account of a paralysis of the vaso-motor nerves, blushing of the skin and sweating occur. The degree of full-

ness of the cutaneous vessels and the larger veins of the skin is in some cases sufficient to tell the patient whether or no the will shortly have a fit.

Innumerable transition forms and countless combinations of different kinds of auræ occur. No definite laws can be given, and we must here again recall the inexhaustible varieties of the prodromes by which the attack may be ushered in.

The question whether the origin of the aura be central or peripheral can as yet not be answered. Certain facts point to the first possibility, others to the second (cf. Oliver, Lancet, April 21, 1888, page 769). That the aura may have an anatomical basis is proved by the case reported by Hughlings Jackson (Brit. Med. Journal, February 23, 1888). The patient, a man of fifty-three years of age, complained regularly of a horrible, indescribable stench which immediately preceded every attack. At the autopsy a tumor was found situated in the temporo-sphenoidal region. We would remark, by the way, that this case is a point in favor of Ferrier's localization of the sense of smell.

The attack itself is characterized by complete loss of consciousness, and is sometimes ushered in by an initial piercing cry or a noise like the roar of a wild beast which the patients emit at the moment of falling. This cry is by no means to be regarded as the expression of fear or surprise, as it does not occur until consciousness is lost and is a reflex act. It is observed in about fifty per cent of all cases, while in the remainder it is either absent or replaced by tears. A tonic muscular contraction accompanies the cry. The head is at the moment of the fall drawn backward or to one side, the jaws are pressed together, the back is spasmodically curved, and the fingers are clenched over the adducted and flexed thumb. Respiration ceases, because the muscles performing the function take part in the spasm, and the face becomes discolored and cyanotic. A convulsive tremor runs over the whole body, and in the muscles of the face as well as in the rigid extremities twitchings begin to appear, which spread, and spare no part of the body. The head is violently knocked against the floor or the couch, the tongue rolled around in the mouth, protruded, perhaps, and retracted alternately, so that it is often injured by the teeth; the eyeballs are deviated, the pupils dilated and inactive. Arms, legs, and trunk are now the seat of violent, irregular, rapidly changing jerkings. Corneal and skin reflexes

are lost. The tendon reflexes can be obtained if the tetanic rigidity of the extremities allows it. The pulse is slightly quicker, the respiration greatly hurried. With each expiration the saliva, often foaming and mixed with the blood coming from the injured tongue, bursts forth and covers the lips. The temperature remains normal. In more protracted cases it may rise from one fifth to half a degree Fahrenheit. The involuntary evacuation of urine and fæces, possibly also of semen, is not rare. In one case only have I seen the attack regularly associated at its onset with vomiting.

Gradually the body becomes covered with sweat in consequence of the excessive muscular strain; next, the convulsions lose some of their violence, the limbs gradually become less rigid, the cyanosis disappears, respiration, though it may still be difficult and snoring, becomes more regular, the coma abates and passes insensibly either into a deep, long sleep or gives place immediately to complete consciousness, so that in some cases the patient may in a few minutes again be in an apparently perfectly normal condition, without, however, having the slightest idea of what has been going on during the attack.

We have said that the symptoms immediately preceding the attack present an endless variety of forms; the same must be said of those that belong to the period following it. These "post-epileptic" phenomena may again be divided into psychical and somatic. The psychical phenomena are very interesting, because they are not always of the same intensity, but may assume all gradations between a complete insanity ("post-epileptic insanity," post-epileptic moria, Samt) and a slight bewilderment. In the first case the patient has to be regarded as a madman, and must not be held responsible for his actions, not excluding any crime that he may commit at such times; in the latter he resembles a drunken man, who, although he later can not remember what has happened, will answer questions if they are repeated often enough and in a sufficiently loud tone. Not uncommonly there exist immediately on waking up speech disturbances, either in the form of a motor or sensory aphasia, which lasts from a few minutes to several hours. Total aphasia following the attack has also come under my notice, and I have seen it persist for half an hour. The patient appeared to have regained consciousness pretty well, he understood, apparently, the questions which were

asked him, but was not able to answer them in any other way than by signs. Fürstner has reported instances of post-epileptic stammering (Arch. f. Psych. und Nervenkrankheiten, 1886, xvii, 2).

Among the somatic post-epileptic phenomena there is, besides the difference in the size of the pupils, which is of some value for the diagnosis of nocturnal attacks occurring during sleep, a concentric contraction of the field of vision, which may last for twenty-four hours. Of this I have been able to convince myself several times positively. Further, there are certain conditions of motor irritation, "cortical movements" (Rindenbewegungen of Zacher), which either consist of typical clonic twitchings, or of choreoid or athetoid movements, and which may persist for hours. Contractures, occurring more frequently in the upper than in the lower extremities, usually on one side, have only been observed in exceptional cases (Lemoine, Deutsche med. Ztg., 1888, 20). Among the vaso-motor changes there are circumscribed reddenings which may occur symmetrically on both sides of the body in the most diverse places. Transient increase of the patellar reflex, transient albuminuria and violent vomiting are common after epileptic attacks.

As to the time at which the attack may be expected, we may broadly say that there is not a moment in the life of the patient in which he can feel safe from them; that any particular time, either of the day or of the night, is especially dangerous in this regard can not be maintained. This much only can be said, that in some individual cases the fits only occur during the night while the patient is in bed and asleep; this so-called epilepsia nocturna possesses great practical importance, because it may persist for a very long time unremarked and unrecognized, especially if the patient sleeps alone. If such be the case, the diagnosis can then only be made from certain characteristic signs observed in the morning—from the pain of the bitten tongue, the dull headache, the slight extravasation of blood into the conjunctivæ, or the unequal pupils (*vid. supra*). In one of my cases of nocturnal epilepsy there occurs after each attack a deep-red spot, the size of the palm of the hand, on the forehead, which does not begin to fade until one or two days have passed. For years the attacks may be confined to the night, and may go on without interfering to any extent with the patient's business and social life. Above all,

he is not exposed to the usual injuries caused by the falls, but he never can feel absolutely certain that some time or other an attack may not occur during the day. These nocturnal fits are heralded by an irregular respiration, snoring, grunting, or moaning. Convulsions may not occur at all, but the whole body gets into a condition of tetanic rigidity which is followed by a relaxation of the muscles; during the whole time the patient does not awake, and has no consciousness of what has been going on.

There are certain things which seem to exert an unfavorable influence upon the severity and the frequency of the fits, and against which the patient must be strictly and repeatedly cautioned. Of these the most important are sexual excitement, alcoholic excesses, psychical emotions, mental strain, etc. The indulgence in, heavy indigestible food is harmful, especially in the evening. After imprudences of this kind the attacks may be found to come on more frequently, and at times when otherwise they would have undoubtedly not occurred. Whether the climate has anything to do with the fits we are not sure, and the idea of the supposed influence of the moon must be relegated to the domain of the unknown. It is interesting to note, however, that when an epileptic is taken ill with typhoid fever, pneumonia, facial neuralgia, etc., he may hope to enjoy immunity from the attacks as long as these diseases last.

On the other hand, there are certain things which exert a favorable influence, either by aborting or preventing for certain periods the occurrence of the attacks. How these factors work is quite inexplicable. Among the former may be mentioned the application of a tight bandage or strap to the part of the body—e. g., the finger or hand—in which the motor aura occurs; to the latter belongs frequent epistaxis, as I have repeatedly had occasion to observe; if it was profuse it seemed to produce an intermission in the occurrence of the attacks which lasted for a relatively long time.

Besides the classical attack which we have just described, and which is known as "*grand mal*," there occur rudimentary abortive attacks, as it were, which have received the name "*petit mal.*" Of this latter kind there exist countless varieties. There may be nothing more than a momentary vertigo, without any loss of consciousness; this is then termed epileptic vertigo; or in place of or following this there may be a short

loss of consciousness, lasting but a few seconds, the "*absence*" of the French writers, of the onset and the duration of which the patient is unable to give any account. An individual may in the middle of any kind of occupation—speaking, eating, reading, and so forth—suddenly stop what he is doing; for an instant he stares vacantly before him, remains as he is, standing or sitting, and immediately after the "attack" resumes his occupation as if nothing had happened; the unfinished sentence is after a short pause completed, the spoon which was ready to bring the food to the mouth after a short stop reaches its goal. If an "*absence*" occurs to the patient on the street when he is out walking, he keeps on mechanically, loses his way perhaps, and only finds it again when consciousness returns. The instances in which such periods take in a much longer time, during which the patients undertake voyages, spend money, transact business of which they are not conscious later, or do things which are against their intention and entail disagreeable consequences, must also be looked upon as coming under the head of epilepsy. They are undoubtedly rare, and up to this time have only been carefully observed by French physicians, more especially by Charcot ("*automatisme ambulatoire*"). Insignificant as *petit mal* may seem, it often has a very deleterious effect upon the general condition of the patient, especially upon the mind; we should be cautious, therefore, with our prognosis.

There are still other seizures in which typical convulsions do not occur, but where the patient suddenly begins to walk first forward, then backward, or to run around in a circle ("*mouvements de manège*"), or spin round and round, or he may rush out of his house and run for long distances without knowing why or whither. This form, which has been described by Bourneville, Ladame, and others, is called "running epilepsy," epilepsia procursiva. It often appears in childhood, and later gives place to the usual classical attacks. Its frequent combination with moral insanity is interesting. Anatomical changes have not been found in the cases which came to autopsy up to the present.

Again, instead of the convulsive attacks, we may have from time to time transient psychical disturbances, which consist either of states of excitement or depression; in such instances we speak of "epileptic equivalents" (Samt). We must leave to the psychiatrists the task of investigating their cause and

their significance. From a medico-legal point of view these puzzling conditions possess great interest.

About the frequency of the paroxysms no definite statement is possible. There are people who during their whole life have not more than one, two, three, six, or ten attacks, and again there are others in whom they recur once a week or still more frequently. Sometimes there are certain periods in which they increase in frequency, and others of months or years during which only an occasional attack occurs. In rare instances, in periods of the former kind, the fits may succeed each other so closely that there may be one or even many every day. Before the patient has had time to regain his full consciousness another attack looms up. This is what we call the status epilepticus, *état de mal.* The temperature may rise steadily for from three to eight days as much as 5° to 7° F., so that it may reach 104° or 106° F. If, then, in the intervals consciousness does not become fully restored, but the patient remains dull and bewildered, there is very great danger that death may occur during the status epilepticus, and the friends should be made acquainted with the seriousness of the situation. Only in exceptional cases does recovery take place and the temperature fall to normal again (Witkowski, Ueber epileptisches Fieber u. s. w., Berliner klin. Wochenschr., 1886, xxxiii, 43, 44).

Course.—The course of the disease, the general condition of the patient in the intervals between the attacks, the influence of the attacks upon the mind and body—all these may present great variations.

The course is very chronic and the disease lasts in most cases years and tens of years. Frequently the patient is subject to the affection during his whole life. The earlier the first attacks make their appearance the less chance is there of their complete disappearance. In some cases of "late epilepsy," "*épilepsie tardive*," in which the affection does not begin until late in life, it may happen that the attacks completely cease as unexpectedly as they came on. Still, a course so favorable as this is rare and can never be predicted with certainty. If the disease has set in in early childhood, the influence of the period of puberty is generally very marked. The attacks become more frequent, and in women the increase in number is observed every month at the time of the menses until the time of the menopause. Pregnancy has little influence on the attacks,

according to my own experience; sometimes it appeared as if shortly after conception the number of fits was considerably lessened, while in other women there seemed to be no change.

The general condition in the intervals between the attacks is by no means the same in all cases. In some, fortunately not rare cases, the paroxysms do not cause any bad effects for years and nothing morbid can be discovered. The mental faculties develop normally or, if already developed, remain good. The disposition is cheerful, social intercourse is enjoyed, as there is nothing in the bodily condition to interfere with such pleasures. The presence of epilepsy does not necessarily prevent the full development of a genius, as is proved by the universally quoted historical examples of Cæsar, Alexander the Great, Rousseau, Napoleon I, and others.

In other instances the general condition in the intervals leaves much to be desired, and as a rule it is the psychical part of the man which suffers most unpleasantly. Either the disposition of the patient is changed for the worse, so that he is easily excited, irascible, suspicious, and peevish, unsociable, and disagreeable to those around him, or the mental faculties suffer, he becomes dull, slow in grasping ideas, indifferent, anxious, abstracted, and so unreliable in his work that he is no longer able to fulfill his duties as a man of business and as a good citizen.

In such cases we are sometimes able to note bodily defects, as, for example, abnormities in the formation of the skull, in the form of the auricle, in the condition and arrangement of the teeth, etc. Such "signs of degeneration," however, are frequently not present.

The final issue of the disease is almost always the same. The patient remains an epileptic all his life, from time to time having attacks, and finally dies from some intercurrent malady. The mental faculties may remain throughout, on the whole, good and the capacity of the patient for following his calling be retained. In other instances the mind becomes gradually impaired, so as to necessitate the transference of the patient to an institution, or again, in very exceptional cases, there may be complete recovery or, at any rate, so marked a decrease in the frequency of the attacks that the patient may well regard himself as cured. This cure may come about spontaneously or may be caused by some unexpected psychical emotion, particularly a fright. However, we should beware of being too

precipitate in calling a patient " well," because now and then even after intermissions of years an attack may again make its appearance.

Death rarely ever occurs during an attack, but indirectly the paroxysms may cause a fatal issue. The patient may receive during his fits serious wounds, or may fall upon his face and be suffocated, or fall into the water and be drowned. The average life of epileptics is considerably shorter than that of other persons.

Ætiology.—Although we may be able to mention some factors which seem to have something to do with the occurrence of the disease, we are unable even to hazard a conjecture in what manner in a given case this or that factor can evoke an attack, more particularly the first attack ; but our ignorance is not surprising if we consider that we have never as yet been able to find any anatomical changes of the brain associated with this disease.

It has been the habit of most writers to distinguish predisposing or general from exciting or special causes, and among the former heredity has been given the first place. There is no doubt but that hereditary neuropathic tendencies increase the susceptibility to nervous diseases in general, and certainly to epilepsy ; but this heredity alone does not suffice to make of an otherwise healthy individual an epileptic. For this usually an additional cause is needed, as, for instance, syphilis. If an individual with hereditary tendencies acquires syphilis, he is more likely to become epileptic—that is, to suffer from a genuine epilepsy which is neither preceded nor followed by any appreciable anatomical changes either in the brain or in its vessels—than a person infected with the same disease but burdened with no family history. Of course there are other cases, too, of cerebral syphilis with the characteristic arterial disease in which epileptic attacks occur entirely independently of any heredity (cf. Barbier, De l'épilepsie syphilitique et de son diagnose differentiel avec l'épilepsie vulgaire. Thèse de Paris, 1885). Important, therefore, as heredity may be, it is by itself not sufficient to constitute a cause of epilepsy. The manner, of course, in which the tendencies were acquired is also irrelevant, and the question whether the father or mother or both were given to alcoholism, and whether both or either of the two were intoxicated at the moment of generation of the child, has no significance. Notwithstanding the relative frequency with which

epilepsy occurs, the number of cases would be much larger if either of these factors could have any decided action upon the appearance of the disease.

Age and sex seem to have little influence in this connection. Although it is true that in the majority of cases the disease affects individuals in the first half of their lives, more especially between the ages of ten and twenty, it may begin much later, after the age of forty or fifty. Indeed, cases in which the first attack made its appearance between the sixtieth and the seventieth year have been recorded.

With regard to sex, it has been noted that during the period of puberty, between twelve and sixteen, more girls than boys become epileptic; if, however, the average of all cases is taken, the difference between the numbers in the two sexes is very slight, and in early childhood, from the fourth to the seventh year, it is *nil*, the cases being equally distributed between the two sexes.

Among the exciting causes there is in reality only one, the mode of action of which we are able to understand to any extent, and that is traumatism, and more especially injuries to the head. It may happen that a person, previously perfectly well, is taken with an epileptic fit after a fall or a blow upon the head; but here we must distinguish between injuries which cause a lesion of the brain cortex and those which do not; in the former case the lesions become the direct cause of the attacks, and we have not a genuine epilepsy, which we said was one based upon no appreciable anatomical changes, and which, therefore, only occurs if the brain be uninjured. We must carefully examine the scalp, and overlook no scar, however trivial, because any one may be the cause of the epilepsy. The probability becomes a certainty if the cicatrix is adherent to the bone beneath it and is painful to the touch, and if it is possible by firm pressure to bring about an attack; in this case, as we shall insist upon later, excision is indicated.

The attack which occurs in consequence of this pain is a reflex act, and this kind of epilepsy we call "reflex epilepsy." Such reflex attacks may also be caused by painful cicatrices on the peripheral nerves at any point of the body, or where there exist ulcerative processes—for instance, of the finger nails. In one of my patients it was possible every time to produce an attack by pressure upon the diseased matrix of the nail, the same thing occurring also when he accidentally struck it

against anything. The amputation of the terminal phalanx was followed by complete recovery after all other measures had proved fruitless. In a similar manner polypi of the ear (" ear epilepsy "), inflammatory processes in the ear, intestinal parasites, an incarcerated hernia—finally, also diseases of the sexual organs, in the male as well as in the female, may give rise to epileptic attacks. If, after a heavy indigestible meal at night, the patient is for the first time seized with an epileptic fit, as by no means uncommonly happens, the attack has also to be looked upon as reflex in nature.

It is well known and beyond question that psychical influences may become the direct cause of epileptic seizures, although how and why we do not understand. The most important of these is fright, which in numerous instances has been known to be the cause of the disease ; but how shall we explain the instances in which persons perfectly healthy up to that time suddenly become epileptic in consequence of the excitement of a shocking spectacle or of a dangerous situation— e. g., an assault by robbers or the menacing appearance of a fierce dog—and then remain so during their whole life in spite of all treatment ? What changes go on in the brain in such a short time, and of what nature can they be that they can persist for many years ? This question, with which we are unfortunately too frequently confronted, presents difficulties which appear insurmountable.

Closely related is the influence which the frequent sight of epileptic convulsions exerts upon healthy individuals, an influence which can hardly be accounted for by fright, but which is to be referred to a peculiar, most remarkable imitative impulse. In the royal prison of Breslau I have known thirteen of a large number of female inmates who were working together in a room become epileptic a short time after another prisoner, who had been suffering from epilepsy for years, had been brought into the same ward. The prognosis in this imitation epilepsy we would hasten to say is the most favorable, far better even than in " fright epilepsy."

Lastly, we would call attention to the fact that epilepsy may be caused by certain substances which are taken into the system, whether as food or for the sake of their agreeable effects, or, again, as medicines, when used in large quantities or for too long a time. Among the first we have alcohol, absinthe, and so forth, then fungi (mushrooms), certain spices (Cayenne

pepper and paprika); to the second belong all narcotics, more especially, as we have recently learned, cocaine. The "cocaine epilepsy" has been described by Heimann (Deutsch. med. Wochenschr., 1889, 12). Under certain circumstances other medicines—e. g., antipyrine—may act as poisons and lead to epileptic attacks (cf. Tuczek, Die Antipyrinepilepsie, Berl. klin. Wochenschr., 1889, 17). In view of the extensive use which this last-mentioned drug has obtained in a short time, and the popularity which it enjoys, and on account of which it is used in all possible kinds of diseases, however different from each other, this observation must be regarded as possessing great practical importance.

Pathogenesis.—The pathogenesis of epilepsy is totally obscure; although we know from the experiments of Kussmaul and Tenner that the source of the attacks must be sought for in the brain, the exact seat of the disease is not known. Since the work of Schröder van der Kolk special attention has been given to the medulla oblongata, and the discovery by Nothnagel of a "spasm centre" in the pons seemed greatly to support the "bulbar theory," but of late years this has more and more fallen into discredit, and it is now the brain cortex which is regarded as the starting point of the convulsions (Hitzig, Albertoni, Franck et Pitres, P. Rosenbach). For a long time the motor area was thought to be the only region concerned, but recently Unverricht, who, with his convincing experiments on animals, has proved himself the most successful defender of the cortical theory (after extirpation of an area in the cortex he found that he could not obtain spasms in the muscle groups corresponding to it) has shown that excitation of the posterior cortical regions is also capable of producing an attack, hence that these too possess epileptogenic properties, and that irritation of the same may by extension of the stimulus to the motor area give rise to general convulsions (Deutsch. Archiv f. klin. Med., 1888, 44, 1).

Binswanger agrees that in the lateral portions of the floor of the fourth ventricle there are points the stimulation of which gives rise to spasms, which, however, he considers to be of a reflex nature, and assumes the reflex centres to be situated in the dorsal half of the pons. According to his opinion, these represent, as it were, a collecting station for the centres of the spinal cord, and can not, in the physiological sense, be termed "spasm centres." He maintains that we never can

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succeed by electrical or mechanical stimulation of the pons in producing real epileptic attacks.

However probable an association of the cortex with the appearance of symptoms of motor irritation may seem, such an association is far from explaining the increased salivary secretion, the involuntary evacuation of the bladder, the increase in the frequency of the respirations, etc., and we must for the present leave the question open whether or not such phenomena depend upon some influence acting on certain centres in the brain and spinal cord, the situation and function of which we do not as yet know. The question raised by Ziehen as to the significance of the subcortical ganglia in the causation of an epileptic attack deserves to be looked into more closely; for the present only this seems certain, viz., that (in dogs) the clonic part of the convulsive movements produced by stimulation of the cortex is connected with the cortex itself, while the tonic and the running movements seem to be of subcortical origin (XIII. Wanderversammlung süddeutscher Neurologen. Archiv f. Psych., 1889, xx, 3, page 584).

Symptomatic Epilepsy.—As we said at the beginning of this chapter, there exists not only a "genuine," but also a so-called "symptomatic epilepsy," which does not constitute a clinical unit, but which comprises a number of different affections based upon various anatomical changes. The appearance of convulsions, during which consciousness can be retained, may be regarded as their chief common characteristic.

Here we must first call attention to that form of cortical lesion upon which we have touched already on page 184. It is clinically characterized by convulsions, which are sometimes confined to one arm or one leg, in general to a part only of the body, and has, therefore, received the name, very inappropriate, indeed, of partial epilepsy, or, after its anatomical seat, cortical epilepsy, or, again, after the author who first described it, Jacksonian epilepsy. The seizures are distinguished from the classical epileptic fits by the fact that consciousness is maintained, a circumstance which, of course, makes the two clinical pictures quite different. An aura may occur, it is true, in this form also, so that the patient knows when the convulsions are going to appear, either from a slight twitching in the fingers or toes or from feelings of formication, etc., signs which appear only in the affected extremity; but all other symp-

toms, the cry, the biting of the tongue, the fall, and so forth, are absent. The patient sees and watches the twitching of his extremity, and may at the same time complain of considerable pain. He himself holds the limb or has it held by others, so as to avoid as much as possible any injury to it. After the attack he feels weak and exhausted, but only on account of the increased muscular work. Headache and all the various "post-epileptic" phenomena are wanting, or if they occasionally do occur, have no connection with the attack, and have, therefore, no claim to be reckoned under this head.

The degree, duration, and frequency of the attacks are extremely variable; sometimes they consist only of a more or less marked twitching, which appears in the affected extremity; sometimes there are shaking movements which can attain to so high a degree that the bed, on which the patient lies, is also shaken under him, and he anxiously calls for some one to come and hold him still. If considerable pain come on during the movements, it usually continues after the attacks and is a great source of trouble to the patient in addition to the motor weakness in the affected extremity. The duration also of the attacks varies: I have seen some which were over in from fifteen seconds to a minute; on the other hand, cases have come under my notice in which a quarter, a half, or even an hour was taken up by the involuntary movements. If these attacks recur often—twice, three times, or six times a day—the condition of the patient is most distressing, and the partial epilepsy can do more to ruin his life than the classical form would ever have done. In other instances the attacks are so few and far between that the patient almost forgets one before he has another, as many months may have passed. The whole course of the disease is eminently chronic; the patient may suffer from the affection for tens of years without presenting any other symptoms. Death is finally caused by an extension of the brain lesion or by an intercurrent disease. Pitres has called attention to the fact that in this form also "equivalents" may occur (Revue de méd., 1888, viii), and has pointed out that they may be sensory or psychical. The former have been called by Charcot *l'épilepsie partielle sensitive* (Leçons du Mardi à la Salpêtrière, 1889, pages 20 and 368); in the latter we have to deal with hallucinations of sight, hearing, and smell, which occur paroxysmally without the appearance of any decided symptoms of motor irritation.

A peculiar kind of epilepsy, which is said only to occur in heart disease, has been described by Lemoine (De l'épilepsie d'origine cardiaque, Revue de méd., vii, May 5, 1887), yet since the connection is not absolutely proved, and since, moreover, the attacks themselves present no peculiarities of their own, we shall limit ourselves to saying that they disappeared under the administration of digitalis.

Lately von Jaksch (Zeitschr. f. klin. Med., 1885, x, 4) has shown that epileptic attacks may be produced by auto-intoxication, not only by urea, but in a similar way also by acetone. In cases of "epilepsia acetonica" large amounts of acetone were found in the urine, which besides contained neither sugar nor albumen. The physiological connection between the occurrence of acetone in large quantities in the urine and epileptiform attacks is not as yet fully established, nor do we know how poisons—for instance, lead—introduced into the organism from outside are able to produce such attacks; as a matter of fact, however, lead workers suffer so frequently from epilepsy that we are justified in assuming the existence of a definite "epilepsia saturnina" (Hirt, Krankheiten der Arbeiter, iii, 49).

Briefly, epileptic attacks may occur as a symptom also in meningitis, dementia paralytica, during delirium tremens, in sclerotic processes, more especially frequently in sclerosis of the cornu Ammonis. They may be associated with tumors, hydrocephalus, or abscess of the brain, in which cases they are the result of the increased intracranial pressure, as we have pointed out above. From what has been said in this and in previous chapters it will be understood that these and the so-called epileptiform attacks above mentioned have in all probability nothing to do with the genuine classical epilepsy.

Diagnosis.—We can well understand, then, how cautious we must be in our diagnosis. Only after repeated and careful examinations, after which we are able to exclude organic brain diseases, abnormities in metabolism, in consequence of which abnormal or poisonous substances occur in the urine (urea, sugar, acetone), are we justified in making the diagnosis of genuine epilepsy. The skin and tendon reflexes should always be carefully examined. Sometimes, from the absence of the abdominal or cremasteric reflex, or from a unilateral increase of the patellar reflex, we may be able to diagnosticate an

organic brain trouble when we otherwise, without any inquiry into the condition of the reflexes, might have regarded the case as one of genuine epilepsy.

The conditions for which the epileptic attack itself or the coma following it could be mistaken have been discussed in our account of cerebral hæmorrhage. We only need add here that cortical epilepsy may be simulated by uræmic attacks if the latter are confined to one side. Chauffard (De l'urémie convulsive à forme de l'épilepsie Jacksonienne, Arch. génér. de méd., July, 1887) has called attention to this. Hysterical attacks may bear a striking resemblance to those of Jacksonian epilepsy; in such cases the recognition of other hysterical symptoms should prevent any diagnostic errors. Mendel has repeatedly observed that progressive paralysis of the insane may commence with attacks of this kind. The foci which were found at the autopsy were in every case situated in the right psycho-motor region, and the paralytic speech disturbance did not occur until late in the disease, whereas usually it is one of the earliest symptoms. The possibility, finally, of confounding epilepsy with eclampsia we shall discuss shortly.

Quite frequently we meet with malingerers who, for some reason or other, feign epilepsy. The situations in which the simulation of this disease would be likely to be advantageous to the deceiver are quite numerous, and it would be impossible to enter into the consideration of them here; we will only mention that epileptics are dispensed from military service, good grounds enough for many to sham this disease. The more cunning the malingerer the more perfect will be the attack, not excluding the foaming of the mouth (made by soap) and the (not very deep) wounds of the tongue; there will be convulsions and the (feigned) loss of consciousness is possibly prolonged more than is necessary; if the rogue has courage enough he will not betray himself either by a reflex motion of defense, or even by the slightest twitching, if hot sealing wax is, as a test, dropped on his chest. Under certain circumstances it may be extremely difficult to unmask the fraud; it might, indeed, be impossible, did we not know one reflex over which the will has no power, namely, the pupillary reaction to light, which in the epileptic is lost, in the malingerer naturally is retained. In doubtful cases, therefore, this reflex has to be carefully observed, and the further measures should depend upon its condition.

Treatment.—The treatment of epilepsy confirms the old experience that the greater the number of remedies which become known and are recommended for a disease, the more difficult and uncertain becomes the cure. In the course of centuries such an array of medicaments have been recommended to combat this disease that there is hardly a drug in the shops which has not at one time or another been regarded and praised as an infallible "specific." Unfortunately, all these claims have been proved to be false. We are to-day as little in a position to cure epilepsy as we were one or five centuries ago. Only by the discovery of some causes which may produce epilepsy, the removal of which lies in our power, has any progress been made in the treatment of the disease. This more particularly applies to the above-mentioned reflex epilepsies. Here a cure is possible—nay, we may say even certain—if we are able to remove the cause. To discover it must be the physician's aim. Sometimes it consists of a bone splinter which is left after an injury to irritate the cortex, in which case a cure will invariably be effected by the operation of trephining for the removal of the splinter. In other instances painful cicatrices have to be excised or affections of the intestinal tract or the sexual apparatus treated. In children the natural openings of the body have to be examined for the possible presence of a foreign body, the removal of which would then be absolutely necessary.

Such are the favorable cases in which it is in the power of the physician to bring about a cure. Unfortunately, their number is not great. In the largest majority of instances we are not able to find any cause, the removal of which would remove also the disease; but to-day, as centuries ago, we are reduced to the sad necessity of trying all sorts of remedies, trusting to good luck that at some time we may hit upon one which is truly efficacious.

Among the internal medicines the so-called specifics possess an interest purely historical; from artemisia (in hot beer, 10 to 20 grm. at a dose—grs. 150 to 300) and valeriana down to squilla, gratiola, sedum, cardamine, and hellebore, many herbs have been lauded as effectual. Asafœtida, castoreum, and camphor have been recommended, although no better results have been obtained from them than from silver nitrate, ammonio-sulphate of copper, and arsenic. A great sensation was created by Meglin's pills, which, in addition to zincum album contained hyos-

cyamus. Some have sworn by oxide of zinc, and Herpin, for instance, claimed that out of forty-two cases he cured twenty-eight with it. To unprejudiced judges who continued their observations for a sufficiently long time these "cures" could not hold their ground. They proved to be deceptive, and we were as helpless as before. Recourse was also had to narcotics, and much was hoped from the action first of opium and later of ether and chloroform. It is true that here and there an attack has been cut short by inhalations of the latter, but that is all. It is not to be wondered at that under such circumstances secret remedies were used to a tremendous extent; and to what a pitch the humbug and impudence were carried may be seen from the composition of some such remedies, for instance, the epilepsy powder of the Institute for Deaconesses in Dresden, which consisted of charred bone of magpies which had to be shot at some time during the twelve nights following Christmas, and again from the epilepsy powder of Wepler, which was nothing but charred and pulverized hemp thread (cf. Richter, Das Geheimmittelunwesen, Leipzig, 1872, pages 15–16).

A new era in the treatment of epilepsy—that is, of the attacks—was initiated when Locock in 1853 recommended bromide of potassium, which obtained a wide acceptation through the efforts of Legrand du Saulle. Its power of diminishing the reflex irritability and of lowering the blood pressure in the brain has placed it first among the antispasmodics, and to-day it has to be regarded as the best and most important medicine in the treatment of epilepsy. In order not to be disappointed, however, in our expectations, it is necessary that we should be familiar with the proper regulation of the dose and with certain unpleasant effects which are apt to arise in the course of the treatment. The small and moderate doses of 0.5 to 4 grm. a day (grs. viij to ℥j) formerly used are generally ineffectual. It is necessary to employ much larger amounts, which are best given in one dose. It is, moreover, better to combine the three bromides, viz., the bromides of potassium, sodium, and ammonium, in equal parts than to give bromide of potassium alone. The minimum daily dose for adults in cases of pronounced epilepsy is eight grammes (℥ij), and we should follow Mendel, who advises that it should be taken in valerian tea immediately before going to bed (potassium bromide, ammonium bromide, āā 2.5 (grs. xxxviij); sodii bromidi, 3.0 (grs.

xlv). For children and young people up to sixteen years of age the daily dose should be half a gramme (grs. vij) for every year. If the two drachms are not sufficient—that is, if an attack still occurs now and then—the dose may be increased to ten or twelve grammes (℥ijss. to ℥iij), and this continued until four or five hundred grammes or from six to nine ounces are taken.

In this way I have treated hundreds of epileptics in private as well as in dispensary and hospital practice and have let slip no opportunity for observing the action of the bromides. This action is by no means the same in all cases. There are people in whom an idiosyncrasy against the medicine rapidly develops, so that it is impossible for them to take it any more. It nauseates them and may cause vomiting, and after repeated unsuccessful trials to resume the treatment we have to discontinue it entirely. In other instances the desired effect on the attacks may show itself; but after a few weeks the patient begins to complain of general bodily and mental feebleness, a constant desire to sleep, some loss of memory, and other symptoms, so that the dose has to be diminished. At the same time, sometimes without these symptoms, an eruption on the skin appears, more especially an extensive, obstinate acne distributed over face, trunk, and extremities, which is most distressing, especially to young female patients. I have seen this eruption particularly after the prolonged use of small doses, and have also seen it disappear comparatively rapidly under the use of mild laxatives and the administration of arsenic in the form of Fowler's solution. Finally, cases come under our notice in which bromide, no matter in what form or dose it be given, is entirely without effect. The attacks occur just as they did previous to the administration of it. Here we have, of course, again to suspend the treatment, more especially if symptoms of intoxication appear in addition to the continuance of the fits. If we wish to express the effects of bromide in epilepsy by percentages, we could say that in about ninety per cent of all cases the paroxysms diminish in number and violence, that in about as many signs of bromism appear which render necessary a diminution of the dose or a gradual suspension of the medicine. In from two to three per cent of all cases bromide is borne so badly that it has very early to be discontinued.

If it is established beyond doubt that the bromides exert a favorable action, we must insist upon their prolonged use for

months and years. To add some variety to the treatment they may be combined with belladonna and pills may be ordered which contain both. If every evening two centigrammes (gr. $\frac{1}{3}$) of belladonna and two grammes (grs. xxx) of bromide are given, about the same results are obtained as with eight grammes (ℨ ij) of bromide alone. [℞ Extr. bellad., 0.5 (grs. vijss.); pot. brom., sodii brom., ammonii brom., āā 15 (℥ ss. circ.); pulv. et succ. liq., āā q. s. ut. f. pil. No. 50. Signa : One to two pills in the evening.] When the action of the bromide gradually becomes lessened owing to the establishment of a tolerance, the administration of belladonna is also indicated, and it may then be given in the form of Trousseau's pills. (℞ Extr. bell., Fol. bell., āā 1.0 (grs. xv), succ. q. s. ut. f. pil. No. 100. Signa : One to two, later three to four, or even six pills, in the evening.)

Stevenson, in the Medical Press and Circular, 1888, 24, 10, has recommended bromide of camphor very warmly (camph. brom., 0.6—grs. ix—at a dose several times a day). Our experience with this drug does not agree with his. We have never obtained any lasting effect with it.

Compared with bromide and belladonna, which, according to our opinion, are the only reliable drugs to be used in the internal treatment of epilepsy, the medicaments which have been recommended of late years—curare, considered to be effectual by Bourneville, antipyrine by Beaumetz, tinct. simulo (the fruit of *Capparis coriacea*), which has been used by White—do not play any important *rôle*, and only deserve a trial in desperate cases. On the other hand, amylene hydrate, recommended by Wildermuth (cf. lit.), must be given a trial in cases of distressing bromism or if the attacks increase to an alarming extent. The watery solution of Kahlbaum's preparation, in the proportion of one to ten, is the best to use in doses of from twenty to forty grammes (ℨ v to ℨ x)—i. e., two to four grammes (ℨ ss. to ℨ j) of the drug itself. It may be given in wine or water or in a glass of beer, well shaken up, and from five to eight grammes of the drug (ℨ jss. to ℨ ij) may thus be used daily.

Surgical interference has also been resorted to, at first with the view of influencing or diminishing the amount of blood in the brain. Several times the carotids have been ligated, and two cases thus treated were reported as completely cured (Hasse, Krankheiten des Nervensystems, page 297). Owing to

the great difficulties of the operation and the great responsibility which the physician takes upon himself, this measure will only in exceptional cases be made use of. With bleeding, strong revulsives to the skin, such as Autenrieth's ointment to the shaved head, moxas, setons, blisters, and purgatives, possibly the same results can be obtained.

Quite recently both vertebral arteries have been ligated (von Baracz, cf. lit.). In my clinic the ligation of one vertebral, the right, was performed several months ago by Janicke without any noticeable effect upon the frequency or the severity of the attacks ; hence the patient was not willing to submit to the ligation of the other. The operative treatment of traumatic epilepsy aims at the removal of bone splinters which press upon and injure the brain cortex; but the operation should only be performed, as von Bergmann holds, if the convulsions constantly occur in the same groups of muscles and extend in a characteristic manner, or if transient hemipareses occur. At the operation the affected area of the cortex has to be carefully excised. If the attack begins like a flash without an aura and is associated with opisthotonus, etc., operative measures are contraindicated.

Marshall Hall's advice to perform tracheotomy, on the ground that the spasm of the glottis is productive of the asphyxia and the clonic spasm, is purely and entirely of historical interest. The operation has been performed several times without, of course, the least benefit to the patient. The same may be said of the cauterization of the glottis with nitrate of silver, suggested by Brown-Séquard, which has been justly condemned in such cases.

In connection with the surgical treatment we should mention the application of strips of cantharidal plaster around the forearm or lower leg in which the motor or sensory aura occurs. Only when the aura constantly appears in the same member can any success be expected from this measure, which has been recommended by Buzzard. The plasters must remain on for a considerable time. Following the advice of Buzzard, I have ordered the application of these plasters in some cases, without, however, having been able to see any good results. In one instance of partial epilepsy a transfer was produced by the application of the plaster (Hirt, Neurol. Centralblatt, 1884, 1).

Finally, we can hardly be surprised that attempts have

been made to combat epilepsy by electrical treatment. Unfortunately, the results with this have been even less encouraging than those from internal medication. Neither the attacks themselves nor the so-called "epileptic change in the brain," the nature of which, as we have above stated, is still obscure, have been influenced by it in any way. The constant current was employed and the sympathetic galvanized by passing the current from one mastoid process to the other, and attempts were made to influence the cerebral hemispheres, and more especially the motor regions, according to Erb's method (Erb, Handbuch der Electrotherapie, page 581). In other cases the current was passed through the lobes of the thyroid gland, as Sighicelli (Rive. sperim. di freniatr., 1888, vol. xiii, 3) has quite recently done, but in none of them could any lasting success be remarked. No better results have been obtained with the faradic current in all its different modes of application.

Although with all our treatment we are practically powerless against the disease, it would be very wrong to assume that to the epileptic the physician can be of no use and can not improve his condition in any way. On the contrary, there is hardly another class of patients affected with nervous diseases who require so much a physician's advice, and hardly another class who have to be so carefully watched by him. Above all, attention has to be paid to the general condition. The bowels must be kept regular and the skin and muscles stimulated to their proper activity by appropriate cold-water treatment and home gymnastics. The patient should constantly be warned against every kind of excess. Too large a supper, a few glasses of wine or beer taken too quickly, any indigestible food, excesses *in Venere*—all these may give rise to an attack, the consequences of which are incalculable. To guard against these, therefore—in other words, to employ prophylactic measures— is the chief task of the physician who is taking charge of an epileptic. Besides this, the bromides, or, if these are not suitable, the next best treatment, should be begun. Finally, care must be taken that the patient does not hurt himself during the fit, and against this he should be protected as well as possible. All tight clothing must be removed and all ordinary emergencies provided for. A regular treatment of the attack itself we do not possess, and all attempts to cut it short should be avoided. Even inhalations of amyl nitrite, which O. Berger suggests, chloroform, and similar remedies are only allowable

if administered with the greatest caution, and it would be better still to discard them entirely.

Note.—Eclampsia is one of those terms which up to the present do not convey to our minds any clearly defined clinical or pathological picture. It is a term under which are comprehended the most heterogeneous conditions which have not the least connection with each other. If a woman during pregnancy or during parturition without any appreciable cause loses consciousness and falls into convulsions, which may recur several times, and which frequently lead to a fatal issue, we speak of eclampsia gravidarum or parturientium. If children, as not uncommonly occurs, have paroxysms, consisting of distortions of the face, trismus-like clinching of the teeth, general spasms, and more or less marked disturbances of consciousness, we designate the affection as eclampsia infantum, and use the same term if at the onset or in the course of acute diseases or certain intoxications (more particularly lead poisoning) attacks occur characterized by (bilateral, more rarely unilateral) convulsions and loss of consciousness, which, therefore, differ clinically either not at all or only slightly from the genuine epileptic seizures. The nature of the attacks is as obscure as their ætiology. Whether in eclampsia parturientium the diminished excretion of urea has to be held responsible for the convulsions, and they thus are to be regarded as uræmic, whether in the convulsions of children reflex action plays the chief *rôle*, or whether we have to deal with autointoxication in which diacetic acid occurs in the urine, is at least doubtful. Every one admits that, in the second form, dentition, digestive disturbances, or intestinal parasites play a certain part, yet there are certainly other factors which deserve consideration in this connection, for instance, heredity, a general neuropathic diathesis, the health of the parents, and the possible existence of rickets. The convulsions of children (eclampsia acuta infantilis) are extremely common. Clinically, all cases of this kind are very much alike, whereas ætiologically different cases differ greatly. In a given case we should, first of all, try to determine whether we have to deal with anatomical lesions (of the cortex, etc.), or whether these can be excluded; and only by the most careful examination can we avoid errors and are we able to make a correct diagnosis. Cortical diseases (cerebral infantile paralysis), epilepsy, spinal paralysis of children, the

initial stage of acute diseases, etc., must be taken into consideration.

The prognosis is always doubtful, both in adults and in children, and the danger is usually greater in pregnant and parturient women than in children. Death not rarely occurs during the convulsions, as we have said above, and we may assume that out of a hundred cases of this kind there are thirty, forty, often fifty who die, and the danger increases with the duration of the labor and the long continuance of the pains. In children a fatal issue is often brought about by a spasm of the glottis, rarely by exhaustion. Recovery frequently is incomplete, and there may be left some psychical disturbances, amaurosis or disturbances of speech, etc.

About the treatment of eclampsia the opinions are even at the present time very much divided. In pregnant or parturient women cold affusions in a warm bath, as recommended by Scanzoni, also the application of large cantharidal plasters to the neck, ought to be resorted to as soon as possible; from the nervines we can expect nothing. Mild laxatives, cautious venesection, regulation of the functions of diuresis and diaphoresis are in most cases indicated. Often we have no time to think of such measures; in urgent cases Veit (cf. lit.) has recommended large doses of morphine, beginning with three centigrammes (circ. gr. ss.) and increasing the dose to two or three decigrammes (grs. iij–grs. ivss.) a day. The eclampsia of children is, according to some, among them Henoch, best treated by inhalations of chloroform, which will soon stop the convulsions. One ought, they think, to first cut short the convulsions, and then proceed to find out their cause. Sometimes this advice is good, viz., in cases in which there exists no cerebral lesion. If one does exist, or if there are grounds for suspecting it, the inhalation will prove to be of no use, and may rather have a bad effect. It will therefore be necessary to attempt to settle this question by as short an examination as possible. If we are unable to make up our minds, a tepid bath and cold affusions, vinegar enemata, or evaporating lotions, etc., to the skin can do no harm. For the beginning this suffices; afterward it may be advisable to prescribe ice to the head in congestive conditions, possibly even leeches to the head, and in cases where collapse seems imminent, vinegar enemata, strong wine, or injections of ether. The nervines may as well be discarded in the treatment of the convulsions, as

they do no good in this stage; they may, however, be used later when the immediate danger has passed. Wrapping the children in warm moist sheets (after the method of Priessnitz), while ice is kept to the head, I have known repeatedly to be effectual. On the whole, even these measures are not reliable, and the part which a physician plays in the presence of eclampsia of children is by no means enviable.

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THE reason why we have not treated of the disease, we are about to describe, in immediate connection with hysteria, but have placed it after the chapter on epilepsy, is because the "attacks" of hystero-epilepsy appear to the observer as a result, or perhaps we had better say as a sort of mixture, of hysteria and epilepsy. It would, however, be a mistake to infer from this that the affection has any close physiological or pathological connection with epilepsy. It is more likely that we ought to regard it as a higher, or indeed the highest, grade of hysteria (cf. page 524).

The "major attacks" have been studied exclusively by Charcot in the Salpêtrière. To him alone and some of his pupils, more particularly P. Richer, we owe our knowledge of their nature and characteristics, and of the rules and definite laws which they appear to follow. Almost every, nay, we can well say every publication on hytero-epilepsy that did not emanate from the Salpêtrière was, at any rate, based upon Charcot's observations and communications, and hardly anything new has come from any other source.

The attacks can usually be divided into four distinct periods, though one or other of them may so predominate, as regards its duration and intensity, that the rest are somewhat obscured. The first period embraces the epileptiform attacks: the body is suddenly shaken, respiration stops, the patient lets fall anything she happens to be holding in her hands, and is thrown to the ground. She is now seized with general convulsions or there develop rapidly extensive contractures affecting almost all the voluntary muscles. In the second stage, which immediately follows this, the patient is bounced up and down in bed, she assumes marvelous positions, stands on her

head, curves the body in the form of an arch (*arc de cercle*), and howls and roars at the same time like a wild beast. This is the period of major movements, "clownism." It is followed immediately by certain hallucinations, under the influence of which the patients assume postures indicative of the most varied passions, the "*attitudes passionelles*" of the French. The face takes on, according to the particular hallucination, an expression of anger, rage, devotion, love, voluptuousness, curiosity, pain, etc., which would give us the impression that the patient is passing in her mind through a period of her life the details of which are unusually vivid in her memory. The postures and expressions may change, although sometimes they remain the same throughout this stage. That of the "crucified" has obtained a certain degree of celebrity, because it seems to be particularly frequent. Finally, the fourth stage is marked by a delirium, in which hallucinations recur with the greatest persistency, some patients imagining they see animals, others terror-inspiring objects of different kinds, and so forth. Automatic movements are not rare; sometimes anæsthesias or at least analgesias are noted. This delirium resembles in many respects an alcoholic intoxication. The duration and frequency of the attacks vary greatly. Some only last from one to five minutes, and recur ten, twenty, or even one hundred times a day (*état de mal*). It is a characteristic feature, and one very valuable in the differential diagnosis, that firm pressure upon the ovaries invariably suffices to cut short an attack.

If this pressure is exerted constantly, as can be done by means of belts provided with pads, the attacks may be kept off for quite a considerable time. At the celebrated ball which every year at *mi-carême* is given to the hysterical and hystero-epileptic patients of the Salpêtrière, in which, of course, only females take part, each dancer wears her belt. If this, owing to the movements in dancing, slips from its proper place, so that the pressure is taken off the ovaries even for a moment, a major attack comes on, and the patient, twisting and turning herself and presenting the most incredible distortions, is removed from the ball-room, without causing the least interruption in the dancing.

The outlook for complete recovery in major hysteria is not favorable. All attempts to cure the patients remain in many instances fruitless, as we may observe in the Salpêtrière, where

some patients, in spite of the best care and the most excellent treatment, remain for years without presenting any marked or lasting improvement either with regard to the violence or the frequency of the attacks.

Sometimes, especially if the patients come early enough under the care of the physician, immediate removal from their homes into an institution does much good. The attacks become rarer and cease entirely after a few months. The treatment in these institutions consists in the "feeding system," which we have mentioned on page 525, as well as the ice-cold douches, to which we have also alluded above.

The brilliant success of Charcot in the treatment of hystero-epilepsy is due to these three factors: (1) The removal from home, (2) the cold douches, and (3) the feeding system. With the use of static electricity and the magnet, the results have been shown to be much less favorable, and we may consider that this procedure, as far as the treatment of the major attacks goes, has in the main been discarded (cf. the references to metallotherapy).

Reliable and correct as are the descriptions given by the Charcot school of the major attacks, which we may incidentally remark are very rarely seen in Germany, accurately as we can follow up the different phases or periods of the attack in many such patients, we still must be very careful in accepting the accounts of the influence of hypnotism upon hystero-epileptics and the conditions produced thereby.

In the Salpêtrière the patients were hypnotized by means of fixation of the eyes, by the action of a bright light, or the sound of an instrument called a tam-tam, or by similar means; and, as every one must know who has been present at Charcot's experiments, certain individuals were hypnotized in a very few seconds. According to Richer, who, as we said, has made the most careful studies of this subject in conjunction with Charcot, which appeared in various numbers of the Arch. de neurol. from 1881 to 1883, there may be distinguished four different stages: (1) The cataleptic, (2) the stage of suggestion, (3) the stage of lethargy, (4) the stage of somnambulism.

In catalepsy, whether artificially produced or whether occurring spontaneously, as it does in hysteria in very exceptional cases, the members of the body remain in any position into which they have been put. Thus, if we passively bend the arm at the elbow and raise it up, it remains fixed in this position.

Flexion or extension in any joint can be produced without the slightest resistance on the part of the patient—"*flexibilitas cerea*"; even the most unusual, uncomfortable, and strangest attitudes are retained without any difficulty. How this most remarkable regulation of the necessary innervation is brought about we do not know as yet, neither have we the slightest grounds whereupon to base any theory by which we could seek to explain this condition, which is not infrequently also associated with disturbances of consciousness.

The state of hallucinations excited by slight stimulation of the special senses (in reality by suggestion), and designated as automatism, is characterized by total analgesia. The eyes remain open, and it is a remarkable fact that positions which are given to the body evoke the corresponding expressions of the face, and, *vice versâ*, the body assumes the corresponding position if on the face, by faradization of the muscles, a certain expression—e. g., of sadness, hilarity, spite, voluptuousness, or fear—is produced. By firmly shutting the eyes of the patient it is claimed that the second stage may be converted into the third, the automatic into the stage of lethargy. In this latter the excitability of all the nerves and muscles is greatly increased, so that, for instance, slight pressure upon the stem of the facial suffices to bring about contractions in all the muscles supplied by that nerve. The contraction lasts much longer than the stimulation, and therefore takes on a tetanic character. At the same time the patient is apparently completely unconscious, and there is total anæsthesia. Now it is impossible to create hallucinations. The tendon reflexes are greatly exaggerated. If we now stroke the patient lightly over the top of the head, the hyperexcitability vanishes and a new stage comes on, that of the hysterical somnambulism. In this condition the patient is susceptible to external influences, inasmuch as the organs of special sense are performing their functions to a certain degree. He answers questions (with closed eyes and somewhat automatically), carries out instructions, and so forth. By local stimulation of the skin—for instance, by vigorous rubbing—we are able to produce contractures. By energetic pressure upon the eyes the patient can again be transferred from the somnambulistic to the lethargic condition. The occurrence of hallucinations and illusions is not constant.

For a long time the theory that this condition was peculiar to hysterical patients when hypnotized, just as the other condi-

tions were peculiar to them when awake, was not doubted, and the so-called major hypnotism, as the hypnosis of the hystero-epileptics was called, created everywhere great astonishment and admiration, especially in those who could actually observe it in Charcot's clinic at the Salpêtrière. It is only recently that doubts have been raised about the correctness of these claims of Charcot. Many are inclined to believe that the above-described four stages, which the hypnosis of hysteria presents, can be produced in any hypnotized individual and not only in those who are hysterical, and that therefore the " major hypnotism " is no neurosis at all and has no characteristics of its own. Whether the members of the Salpêtrière school will be able to defend their former assertions, and what arguments they can put forth, and whether they will be able to continue to uphold the existence of different stages of hysterical hypnosis after all possible sources of error have been excluded, we can not tell. How they will be able to demonstrate the neuro-muscular hyperexcitability as physiological and not perhaps as produced voluntarily, as many are inclined to think now, has to be left to the future to decide, and more especially to the absolutely necessary repetition of the experiments. Here it is our part only to show on what grounds Charcot's doctrines have been attacked, what proofs have been brought forward to show his doctrine to be untenable, and to state clearly the stand-point which is now generally held as regards the origin and the phenomena of hypnotism.

This is not the place to enter into a consideration of the mysticisms and the charlatanisms of a man who a hundred years ago propounded the doctrine of the so-called magnetic fluid, which, emanating from the magnetizer, and being capable of spreading itself in space, could receive all impulses of motion and impart them, but as a matter of historical interest and justice we are compelled to state that it was Franz Mesmer, born in 1733, who gave the first impulse to a movement which, founded on his arrogant and wild teachings, has passed through manifold phases, and to-day still exists, now that it has been found possible to sift the chaff from the wheat. Magnetism to-day has succumbed to the same fate as alchemy, and has been discarded, but both bore good fruit; the one opened the door to chemistry, the other to hypnotic suggestion (Bernheim).

The fact that there is no such thing as a magnetic fluid, that hypnosis and the phenomena occurring during it are entirely

subjective in nature, and are to be attributed to external influ- ences upon the nervous system, was discovered by James Braid, of Manchester, in 1841, and we are justified in opposing " braidism " to " mesmerism " just as we oppose truth to false- hood. Braid concentrated the attention of those he wanted to put to sleep by making them keep their eyes fixed upon a bright object; he assumed that the fatigue of the levator pal- pebræ superioris, which was simultaneously produced, was the cause of a sleep during which the imagination was so active that spontaneous mental pictures, as well as impressions imparted by others, ("suggestions") obtained the power of actual percep- tions. If such impressions are imparted frequently, according to his observation, a certain habit is established, so that it be- comes, *cæteris paribus*, easier and easier to put the patient to sleep. Braid was also acquainted with the fact that corre- sponding sensations and passions can be produced in hypno- tized persons by putting their facial muscles and their extremi- ties in appropriate positions, although he made no attempt to explain these phenomena physiologically. This has only been done quite recently, and even then the study was evoked only by a purely external stimulus, viz., the exhibitions which a Danish magnetizer named Hansen gave in the German cities. The impression which these made upon the public at large was of such an exciting and uncanny nature, and the whole thing was so puzzling to men of learning, that physiologists and neu- ropathologists were impelled to approach the subject to see whether the apparently supernatural and inexplicable could not be traced to natural physiological laws. One of the most prominent physiologists, Heidenhain, put forward the theory that, by weak but steady stimulation of the nerves of special sense, the cells of the cerebral cortex were induced to discon- tinue for a time their activity, thus causing the subcortical reflex centres to fall into a state of irritation, partly because, owing to this inactivity, the reflex inhibitory influence of the cortex was suspended, and partly because every impulse reach- ing the brain was propagated to a limited area which neces- sarily led to stronger excitation of the part of the excitomo- tor apparatus belonging to it. With this ingenious hypoth- esis, which many others—Weinhold, of Chemnitz, Grützner, Rumpf, Berger, and Schneider among them—have accepted, we had to be satisfied, and for the physiologists the interest in the matter was thus exhausted, and the subject was abandoned.

In pathology and general practical medicine, including, as we shall see, surgery and obstetrics, the matter obtained a new and increased significance when, quite recently, the observations, which twenty years ago had been made by an investigator in Nancy, Liébeault, were again taken up. Liébeault had published a work in 1866 with the title Du sommeil et des états analogues considérées surtout au point de vue de l'action du moral sur le physique, in which he expanded upon the observations of Braid; he showed that it only needed a concentration of the thoughts on a single idea, viz., the idea of going to sleep, to make the body immobile, and to produce a certain kind of sleep which, however, differs from the physiological form (suggestion theory of hypnotism). The same author was the first to show that neither an optical, an auditory, nor a tactile stimulus was necessary to bring about hypnosis, but that the impressions from outside, the suggestions that the sleep must and will occur, are perfectly sufficient; the hypnotized sleeper—whose ideation, in contradistinction to that of the ordinary sleeper, remains in contact with that of the hypnotizer —can be influenced by the latter in his ideas and actions. The fundamental observations of Liébeault remained unappreciated for twenty years; the work was not read, hypnotism remained a curiosity, and it seemed inadvisable for a scientific physician to occupy himself with it, unless he was willing to gain for himself the reputation of a charlatan or of a man whose actions were suspicious or even dangerous. The credit of bringing to light the work of Liébeault, we might almost say of having discovered Liébeault, belongs to Bernheim, of Nancy, whose merit was still more augmented by his own contributions to the subject. He published his first article on hypnotism in 1884, and with his book, De la suggestion et de ses applications à la thérapeutique, he has, to use a popular but expressive phrase, "hit the nail on the head." He and the Nancy school have to be regarded as the founders of the successful attempt to make a systematic use of hypnotism for therapeutic purposes, and when once the treatment by suggestion becomes generally accepted, and becomes an integral part of our therapeutic armamentarium, although at present there seems little prospect of this, Bernheim will be mentioned as its scientific originator. The adverse attitude which certain well-known scholars and investigators have thought proper to adopt toward the treatment by suggestion, we can well under-

stand, if we remember that for the practice of hypnotism much time and patience is needed which is not at the disposal of everybody, but this opposition can do no permanent harm to the method if it is really good, since men like Krafft-Ebing and Forel, with their names as scientific investigators, and emi. nent practitioners like Wetterstrand, in Stockholm, and van Renterghem, of Amsterdam, and others, with their reputation as physicians, have ranged themselves on the other side. The attempt also, which has been made from various quarters, to be done with the whole matter and stifle it by pronouncing it, in a lordly manner, a humbug, must be considered to have utterly failed, and the second argument of the opponents, now put most prominently forward, that the use of hypnotism in therapeutics is dangerous, and should be prohibited by law, will be groundless and without effect if hypnotism is never used carelessly and promiscuously, and if, what is very important, it remains in the hands of those whose calling it is to make use of therapeutic measures—that is, in those of the physicians. Nobody, certainly, who is acquainted with hypnotism will deny that pathological conditions may be produced by it and that it may be dangerous, but is this a ground upon which to simply discard it without a further hearing? Have we given up chloroform narcosis because it has now and then proved dangerous in the hands of the inexperienced and careless operator, or have we given up the use of morphine on account of its poisonous action when used too freely and for too long a period of time? As in all other measures, we must recognize here indications and contraindications, and this can be done in the majority of cases without difficulty; and as everything in this world, especially in the practice of medicine, even the smallest operation—that of vaccination, for example—has to be learned, so the art of hypnotizing has to be acquired, and one can expect to comprehend the subject and to have success with the practice of the treatment by suggestion only after careful and painstaking study.

The technique of hypnotizing is somewhat as follows: The patient, having been placed in a comfortable armchair, is asked to think of nothing else than of going to sleep. We "suggest" to him that he is beginning to feel tired, that he is no longer able to completely open his eyes, which are already beginning to close, etc. At the same time he is asked to look steadily at two fingers of the hypnotizer, which at first are held directly

in front of his eyes, but are gradually lowered, by which procedure the closing of the eyes, which we desire, is easily accomplished. Now either a difficulty in moving the arms or legs is suggested, a loss of sensation in certain parts of the skin, or some similar idea. The tone of voice in which all this is said should not be loud, but monotonous. The same suggestions must again and again be repeated, and care must be taken that disturbing noises, the slamming of doors or the striking of clocks, and such like, be not heard, so that the mind of the patient may as much as possible be concentrated upon the hypnotizer. Sometimes, but by no means always, the very first attempt to bring about hypnosis is successful, as I have seen in some of Forel's as well as Wetterstrand's cases, and the hypnosis may be so profound that we can already venture to give therapeutic suggestions. Sometimes the first, second, and third attempts fail completely or partially ; then we must, if no contraindications exist, try again and again, but under no consideration should the individual trials be prolonged beyond two or three minutes. Without question external circumstances are of great significance. If a patient who is to be hypnotized enters a room, in which eight, ten, or twelve persons are lying sound asleep stretched out on easy-chairs and sofas, and is left sitting there quietly for a quarter of an hour without any attempt to put him to sleep, his suggestibility—that is, his susceptibility—will sometimes be materially increased, and it will be a comparatively easy matter to hypnotize him. But there are certain internal conditions also which may throw great obstacles in our way, and which must, therefore, not be overlooked. Thus, if a patient does not believe that he can be put to sleep, or if he makes up his mind to resist us, a certain amount of *finesse* is necessary ; we have to outwit him in order to produce hypnosis without his consent or even against his will. Such exceptions, and the behavior of the physician who has to contend with them, can not here be treated of. Only one artifice we may mention which we have repeatedly used with very good results in producing hypnosis quickly and surely. We apply a large curved sponge electrode (anode) to the forehead, a second to the neck, close the circuit and allow a very weak (constant) current, just sufficient to produce the characteristic taste upon the tongue, to pass through the head for a few seconds, and then, without the knowledge of the patient, open the circuit and tell him that the electricity passing

through the brain will put him to sleep, and as a matter of fact this· "suggested" current does so very promptly and surely. Secondly, the mental condition of the patient may stand in our way. It is an observation confirmed by all investigators that it is difficult or impossible to hypnotize insane patients, and that hysterical patients and hystero-epileptics are the least favorable subjects. In the domain of psychiatry the treatment by suggestion, so far as we can judge at present, remains without significance; on the other hand, it seems as if certain disturbances in nutrition—for example, general anæmia and chlorosis —facilitate hypnotization greatly, while an absolute confidence in the physician, the absence of all attempts to analyze and to test our procedures on the part of the patient while we are trying to hypnotize him, will also materially increase the susceptibility to suggestions. If all factors, favorable and unfavorable, are taken together, we may say that by far the greater number of people can be hypnotized; perhaps one might go so far as to say all, without exception, are susceptible if time and circumstances allow sufficient repetitions of the trial. For hospital practice the dictum of Bernheim may for the present be accepted, that the physician who does not succeed in hypnotizing eighty per cent of his patients for therapeutic purposes does not understand the method.

The manner in which hypnosis comes on and the phenomena observed during this state are extremely varied. Sometimes the eyes close suddenly and the patient is asleep at once; more frequently this is preceded by twitchings of the lids, and moisture in the eyes, which are repeatedly closed and opened. Sometimes the lids are shut during hypnosis, sometimes a fine tremor is noticeable in them; again, fibrillary twitchings in the muscles of the face may be remarked. The hypnotic influence does not always produce sleep, but there are different degrees, from the waking state to slight dullness of the senses and somnolence, and, finally, deep sleep, which latter is called somnambulism. Bernheim in his explanation bases his arguments upon the ideas of Luys, that the different layers of the cortex are endowed with different functions: those nearest the surface are supposed to serve for the sensorium, the middle ones for the mental faculties, and the deepest for the transference of the will. He distinguishes accordingly nine degrees of hypnosis, and characterizes them in the following manner: (1) The patient remains quiet with closed eyes during the suggestion, but

can open them without difficulty when asked to do so, and claims not to have slept at all. (2) The patient is not able to open his eyes when asked. (3) The patient presents suggested catalepsy and analgesia, and remains in the position in which he is placed, but is able, after it has been suggested to him, to change from one position to another without assistance. (4) The patient is no longer able by himself to overcome the suggested catalepsy, and automatic, rotatory movements, especially of the arms, can be evoked. (5) Besides the catalepsy, contractures can be produced which the patient himself is not able to do away with. (6) The patient presents an automatic obedience ; he stands motionless if ordered to do so, he rises, walks, and acts, in fact, just as the hypnotizer may suggest. Intelligence and the activity of the senses are intact in these six stages. The patient on awakening remembers everything that has been done to him. (7) In the seventh stage the patient presents the same phenomena as in the preceding six stages, but on awakening has quite forgotten what has been going on. (8) Besides this amnesia on coming to, hallucinations can be produced during hypnosis which vanish after the return to the normal condition. (9) The suggested hallucinations persist after waking up—post-hypnotic suggestions—everything that can be produced in a patient when in a state of hypnosis can be brought about after he has awakened simply by suggesting to him during hypnosis that it will happen after he has awakened. In this possibility, of exerting an influence upon the patient for a longer or shorter time after he is awake, lies the whole therapeutic significance of the treatment by suggestion. This (post-hypnotic) action, which in certain cases can be obtained in no other way than by suggestion, is sufficiently important to warrant and insure to hypnotism a lasting place in science. It is unnecessary to distinguish nine different stages of hypnosis as Bernheim did ; three are quite sufficient (Forel). The first is the stage of somnolence, corresponding to Bernheim's first stage ; the second is that of hypotaxia (light sleep), embracing all the stages from the second to the sixth of Bernheim ; the third is the stage of deep sleep (somnambulism), corresponding to the seventh, eighth, and ninth of Bernheim's classification. It is of practical importance to note that frequent hypnotizing usually increases the susceptibility of the patient, and that we can only succeed after repeated successful trials in producing deep sleep with amnesia, and be able to expect that our post-

hypnotic suggestions will be carried out. This is the rule, although we occasionally meet with individuals in whom on the first trial we are able to recognize all the characteristics of the state of somnambulism.

What are, then, the diseases in which we can, with good conscience and good hopes of success, venture to employ the treatment by suggestion? We need hardly say that affections in which we have to deal with inflammatory processes, new growths, infections, or, in a word, with organic lesions, do not belong to this class; and, as a matter of fact, it would hardly enter any one's head to attempt to cure pneumonia, typhoid fever, brain tumors, syphilis, tetanus, etc., by means of hypnotism. It is a different matter if we are dealing merely with certain symptoms of such maladies, for instance, insomnia, difficulty in breathing, or pains of the most varied kinds, not excluding the lancinating pains of tabes. Here hypnotism should, at least, be given a trial, yet the main field in which the treatment by suggestion should be employed will not be the diseases we have mentioned, but rather all those which we have designated and described as functional disorders of the nervous system. Here, motor as well as sensory disturbances can be influenced, various neuralgias, including tic douloureux and migraine-like paroxysmal headache, sensory paralyses, the anæsthesias and paræsthesias of hysteria, etc., symptoms of motor irritation and paralysis, tic convulsif, writer's cramp, chorea, the tremor of paralysis agitans, and so forth. According to Forel, it is possible to influence certain somatic functions to some extent— e. g., the menstruation and digestion—in such a manner that the menses can be brought on at a certain day and a certain hour, and a regular evacuation of the bowels every day can be insured by suggestion. Although these accounts come from the most indubitable source, the experiments must again be tested and confirmed. They can certainly only be successful, we should think, in individuals who have repeatedly been hypnotized and are, as it were, "trained." With the treatment of alcoholism by suggestion Forel has had uncommonly good results in his institution. The heaviest drinkers were not only for a time, but lastingly cured; but no little influence certainly has here to be attributed to the temperance societies, which such individuals entered. Without constant watching and moral support, relapses would surely occur in such patients; nevertheless, the influence of hypnotism in inebriety is of

great importance. The behavior of morphinists toward suggestion has yet to be studied. The results so far obtained seem not to be very encouraging. The communication of Wetterstrand (cf. lit.) that it is possible in idiopathic epilepsy to diminish the frequency and severity of the attacks deserves to be remembered and tried in cases in which bromides are not well borne. Finally, we would call attention to the anæsthesia and analgesia which can easily be produced by suggestion, and which in surgery, as well as in obstetrics, may be very useful. I was present at Forel's clinic at the extraction of two obstinate teeth, which, after the proper hypnotization, were taken out without the slightest sign of pain on the part of the patient. Possibly the pains during labor may be removed by hypnotism. The anæsthesia of the mucous membrane of the fauces may be very valuable in making laryngoscopical examinations and the like. We purposely refrain from entering into further details, but shall content ourselves with advising every one, who approaches the subject of treatment by suggestion with sufficient true and serious interest, to study the work of Bernheim, which constitutes the foundation of this subject.

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PART II.

DISEASES OF THE GENERAL NERVOUS SYSTEM WITH KNOWN ANATOMICAL BASIS.

THE anatomical changes, which are found in the diseases belonging to this category, concern the central nervous system as well as the peripheral nerves. The former always suffers, the latter are only in certain cases affected. Whether the changes in the peripheral nerves are to be regarded as secondary, or whether the entire nervous system becomes affected in all its parts at the same time, so that the peripheral and the central lesions progress *pari passu*, can not be definitely decided. The nature of the anatomical changes will be discussed under the head of each individual affection.

CHAPTER I.

MULTIPLE SCLEROSIS—DISSEMINATED SCLEROSIS—INSULAR SCLEROSIS—SCLÉROSE EN PLAQUES—SCLEROSIS CEREBRO-SPINALIS DISSEMINATA S. MULTIPLEX.

ALTHOUGH multiple sclerosis is not one of the common affections of the nervous system, it is desirable and important for the general practitioner to possess a clear understanding of it, because the clinical appearances by which the different cases manifest themselves vary within such wide limits and remind us now of this, now of that spinal or cerebral affection, without ever completely simulating any one definite disease. The typical course given in the books is not very often met with in practice. Much more commonly one or the other of the classical symptoms is not present at all, or, if present, only very slightly developed. On the other hand, symptoms are occasionally encountered which are not included in the usual

descriptions of the disease. In a word, multiple sclerosis is very inconstant in its manifestations, a circumstance which often makes the diagnosis very difficult. In the investigation into the pathology as well as the clinical aspect, Charcot has done admirable and lasting service.

Symptoms and Course.—The course of a classical case is usually as follows: The patient first complains of general symptoms—headache, vertigo, digestive disorders—soon, also, of sensory disturbances in the upper and lower extremities, slight weakness, and a readiness to become fatigued. These symptoms may persist for months, yet relatively early one or several apoplectiform attacks may occur which sufficiently in-
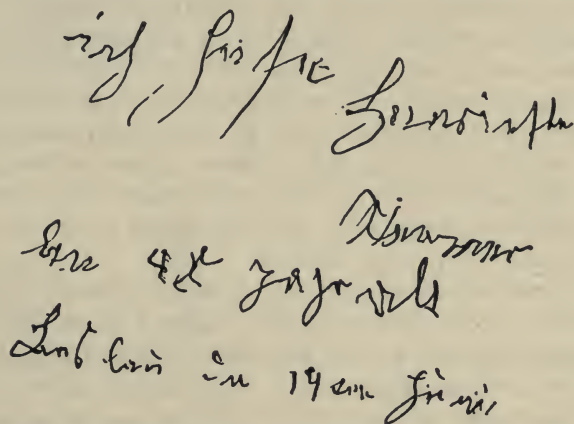
Fig. 161.—Specimen of Handwriting in a Case of Multiple Sclerosis. (Ich heise (heisse) Henriette Sterner, bin 48 Iahre alt. Breslau, den 19 Juni).

dicate the seriousness of the condition. It strikes the patient, as well as those who surround him, as a peculiar thing, that whenever he attempts to pick up something with his hands, or to make any other movement, a tremor appears which frustrates the intended movement more or less completely. If he attempts to raise a full glass to his mouth, he spills some of the contents. If he attempts to eat, the food is jerked off his fork, etc. Co-ordinated movements, such as are required for writing or playing the piano, become difficult, the handwriting becomes almost illegible (Fig. 161), and the condition is materially aggravated if the tremor is not confined to the upper, but if also the lower extremities, the trunk, neck, and head are attacked, so that on voluntary movements—on attempts to walk, for instance—the whole body first begins to tremble, and finally

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shakes so violently that the patient is forced to sit or lie down at once. This symptom, which is almost pathognomonic for multiple sclerosis, or at any rate most significant, is called "intention tremor," a term which does not, however, imply that the tremor is "intentional," but only that it appears on voluntary ("intended") movements. During rest no trace of it is observed. When the patient lies quietly and undisturbed in bed no tremor is present, whereas, if he is spoken to, examined, made to answer questions, and the like, a tremor over the whole body develops, which of course presents various degrees of intensity. It is most marked and characteristic if the patient is asked to bring his hand slowly to an object—for instance, to a pin laid upon the table. At first the motion is fairly good and steady, he trembles but little or not at all, but the closer he approaches to the pin the more unsteady becomes the hand and the larger become the excursions of the tremor, so that to grasp the pin becomes impossible. In some exceptional cases I have seen the shaking movements appear on one side only, so that the patient was capable of performing normal movements with one hand and one leg, when those of the other side had become entirely useless.

In this intention tremor the eye muscles also take part; as soon as the patient attempts to fix a point with his eyes nystagmus appears, which, however, differs from the tremor of the other voluntary muscles, inasmuch as it does not completely disappear during rest. As a subjective symptom the very annoying sensation of giddiness must be mentioned in this connection, which leaves the patient only when he lies quietly in bed, whereas it otherwise impedes him a good deal in his movements, especially in walking. Owing to the faulty innervation of the tongue and larynx, we meet with a peculiar speech disturbance; the patient talks slowly, in a monotonous tone, and awkwardly, and his speech is scanning, as he makes a pause after each word, almost after each syllable, so that it takes him a much longer time to express his thoughts than a healthy man: "Yes—doctor—I—am—very—much—fa—tigued —and—worn—out." As this is spoken in the manner indicated, without any change of intonation it is very characteristic indeed, and it is, together with the intention tremor and the nystagmus, pathognomonic for multiple sclerosis. It impresses itself so much upon the mind that once heard it can never be forgotten or misinterpreted.

To give a physiological explanation of the intention tremor is out of our power, and it is more especially not clear why it is so extremely common in multiple sclerosis, where we have such an irregular distribution of the anatomical lesions, whereas in most of the other cerebral affections it is absent. Whether Charcot's idea is correct, according to which the long persistence of the axis cylinders in the sclerotic foci has some connection with the tremor, or whether we should hold with Strümpell that the loss of the myeline sheaths, in consequence of which an abnormal diffusion of the nerve current from fibre to fibre occurs, is responsible for this, we can not decide, nor have we any proof of the correctness of Stephan's view (cf. lit.) that the existence of sclerotic foci in the thalamus gives rise to the phenomenon, nor of Cramer's (cf. lit.) that the intention tremor has to be explained as analogous to the tremor which comes on after hard muscular exertion.

Though we may be justified in looking upon these three symptoms as constituting in a manner the typical picture of multiple sclerosis, we must, as we have said above, at once familiarize ourselves with the fact, that even these may not all be pronounced, or again that there may be others to be found in conjunction with them, developing in the course of the disease. Among these latter we may mention certain spastic symptoms, rigidity of the muscles, increased tendon and skin reflexes, the above-described spastic walk—which, together, are liable to simulate, at least for a time, the picture of spastic spinal paralysis. This is the more likely as there are no sensory disturbances at all to be noted in multiple sclerosis; only in rare exceptions paræsthesias are observed, owing to which tabes and myelitis may be diagnosticated, especially if, as sometimes happens, bladder disorders are superadded. That these latter are by no means as rare in multiple sclerosis as was formerly supposed, has recently been pointed out by Erb, and after him by Oppenheim (Deutsche med. Ztg., 1889, 32). Glycosuria will be found associated with the disease if there are foci situated in the floor of the fourth ventricle (Richardière, Revue de méd., July, 1887).

Participation of the optic and other cranial nerves is not very rare, yet it is here much less important for the diagnosis, and much less significant for the course of the disease than, for example, in tabes. Diplopia is rarely met with; and equally uncommon is the neuritis and atrophy of the optic which leads

to amaurosis.   Uhthoff (cf. lit.) has recently pointed out in an admirable study, that if optic atrophy occurs it is not like the primary atrophy in tabes, but that here it is a secondary process, which follows an active increase of the fine connective-tissue elements.   It is self-evident that various disorders of sight are associated with this, yet they often present temporary improvement, and have usually a less serious issue than those of tabes.   In general, it is characteristic of multiple sclerosis that its course is not uninterruptedly progressive, but that it shows remissions, during which the hopes of the patient as well as of his friends for his complete recovery are aroused. I have seen instances in which such remissions lasted for years and the symptoms disappeared to a great extent, and in which, just owing to this peculiarity in the course of the disease, the diagnosis could be made with some certainty.

Cerebral manifestations are not uncommon, and frequently a slight degree of dementia—a psychical weakness—develops, which to the patient himself makes his condition more bearable.   It must also be regarded as a sign of beginning mental weakness, I think, that in some cases the patient frequently laughs boisterously without a cause.   One of my patients had spells of loud laughter, which lasted from one to three minutes, and which appeared usually without sufficient motive.   I have never had occasion to observe pronounced states of depression or exaltation in the course of this disease.   The vertigo, which of course must also be regarded as a cerebral symptom, has been spoken of above.   Apoplectiform attacks in the beginning of the disease are not rare; epileptiform seizures may be found, if the cerebral cortex is more especially implicated.

It has been shown by Charcot that in certain cases the development of the symptoms appears to be abortive and the affection, one might almost say, remains latent and can only be recognized by the peculiar shaking tremor.   He proposed for these instances Trousseau's designation, "*formes frustes*," and it seems that in multiple sclerosis such forms are observed relatively frequently.   As an example of the general course which the disease may run I insert here the following history of a patient in my wards, who is still living:

Paul W., thirty-one years old, began to be sick ten years ago, during his military service.   At first, at times he could not feel his rifle in his left arm, and then in the same year he was often conscious

of a slight feeling of fatigue, which was associated with vertigo. He had a good deal of difficulty with his arms and his legs; they always felt as if they were asleep, and any muscular action necessitated the greatest exertion. He could not go through his salutes in the proper manner, and he was repeatedly punished on this account. At the same time he had now and then vomiting and weakness of the bladder for quite a long time, so that, on coughing, small quantities of urine were passed involuntarily. On examination, we are told, Romberg's sign was absent and the patellar reflexes were increased. A few months later, marked weakness in the right arm and the right leg became manifest, and the acuteness of hearing became diminished on that side. The patient complained of an annoying double vision. In 1879 he had some difficulty in swallowing; the bolus would stick in his throat, so that he had to force it down. In 1880 pronounced deliberation in speaking is said to have been noticeable, and the patient at that time also complained that he could not lay his tongue upon certain words which he wished to use. The speech disturbance soon passed off, but the patient suffered from various troubles till January, 1884, in which month I saw him for the first time. He then presented the symptoms of an incipient tabes, but it was noted as a remarkable feature that the patellar reflexes were retained. The lancinating pains, however, the paresis of the legs, the diplopia, the paresis of the bladder, the unsteadiness, which appeared especially in the dark, seemed sufficient to warrant the diagnosis of tabes, and, in the out-patient department, this diagnosis was made, although with some reservation. The patient declined to enter the hospital. He was therefore ordered galvanism, but was lost sight of in the summer of 1884. Two years later he was treated at a hospital in this city for six months. Although I was unable to obtain a record of the case, I heard that the tabetic symptoms were very indistinct, and that the condition suggested rather a spastic paralysis. The patient was again lost sight of. Finally, on January 8, 1888, he was admitted to the medical ward of the city almshouse, where he still remains. From a note made on January 10, 1888, the following is extracted: The patient is a well-nourished man; as he lies quietly in bed, the general aspect suggests nothing abnormal; if, however, he is asked to perform any movement, the whole body—trunk, head, and extremities—are seized with a violent shaking tremor, which makes it difficult for him to get up, and impossible for him to walk without assistance from another person, even when supported by two canes. If he is allowed to discontinue all attempts at moving, the tremor gradually abates, and five or ten minutes later he is perfectly quiet again. The patient is unable to feed himself, and can not occupy himself with anything. The mus-

cular strength is retained everywhere.   In the domain of the cranial
nerves nothing but nystagmus can be noticed, which is especially
well marked on the right side.   The facial, hypoglossal, etc., are
normal.   The tongue is protruded steadily and straight.   Speech is
slow, although not distinctly scanning.   There are no motor or sen-
sory speech disturbances.   The tendon reflexes in the upper, but
more especially in the lower extremities, are increased, and the skin
reflexes are without exception well marked.   Sensory changes can
nowhere be demonstrated, and the bladder disturbances, which were
present on previous occasions, have disappeared.   The spinal column
is nowhere tender on pressure.   Among the subjective complaints
of the patient the dizziness is alone to be mentioned, which, however,
even if the shaking movements were not present, in itself would be
sufficient to keep him from doing anything.

As a result of this examination the diagnosis of multiple sclerosis
was made, and will certainly be proved to be correct at the post-
mortem examination.   It is interesting, however, that in this case
the course of the disease suggested in its initial stages Thomsen's
disease (although not congenital), later tabes (with retained knee-
kick), then spastic spinal paralysis (conjectural diagnosis), before
the picture of insular sclerosis developed.

Diagnosis.—The diagnosis presents difficulties in almost
every case, owing to the protracted course and the changes in
the picture of the disease during the different periods.   Even
the most careful examination will not always keep us from
errors, and we must never be surprised if the autopsy does not
always confirm the diagnosis made during life.   The case of
Westphal in which a multiple sclerosis was diagnosticated,
but where *post mortem* no lesions at all were found, has been
alluded to before.   In another instance, reported by Frey (cf.
lit.), there was found, instead of the confidently expected foci
of sclerosis, a leptomeningitis, and similar errors would not be
difficult to find on a careful perusal of the literature.   The pos-
sibility that we are dealing with hysteria, in a given case, must
always be considered, and then, of course, great weight must
be laid upon the presence of other symptoms which would
indicate such a condition.

Pathological Anatomy.—The anatomical changes of multi-
ple sclerosis are extremely characteristic.   Even with the
naked eye, here and there, grayish white foci are seen in the
brain, in the white matter of the hemispheres, in the walls of
the lateral ventricles, in the corpus callosum, in the pons, and on

its surface, in the medulla oblongata, in the floor of the fourth ventricle, and in the spinal cord, where the white matter is decidedly more affected than the gray. The foci are distributed in a very irregular manner; sometimes they are more numerous in the brain, sometimes they are more numerous in the cord, often they are found scattered equally over the entire central nervous system. If they are situated on the surface, they are seen through the pia, and are somewhat more prominent than the parts which surround them. They are
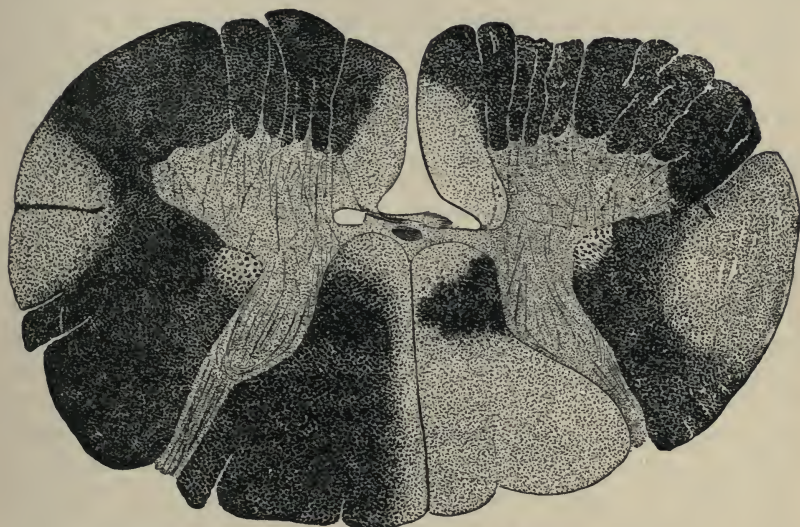
Fig. 162.—Cross-section through the Cervical Enlargement of the Spinal Cord in a Case of Multiple Sclerosis. Hardened in osmic acid. The lighter colored areas in the white matter represent the sclerotic foci. (After Bramwell.)

generally harder and firmer than the rest of the substance, and on section they assume a light-pink color when exposed to the air. If they are examined microscopically, they are found to consist of reticulated fibrillary connective tissue, and only contain a few intact nerve fibres; after the death of the medullary sheaths, the axis cylinders are preserved for an extraordinary length of time (Charcot). Secondary degenerations in the spinal cord are often absent (Strümpell), yet they are occasionally seen (Werdnig). The vessels show an increase in the nuclei, later a thickening of their walls, and are seen as yellow dots in the sclerotic foci. Whether the disease of the vessels actually gives rise to the foci is not yet established (Fig. 162).

Disease of the peripheral nerves has never been demonstrated in multiple sclerosis.

Ætiology.—About the ætiology we know practically nothing. It is possible that heredity deserves some consideration in this connection, but there are relatively many cases where this factor can positively be excluded. The influence of infectious diseases upon the development of insular sclerosis has recently again been dwelt upon by Marie (cf. lit.). Typhoid fever, variola, scarlet fever, measles, whooping-cough, and intermittent fever have repeatedly been known to precede the disease, although the material at our disposal is not as yet sufficient to prove a causative relation between the two. With regard to syphilis the connection here is by far less definite than, for instance, in tabes (cf. the case of Buss, lit.). Age and sex seem to be of some significance, inasmuch as children and aged people seem to be exempt. Westphal and others have only exceptionally seen it in children. Strümpell has observed it in a man of sixty. Both sexes seem to be attacked with about the same frequency.

Treatment.—An effectual treatment for multiple sclerosis does not exist. We possess no remedy which will arrest the development of the foci. The symptomatic treatment must always be tried, however, and the patient particularly seeks relief from the annoying tremor. For this we may administer, although without raising our expectations too high, veratrine, physostigmine, one to three milligrammes (grs. $\frac{1}{60}$–$\frac{1}{20}$) daily, in pills, or solanin, recently recommended by Grosset and Sarda, and even termed by these authors "*médicament du faisceau pyramidal*" (Progr. méd., 1888, 27). It may be given in doses of from two to three centigrammes (grs. $\frac{1}{3}$–$\frac{1}{2}$) from three to five times daily. In other respects the treatment is the same as in myelitis (cf. p. 448).

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CHAPTER II.

THE second of the diseases belonging to this group certainly deserves to be considered as one of the most important of those with which we are acquainted, not only because it is one of those diseases of the nervous system which occur relatively frequently, and with which the general practitioner is not rarely brought face to face, but also because its clinical picture presents so many essential differences that it requires a large experience to feel at home with it on all occasions. Nobody questions the importance of recognizing the disease in its early stages if only on account of the bearing of this upon the treatment, but many do not appreciate the difficulties which this early diagnosis entails. The more cases of tabes we see, the more are we surprised at the protean character of the symptoms, and the more are we convinced that almost every case offers some point of particular interest, and that occasionally even an expert can be sure of the diagnosis only after repeated examinations and long observation.

Symptoms.—The symptomatology of tabes is so comprehensive that in order to get a clearer idea of it we shall separate the cerebral from the spinal symptoms in our description.

The cerebral symptoms which appear in the course of the disease are referable either to the cranial nerves or to the brain substance. When the latter is affected, it is sometimes the cortex, at other times the white substance, and again the basal ganglia, which are most implicated.

When we consider the cranial nerves, we shall find that there is hardly a single pair which can not be affected and in which lesions have not been repeatedly described in cases of tabes. However, not all of them are implicated with the same frequency, as we shall see later. The nerves supplying the muscles of the eye are those most commonly, the facial that

most rarely attacked. Between these extremes we may put in descending order the vagus, the optic, the fifth, the olfactory, the glosso-pharyngeal, the accessorius, the hypoglossal, and, lastly, the auditory. We shall take them up here in the order of their anatomical position, as we did in Part II.

The lesions of the olfactory nerve possess no great practical significance, and it is not quite certain that they are not more frequent than is generally supposed. They consist of a weakening or even a total loss of the sense of smell, or in the perception of peculiar, often disgusting, odors, as we have shown on page 25. We do not know whether these changes are due to anatomical lesions or only to some functional disturbances, and but little is known about the course of such disorders of the sense of smell. Occasionally, when examining into the condition of the sense of taste, one may accidentally discover an affection of the sense of smell without being able to ascertain how long it has already existed, as it can easily have escaped the notice of the patient. Only those who use tobacco or snuff perceive the defect very early and appeal to a physician for advice and help. Unfortunately, we can do but little. The treatment of these affections has been described on page 27.

The most frequent lesion of the optic nerve in tabes is atrophy or gray degeneration. Usually both eyes become affected, if not simultaneously, at least within a short time of each other, and it is quite rare for one eye to be diseased while the other remains healthy for any great length of time. The patients complain that everything seems as if covered by a gray veil. The loss of vision is particularly rapid at first; it then becomes much slower, and the complete amaurosis occurs much later than one would have expected from the brusque onset of the trouble. Along with this, a narrowing (not always concentric) of the visual field appears, as the peripheral portions of the retina are the first to become impaired in their functions. The perception for color may also be affected, as we pointed out on page 33. The order in which these changes occur is not always the same. As a rule, however, the loss of color perception and the narrowing of the visual field precede the lessening in acuteness of the central vision, and it is exceptional to find diminished acuteness of vision and marked disturbances of color sense combined with a normal visual field.

The ophthalmoscopic examination shows a pale grayish white or bluish white, but not pure white, discoloration of the disk, which is thought to be produced by the obliteration of numerous fine vessels in the optic nerve. When the amblyopia is marked but no perceptible changes in the disk are found, we must think of a retrobulbar degeneration of the optic nerve. From a pathological standpoint we are dealing with a degenerative atrophy, first of the medullary sheaths, and then of the axis cylinders. The theory that these changes are due to an action of the sympathetic nerves or to changes in the vaso-motor nerves brought about by the spinal disease is quite untenable, for the process is a neuritis in which we have a wasting of the nerve fibres and changes in the interstitial tissue, such as have been described on page 328.

The prognosis of the optic atrophy is altogether unfavorable ; although a slight improvement or a temporary arrest of the process may give the patient a delusive hope of recovery, the termination is always in total blindness. It is true that the process may take several years, during which the patient is still able to find his way about by himself with the aid of a stick.

With such a prognosis we shall not be surprised if the treatment is without avail. The subcutaneous injections of strychnine, one milligramme (grs. $\frac{1}{60}$) twice daily in the neighborhood of the eye, as proposed by some, are of value only because they give the patient the comforting satisfaction that something is being done for him, but they really have no curative properties, and it is improbable that they even postpone the unfavorable issue.

In a few isolated cases transient lachrymation has been observed (Patrolacci, Thèse de Montpellier, 1886; Féré, L'Encèphale, 1887, vii, 4).

The nerves which supply the eye muscles—the third, the patheticus, and the abducens—the affections of which have already been considered in Part II, Chapter III, frequently become attacked in the course of tabes. As a consequence of paralysis in the ocular muscles, one often notices diplopia, which may appear suddenly, and after a longer or shorter duration disappear as quickly. It may recur repeatedly and be a source of great annoyance to the patient in his daily occupations. An abducens paralysis may also occur by itself, and, finding this, one should always look for a commencing tabes, for it is fre-

quently the first sign of this disease in an apparently quite
healthy person. If the affection remains stationary, it is to be
regarded as being due to a nuclear lesion ; the same remarks
apply to a ptosis which, occurring by itself, is also a suspicious
sign, and should lead us to look for tabes. In cases of oculo-
motor paralysis the lesion is also relatively frequently nuclear
(page 44).

Several of the extrinsic eye muscles may be affected at the
same time, and an ophthalmoplegia externa is not infrequently
observed in the course of tabes. De Watteville especially has
directed attention to the paralysis affecting the movements of
convergence in the initial stages of the disease.

The behavior of the intrinsic eye muscles is not less inter-
esting, and the condition of the pupils deserves the most thor-
ough examination ; they are rarely normal and of the same size
in both eyes. Frequently some abnormity of reaction is de-
monstrable ; the marked contraction (myosis), the difference in
the size of the two pupils (anisocoria), and the loss of the light
reflex have already been mentioned.

The ophthalmoplegia interna of Hutchinson, in which be-
sides the loss of the light reflex there is also paralysis of the
muscles of accommodation, is much rarer. The pupils of those
afflicted with tabes may frequently be found to dilate promptly
and normally under strong and painful irritation of the skin, as,
for example, that produced with the faradic brush.

The *rôle* which the affections of the fifth nerve play in this
disease is quite subordinate ; paralytic conditions of either its
motor or sensory branches as the result of tabes have, it seems,
never been observed except in Westphal's case, in which there
was degeneration of the ascending root of the fifth, and among
the signs of irritation only the headache, traceable to the nerve
endings in the dura, is occasionally met with. A certain rela-
tionship is said to exist between tabes and genuine migraine,
but in considering these cases one must make sure that the mi-
graine has not been inherited, and furthermore note whether
the attacks become more or less severe after the development
of the tabetic symptoms. According to some observations, in
such cases the headache of the migraine becomes less and
less severe, and eventually disappears, while the nausea and
vomiting still persist, so that it is then impossible to say
whether we are dealing with a gastric crisis of tabes or with
an abortive attack of migraine. In certain cases of hemi-

crania, if there has been, for instance, a syphilitic infection at some previous time, it is always well to examine carefully for any traces of tabes, more especially for the absence of the patellar reflex. Occasionally one meets with a paræsthesias in the face, the patient complaining of a sensation as if one half of the face and the lips were swollen ; this is probably also due to an affection of the ascending root of the fifth nerve.

Lesions of the facial nerve are so rare in tabes that, when they occur, one can not help raising the question whether they are not merely to be regarded as accidental complications. Among two hundred and forty-seven cases of locomotor ataxia, I have not observed one in which any of the muscles supplied by the facial were affected.

About the same may be said of the auditory nerve. There is no question but that lesions of this nerve may be caused by tabes or develop in the course of the disease, but they are very rare indeed ; they manifest themselves by a diminution or a total loss of the power of hearing. The patients complain of deafness, which may have developed gradually or have come on acutely. In both cases the symptoms are due to organic disease of the nerve ; in the former we have to deal with a gradual gray degeneration of the nerve trunk, in the latter with a nuclear affection. Too few cases, however, of involvement of the auditory nerve in tabes have as yet been observed to enable us to speak with much certainty of their pathology (Hermet, L'Union méd., 1884, 86). Under what conditions the so-called Ménière's symptoms appear in the course of tabes requires to be studied more closely. I have seen them in two of my cases, but they disappeared again in a few weeks, and in these cases, unfortunately, no anatomical examination of the internal ear could be made.

Functional disturbances of the nerve of taste have now and then been described in the course of tabes. In a few instances, as in the case of Erben, which we considered on page 106, the nucleus of the glosso-pharyngeal nerve was degenerated, and during life such derangements of the sense of taste existed that the patient was at times unable to distinguish sweet things from those which were acid or salty. To these lesions no great practical significance can be attached.

On the other hand, there is a great variety of manifestations associated with tabes which are due to lesions of the vagus. In this connection we have disturbances more gener-

ally of the digestive, but also of the respiratory and circulatory organs. They occur with irregularity, and may disappear again quite suddenly. Following the suggestion of Charcot, we designate them "crises." Of those affecting the digestive system the so-called "pharyngeal crises" are relatively the rarest. These consist of paroxysmal movements of deglutition, which occur from twenty to twenty-four times a minute, and succeed one another in this way for ten or twenty minutes; the attacks may be associated with a noisy inspiration, and may suggest hysterical singultus; in some cases they can be produced at will by pressure on the side of the larynx (Oppenheim).

The gastric crises are far more frequent; they consist of paroxysmal attacks of retching and vomiting, during which the patient, without any particular exertion, may vomit large quantities of strongly acid, slimy, or watery material, after which he feels greatly relieved. These attacks are repeated for several days, sometimes for a week or two, once, twice, or even oftener, every day, and then disappear entirely for a longer or shorter period. In some cases the vomiting is associated with cardialgia, but usually it is uncomplicated. It is not at all connected with the taking of food; indeed, it not infrequently occurs early in the morning when the stomach is empty, and if the patient be a drinking man it may arouse a suspicion of the morning vomiting of drunkards. The differential diagnosis is, however, not at all difficult; if the vomiting be associated with vertigo, a sensation of anxiety, and a quickening of the pulse, it can not be considered simply as a "gastric crisis." This paroxysmal vomiting is of the greatest importance for the diagnosis of tabes. It is frequently regarded as dependent upon some stomach trouble and treated as such for a long period without any sign of improvement, until finally, perhaps by accident, our attention is drawn to some other symptom which places the diagnosis beyond doubt. If a person have paroxysmal vomiting and complain occasionally of violent rheumatoid pains in the legs, we should examine most carefully for tabes, and we shall frequently be surprised at the ease with which we can make the diagnosis, and wonder that we had ever been under the impression that the patient had simply "chronic gastritis" and "rheumatism." The statement of Eckert (Die intestinalen Erscheinungen der Tabes. Inaug. Diss., Berlin, 1887) that gastric crises must be divided

into those of central and those of reflex origin deserves to be investigated more closely. In the central form he assumes, besides a general condition of irritation in the brain, some affection of the nucleus of the vagus, in the reflex form a peripheral irritation of the vagus which, under certain circumstances, may be produced by the ingesta. He holds that in the latter cases the vomiting is not associated with any distressing nausea, so that the patient suffers relatively little.

Sometimes intestinal disturbances manifest themselves by intense "lightning" pains about the rectum and anus, the "anal crises"; in other cases by tenesmus, which forces the patient to go frequently to stool, though he is able to pass little or nothing; and, lastly, by the so-called tabetic diarrhœa, about the causation of which we are absolutely ignorant. This diarrhœa may be more or less persistent, and be followed by an equally protracted and obstinate constipation. Incontinence of fæces is rarely present, though on rectal examination we shall occasionally discover sensory disturbances, particularly anæsthesia of the mucous membrane. Paræsthesias may also occur, and the patient may experience a sensation as if he had a foreign body in the rectum.

By "laryngeal crises" we mean those paroxysms of dyspnœa which may occur when the patient is lying down, or, in other cases, only when he attempts to move or walk about. Sometimes they appear in the form of peculiar suffocative attacks, accompanied by violent coughing, and are often preceded by a sighing or whistling inspiration. These attacks may last several minutes, during which the suffering may be so intense that the patient gives up all hopes of recovery. Attacks of even moderate intensity, in which a long, sonorous inspiration follows several short expirations, are most disagreeable for the patient, and appear very serious; under some circumstances they may be mistaken for whooping-cough. These crises are caused by changes of temperature, speaking for a long time, or by strong odors, smoking, etc. The result of the laryngoscopic examination is frequently negative; in other cases one finds paralysis of some of the laryngeal muscles; here also, in all probability, we should distinguish a central and a reflex form.

Abductor paralysis—i. e., paralysis of the muscles that open the glottis—sometimes occurs among the early signs of tabes, and may lead to serious danger of suffocation; but we are

unable to say whether this should be attributed exclusively to paralysis of the abductors, or to spasm of the adductors alone, or to both conditions. We may consider the condition described by Gray (Brain, January, 1888), in which the voice often breaks and takes on a high falsetto, as a kind of " laryngeal ataxia."

Attacks of angina pectoris, with all its characteristic symptoms, are rarely met with in tabes, though Vulpian, among others, has seen them (Revue de méd., 1885, v, 1).

Lesions of the accessorius are considered as rarities in the course of tabes. In a case observed by Martius (cf. lit., page 141) there was an atrophic paralysis of the upper portions of both trapezii, while the sterno-cleido-mastoids, which also receive fibres from the cervical plexus, were not affected. Whether, and if so under what circumstances, one or both of the two nuclei of the accessory nerve are affected (the nucleus accessorius vagi and the nucleus spinalis) we are absolutely ignorant. It is also uncertain whether symptoms of irritation in the domain of the accessorius—e. g., torticollis—do occur in the course of tabes.

Among the lesions of the hypoglossus there is one which deserves a special mention in this place—that is, the hemiatrophy of the tongue described above, which Ballet (lit., page 148) stated was relatively often observed. He even went so far as to say that, when one found this hemiatrophy, tabes should always be suspected. We can only agree with him to a limited extent. We have certainly found hemiatrophy in cases of tabes, but we must remember that it is in itself a rare affection, and that it exists more often independently than associated with tabes. In addition to the two cases which we mentioned on pages 146 and 147, we desire to record another (Fig. 163), in which there was likewise no trace of locomotor ataxia. It seems that the hypoglossal nucleus is not very liable to the degenerative processes of this disease.

Cerebral disturbances of the most manifold variety appear in the course of tabes, and we would in the first place call attention to the paroxysms of vertigo which come over the patient when he looks up or makes quick movements of the head, and which impel him to seize the nearest object to prevent himself from falling. There may also be found psychical depression and a feeling of dread and anxiety, which in some cases may be followed by well-marked psychoses. Among

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the not very rare forms of psychoses in this disease we may mention paranoia, melancholia, and simple dementia ; but far more frequent and important than all these taken together is the progressive paralysis of the insane, which very frequently accompanies tabes. But here we must try to make out which of the two affections was the first to develop, for in some instances the tabes precedes the paralysis, while in others the reverse is the case. The process can extend from the brain to

Fig. 163.—Hemiatrophy of the Tongue in an otherwise perfectly Healthy Child (personal observation).

the cord or from the cord to the brain, as the case may be, and Westphal was certainly justified in making the statement that "in certain persons there is a peculiar disposition of the nervous system, and that this, under the influence of different exciting causes, the action of which we do not understand, expresses itself in the form of affections either of the spinal or cerebral portion of the nervous system or of the peripheral cranial nerves, the different affections occurring in some cases nearly at the same time, in other cases at varying intervals."

Epilepsy occurring in connection with tabes has already

been considered in the chapter on the former disease. On this subject Schlieper, working under my direction, has published an article (Inaug. Diss., Breslau, 1884):

The cases of hemiplegia which occur in the course of tabes are mostly of the indirect variety—that is, they disappear in a shorter or longer time—and do not owe their origin to the rupture of vessels or to lesions of the internal capsule. The face is usually only slightly affected, and that only for a short time, and the extremities are not wholly paralyzed, but are only in a paretic condition, which usually disappears without any sort of treatment. I have repeatedly seen such cases of hemiparesis come on without any warning and with only a slight disturbance of consciousness and entirely disappear in a relatively short time. A. Bernhardt (Archiv f. Psych. u. Nervenkrankheiten, 1883, xiv, 1) has recorded instances in which they were accompanied by aphasic conditions.

So great importance has been attached to the spinal symptoms that they usually occupy the greater part of all descriptions of the clinical history of the affection, and have been allowed to predominate so far, that all other symptoms have been treated of as being of little importance and as if the only lesion was that in the spinal cord. And still, it is not rare to meet with cases in which the spinal symptoms have been for a long period of very little importance, and with a few, in which they have never attained to any prominence, while the majority of the troublesome symptoms were due to affections of the brain and its nerves, and the lesions of the peripheral nerves gave rise to more marked symptoms than those of the spinal cord. Observations of this kind, the number of which will be rapidly increased by conscientious examinations, go to show that the entire nervous system participates in the morbid process, and to consider this participation to be the rule is absolutely necessary for a correct comprehension of the pathology of this disease.

The symptoms produced by the spinal lesions concern motility, sensibility, and the reflexes.

The disturbances of motility are manifold; they depend partly on a decrease in the strength of the muscles, partly on disturbances of co-ordination. The first is not very common; on the contrary, one can frequently observe that the mere strength in the extremities has not been at all affected, and yet the motility has suffered. This condition depends, then,

upon a faulty co-ordination, and is broadly designated as "ataxia." Movements, such as walking, writing, taking hold of an object, etc., for the proper execution of which the simultaneous working together of several muscles is necessary, are designated as "co-ordinated." For such movements more than a simple innervation of the muscles is requisite; it is necessary that each concerned should receive, so to speak, the proper amount of innervation and at the proper time, so that the contraction of the various muscles may take place at the right moment. It is only when all these various factors are properly combined that the movement is correctly executed, and if one of them be disturbed the entire movement becomes ataxic. Even if not pathognomonic, it is certainly very characteristic of tabes that in the later (rarely in the earliest) stages, certain movements become ataxic, particularly those of the lower extremities, and, above all, the gait. Such abnormities are met with much less frequently in the upper extremities, and the movements necessary for writing, handling a spoon in eating, and the like, usually remain normal.

The gait of a tabetic is readily recognized even by one who has had little experience in that direction; one notices particularly that the patient exerts his eyes almost as much as his feet, that he watches every step, and in passing over small obstacles, as for example a curbstone, determines exactly where he must place his foot. If he ceases to use his eyes in this fashion for any reason, even for a short time, the movements of the legs become uncertain, and he is in danger of falling. But not even with the help of the eyes can he walk without difficulty. He does not step out in the usual way; the legs are thrown out loosely, and in putting the feet to the ground the heels come down first ("strutting gait"). The manner in which the feet are raised, the legs thrown out, the stamp with which the feet touch the ground, readily enable one to diagnosticate the tabetic gait at a distance, and we shall seldom make a mistake if we consider a person who walks in this manner, supported on a stick or by an attendant, as affected with locomotor ataxia. Acts of politeness, such as greetings and stopping to talk on the street, do not afford these persons much pleasure, for they distract their attention, which has to be kept undivided if they would walk in safety.

The uncertainty and insufficiency of the innervation of the different groups of muscles is apparent not only in the walk,

but even while the patient is standing still. He is not able to stand up straight without tottering, particularly when he closes his eyes, and he sways to and fro and falls unless some one is at hand to support him (" Romberg's sign "). The smaller the supporting basis—that is, the nearer together the feet—the more pronounced does the phenomenon become. In some cases it may be accompanied by irregular contractions of the calf muscles.

The much rarer ataxia of the upper extremities produces inability to write, to play the piano, to sew, etc. With closed eyes the patient is unable to describe circles in the air with his arms, to bring the tips of the index fingers together from a distance, or to touch the end of the nose quickly with his finger. All such movements are carried out with more or less irregularity. It is exceptional for the upper extremities to become affected at an early period or severely : as a rule, we can not detect ataxic movements in them in the earlier stages, and when they do occur they can, at least in some instances, be traced to some special cause. In the case of Bernhardt (Zeitschr. f. klin. Med., 1888, xiv, 3, page 289) they were due to the occupation of the patient. Remak (Berlin. klin. Wochenschr., 1880, 22) has also published a similar case of ataxia affecting only the upper extremities. It was associated with ephidrosis unilateralis. The helplessness of the patient reaches the most extreme degree when the ataxia affects all four extremities, as in the case of Fort (Dublin Journal of Medical Science, 3, s., 1886, clxxiii).

But we must also distinguish between spinal and cerebral or the so-called cortical ataxia (page 184). A conclusion important for the differential diagnosis may be drawn from observing the influence which the eyes exert over the co-ordinated movements. In spinal ataxia these become better regulated and more certain when they are under the control of the eyes, while in cortical ataxia such a control has no influence.

The physiological cause of ataxia is not as yet positively known, but even to-day is a source of contention and still the object of continued investigations. While some, as Benedikt, Cyon, and Jaccoud, consider that we have to do with a disturbance of the reflex activity in the cord, others, with Friedreich, and after him Erb, are of the opinion that there is a disturbance in co-ordinating fibers, the course of which they confess can not as yet be made out. Thirdly, others, with Leyden at their head, consider disturbances of sensibility to be responsi-

ble for the ataxia. According to these, interruption of con-
duction in the sensory tracts of the gray matter causes a break
of the reflex arc between the sensory nerves of the muscles
and the motor nerves. "Owing to this interruption, the un-
conscious regulation of the movements, which adapts them
to the state of contraction or relaxation of the musculature,
disappears" (Wernicke), and ataxia is the result. This "sen-
sory ataxia" has always had many opponents, for one was
obliged to confess that ataxia often occurs when no sen-
sory changes are found; but in spite of this fact some one is
constantly returning to this theory, which has lately found an
advocate in Goldscheider. In a comprehensive article (Zeit-
schr. f. klin. Med., 1888, xv, 1, 2) he subjects the meaning of the
term "muscular sense" to a fresh examination, and comes to
the conclusion that four factors are combined in the formation
of the muscular sense, viz.: (1) the sensibility to active, (2) to
passive movements, (3) the perception of position, and (4) the
perception of weight and resistance. He then states that in
all cases of ataxia in which the sensibility had been tested the
examination had been imperfect in some detail; he pointed out
that, for example, in the otherwise admirably conducted ob-
servations of Friedreich, the examination of the sensibility to
movement had been omitted. According to his view, there-
fore, it is only necessary to perfect the examination of the
sensibility in order to come to the conclusion that sensory dis-
turbances are responsible for the ataxia.

When one considers that we are ignorant of the origin of
the normal co-ordination, and remembers that it is not congeni-
tal but must be learned by practice, in which controlling and
correcting influences, which arise from the periphery, come
into play, it is not difficult to agree with Strümpell, who con-
siders that the ataxia takes its origin from the disappearance
or insufficiency of those regulating influences, because "the
possibility of successfully transferring them to the motor ap-
paratus is removed" (Nervenkrankheiten, Aufl. iv, pages 209,
210). We should then have to regard the gray substance and
the ganglionic cells as the place where this transfer probably
occurs. Which of the theories above mentioned will at last
be recognized as the correct one, and whether or not other
factors, which have not yet been considered, play a part in the
production of the ataxia, it is at present impossible to state.

Later on in the course of tabes there is a diminution in the

actual strength of the voluntary muscles, particularly in those of the lower extremities. This first manifests itself by weariness on walking, which gradually increases, and finally ends in total paralysis (paraplegia). The patient first notices that he has to rest in the course of walks which he previously was in the habit of taking without any feeling of fatigue, that it takes him much longer than formerly to cover a particular distance, and that he is in general unable to take the exercise to which he was formerly accustomed. As the disease advances, the power of locomotion becomes more and more diminished, and the patient is only just able to drag his legs along, and

Fig. 164.—SPECIMEN OF HANDWRITING IN A CASE OF TREMOR IN TABES (personal observation).

at last, becoming unable to move at all or even stand without help, is obliged to spend the rest of his life in the invalid's chair.

Signs of motor irritation are rare and are limited to paroxysmal twitchings in the fingers and toes; sometimes, however, involuntary movements occur in the limbs which the patient has absolutely no intention of moving. Stintzing (Centralb. f. Nervenheilk., 1886, 9, 3), for example, observed an involuntary flexion of the hip joint when the patient coughed. Similar associated movements in the fingers or toes have been described by Strümpell (Neurol. Centralblatt, 1887, vi, 1) and Oppenheim (Sitzung der Charité-Gesellschaft, 20 März, 1884).

The athetoid and choreiform movements described by

Andry (Revue de méd., 1887, 1), sometimes found in tabetics, are to be regarded as due to simultaneous disease of the lateral columns, and accordingly rather as complications. We must regard the tremor as one of the signs of motor irritation, al-though we are at present unable to localize its anatomical seat. This symptom is sometimes observed either in the initial stage or in the further course of the disease.   If the upper extremities become affected by it, the handwriting is altered, in the man-ner represented in Fig. 164.

The disturbances of sensibility in tabes are either experi-enced subjectively by the patient, or can only be discov-ered by an objective examination.   Their number is exceed-ingly large, and it is safe to say that in almost every case some interesting observation of this character may be made. Symp-toms of irritation alternate with those of paralysis, and one also meets with other different disturbances of sensation which belong to neither of these groups, and which are more variable in tabes than in any other affection.

Among the subjective symptoms we shall consider first the symptoms of irritation, more particularly the pains, which in the life of tabetics play such an important part.   They, too, are of a changeable nature, and vary considerably in their situation and intensity.   In the first place we desire to direct attention to the muscular pains, which, if they do occur at all, occur very early in the course of the disease, and affect sometimes the shoulders and sometimes the legs, and recall the well-known muscular pains which follow marked exer-tion in the gymnasium, mountain climbing, rowing, etc.   As a rule, it is true, they are not very intense, but when they come on suddenly, without any appreciable cause, the patient is obliged to remain perfectly quiet for several hours, for every motion is difficult to him, and if he persists in his attempts, movement becomes impossible on account of the feeling of weakness and fatigue which at last overcomes him.   Pitres calls these pains " crises de courbature musculaire " (Progr. méd., 1884, xii, 28), and considers that they are precursors of tabes.

We must separate from these the nervous pains of tabetics which are dependent upon irritation of the posterior roots. They are usually situated in the lower extremities, and manifest themselves either as dull, boring sensations, or as sharp pains which last for hours and then disappear for a time ; they may

also be felt in the back and sacral region, and for years be attributed to rheumatism, lumbago, etc. As long as only these pains exist, the life of the patient is bearable, although it may be marred and his occupation interfered with, but there is a second class of nervous pains which, appearing and disappearing like lightning, are known as shooting or lancinating pains, "*douleurs fulgurantes.*" It is these that make the existence of the tabetic most miserable, and make him wish that he were dead; it is these, again, that can reach an intensity which causes the most resolute sufferer to lose his energy, and converts him into a complaining and whining weakling. They also occur paroxysmally, and may continue for minutes, hours, or even days, and then disappear for variable periods, sometimes for months. In many cases they recur often, sometimes every week, but they then usually only last for a few moments.

In some cases, in connection with these attacks, cutaneous ecchymoses may develop, which are to be noted in the portions of the body subjected to the pain, and may attain a considerable size, so that one who does not know their significance, on examining the patient, may come to the conclusion that he had been injured by a blow or a fall. In still rarer instances swellings have been observed instead of the ecchymoses, which in the same manner as the latter disappear in a few days.

Along with these pains the patient may suffer with hyperæsthesias of the skin to such an extent that in certain parts of the body—very frequently, for instance, on the back—he can not bear the slightest pressure, and even his clothes will be a source of annoyance to him. These cutaneous hyperæsthesias may persist for months unchanged without being affected in the least by the paroxysmal pains.

Among the symptoms of sensory irritation the so-called girdle sensation may also be reckoned. This likewise occurs paroxysmally, at which times the patient experiences a feeling as if a belt were being drawn around his chest and abdomen, which interferes with his breathing.

Manifestations of sensory paralysis may also be subjectively perceived by the patient. Not infrequently he will say that he does not feel the contact of the clothing on certain portions of the body, or that the soles of the feet are without sensation. In a case under my observation the patient complained of a widespread loss of sensation in the perineal region, which on

objective examination proved to be anæsthetic as well as the inner surfaces of both thighs. To the anæsthesias, which are particularly unpleasant to the patient, belong those affecting the mucous membranes—as, for example, that of the rectum—owing to which the bowel may empty itself without the patient being conscious of it. Again, there are anæsthesias of the mucous membrane of the sexual organs—as of the vagina, for example—owing to which the pleasurable sensations attending coitus are either absent or greatly diminished.

Among the perverted sensations which are experienced subjectively may be mentioned the alterations of feeling in the soles of the feet, owing to which it appears to the patient that he is not walking upon solid ground, but rather upon a soft, yielding surface, such as moss, cotton, etc. To these may be added the sensation as of ants crawling over the skin, a feeling of numbness, which usually appears in the lower extremities, but sometimes also in the hands. In the latter case it may become impossible for the person to write, sew, etc., in spite of the fact that he may be suffering from no disturbance of motility whatsoever.

Many anomalies of sensation in tabes can only be discovered by means of objective examination. They constitute the second group of sensory disturbances to which we referred above.

We would here insist upon the necessity of making the examination as carefully as possible, and of remembering in the first place that when the patient is repeatedly examined he ceases to give us his attention and makes careless answers to the inquiries made of him, and in the second place that there are certain sensations, the so-called spontaneous sensations, which the patient experiences without any external irritation whatever. Rosenbach (Deutsch. med. Wochenschr., 1889, 13) holds that accumulations of weak sensory stimuli occur, the intervals between which vary according to the strength of the stimuli and the better or worse condition of the patient. If one remembers this and the fact that the so-called after-sensations must also be taken into account when making the test, one will be able to avoid gross errors. B. Stern (Arch. f. Psych. und Nervenkrankheiten, 1886, xvii, 2) has not been able to confirm the statement of Belmont (Gaz. méd., 1877, 19) that points of predilection exist for the disturbances of sensation in tabetics, as, for example, in the soles of the feet, the area about

the malleoli, and the lower extremities in general. Were it true, it might constitute a new source of error in the examination of the anomalies of sensation. The methods of examination are as simple as possible, and the necessary instruments are an induction apparatus, Weber's æsthesiometer, needles, mounted brushes, and test tubes filled with hot and cold water. With these one is able in most cases to obtain all the necessary information.

Among the symptoms of irritation, hyperæsthesias, as we stated above, are not of very frequent occurrence, but when they do occur they can very easily be recognized. They are frequently quite transient, so that a point, which yesterday was sensitive to the slightest touch, presents to-day a perfectly normal condition. The exaggerated sensitiveness is probably always confined to the perception of pain, but is not found associated with the other qualities of sensation. We recognize another symptom of irritation in the so-called double perception of painful impressions, polyæsthesia (Fischer), by which we mean that from one external irritation, as the prick of a needle, the patient experiences two painful sensations in succession.

In the objective examination of the sensibility the symptoms of paralysis play, without doubt, the more important *rôle.* In the first place there are the anæsthesias, which may affect all qualities of sensation, the sense of pain, touch, and temperature. The most interesting is an analgesia, to which Berger first directed attention, who demonstrated that while the patients reacted normally to slight stimuli, they scarcely did so at all to stronger ones. We must consider it as an anomalous analgesia, when a patient experiences only one kind of pain in response to the most varied kinds of painful stimuli. It sometimes happens that the tabetic can not tell the difference between the action of the thermo-cautery, the simple prick of a needle, or a violent pinch, and designates the pain produced by these various agents as simply a burning one. The painful sensation on electrical stimulation may also become abolished, so that we can apply the strongest currents or the faradic brush to the most sensitive parts, such as the inner surfaces of the thighs, the perinæum, or the scrotum, and the patient not give the slightest evidence of pain.

Lastly, delayed sensation is to be considered as a symptom of a paralytic nature. In these cases, when the patient is

pricked with a needle, he does not experience pain until one, two, or three seconds later. The delay of perception may vary for the different qualities of sensation—for example, for touch and pain—as Osthoff and Remak have pointed out.

We must attribute to disturbances of the muscular sense, which we alluded to in discussing the cause of ataxia, the fact that the patient with his eyes closed is unable to state accurately in what position his extremities are, and if one, for instance, changes the position of a limb, he is not at all certain into what position it has been put. He is unable to estimate the weight of an object placed in his hands, and so forth. All these conditions are to be remembered when one is testing the muscular sense, and at the examination one will have to ascertain what is the minimum change of position which can still be recognized by the patient.

Of the disturbances of the reflexes, those of the skin interest us less than those of the tendon reflexes; of the latter, the patellar reflex is the most important, the anatomical localiza-

Fig. 165.—Two Cases of Tabes. (After Westphal.)

A. The lines *hh* show the limits of the so-called "root zone." The degeneration is progressing from within toward them, but only reaches the border line. The patellar reflexes were retained until death.

B. The degeneration is progressing from within outward, and has extended far into the "root zone." The patellar reflexes were lost five years before death.

tion for which is in the so-called root zone (Westphal). This zone is situated at the junction of the lower dorsal portion of the cord with the lumbar enlargement at the level of exit of the second, third, and fourth lumbar nerves (cf, page 420), and constitutes the area which the roots entering to the median side of the posterior horn must traverse in order to reach the substantia gelatinosa of the posterior horn. If this field is degenerated, then the patellar tendon reflex disappears, but if it

is normal, the reflex is preserved (cf. Fig. 165, A and B). The rare cases in which it remains preserved on one side also confirm the localization assumed by Westphal ; at the autopsy it has been repeatedly noted (cf. Berlin. klin. Wochenschr., 1887, 31, page 586) that there was a degeneration of the posterior columns and of the "root zone" on the affected side, while this zone on the healthy side was intact.

The disappearance of the patellar reflex, "Westphal's sign," was formerly considered as pathognomonic of tabes, and whenever the knee jerk could not be obtained, the diagnosis was made without hesitation. This. was the standpoint taken in the earlier works of Westphal, Erb, and others, and it must be confessed that "Westphal's sign" is observed in by far the greater number of cases of tabes, and usually early in the course. However, it began to be doubted that the rule was without exceptions, and toward the end of the seventies several undoubted cases of tabes were reported (Berger, Fournier) in which the patellar reflex was retained to the end of life, and, since then, other similar cases have been added. Westphal himself pointed out that the knee phenomenon might persist with degeneration of the posterior columns (Arch. f. Psych. und Nervenkrankh., 1886, 17, 2), and precisely at this time I myself reported two such instances (Berlin. klin Wochenschr., 1886, 10). Accordingly, it is an undeniable fact, and one which, anatomically, can be readily explained, that under certain circumstances—that is, whenever the "root zone" remains free from degeneration—the patellar reflex may continue to be present during the entire course of the disease. By repeated and accurate examination, in which Jendrassik's method of reenforcement should not be forgotten, one is sometimes able to follow up the gradual disappearance of this reflex, and to observe that the time of its diminution and final disappearance may differ in the two legs—for example, the reflex may still be well marked on one side, while it has completely disappeared on the other. Among others, Goldflam has reported observations on this point (Neurol. Centralblatt, 1888, 19), and has supposed that interference with conduction, produced by pathological changes in the peripheral nerves, may also be the cause. The patellar reflex which has once disappeared in the course of tabes can never reappear, since destruction of the corresponding portions of the cord has taken place, but in traumatic neuroses this may very well happen, and in doubtful instances it

may become an important point in the differential diagnosis. The patellar reflex can only be increased in tabes when there is a coincident degeneration of the lateral columns.

While, then, for the reasons we have given, " Westphal's sign " can not be regarded as pathognomonic, there are still others which should warn us against laying too much stress on the condition of the patellar reflex in the diagnosis of tabes. Unquestionably it may also disappear under certain circumstances in the course of other affections—as, for example, in certain diseases of the brain—if the muscular tone necessary to its production has been lost; also in neuritis, poliomyelitis, diabetes, chronic alcoholism, and in affections of the knee joint when the movements of the tendon are interfered with. When we add that it can not be demonstrated in all healthy persons—a small number being entirely without it, as Berger and others have stated—and moreover consider the fact that in old age and in conditions of marked nervous exhaustion it may entirely disappear without any apparent reason, perhaps from a diminished tone in the muscles, we shall have sufficient grounds for not overestimating its significance, important as it may still be for the recognition of tabetic processes. The measurements of its strength, which have lately been made a good deal of, may for the present be omitted in practice without disadvantage for the diagnosis.

The reflex centres for the functions of the bladder, rectum, and sexual apparatus, which are situated in the lumbar portion of the cord, are naturally also greatly disturbed in the course of tabes. The reflex processes, which come into action here, are but little understood, but their pathological condition has been studied with great care. Much attention has been directed toward the bladder troubles of tabetics, and attempts have been made to distinguish between the different kinds of affections. They are motor or sensory, or both, according as only the one or the other or both centres have been destroyed by the degenerative process in the cord.

Among the motor disturbances there are symptoms of irritation as well as of paralysis, which may affect equally the sphincter and the detrusor, so that the will may have but little influence over them, or finally none at all. According as one or the other condition is the more prominent, the complaints of the patient differ; sometimes he is obliged to strain for a long time before the bladder will begin to empty itself, and

even then the stream is often interrupted; sometimes he is unable to urinate at all in the erect posture, but must squat down or sit on the closet to bring the abdominal muscles into action in order to expel even a few drops of urine, and the act of micturition may take so long, that the patient feels ashamed to use the public conveniences. In other cases, where there is not only paresis of the detrusor, but at the same time a spasm of the sphincter, the patient can not urinate at all, and the retention must be relieved by means of the catheter; in other instances, again, where there is a paresis of the sphincter, he has to urinate very frequently. Long before the bladder is full—every hour or two—he feels an irresistible desire to empty it, which he must satisfy or run the risk of an involuntary passage of urine. He is unwilling to go upon railroad journeys, to go into society, to lectures, or to the theatre, for fear that he will not be able to reach a convenient place in time where he can urinate in peace. Paresis of the sphincter is often a reason why the patient sleeps poorly, because he has to get up so often to urinate, and if he sleeps soundly he does not appreciate the calls of Nature, and will pass his urine in bed. When he coughs or sneezes the under-garments are moistened with urine, and, despite his utmost efforts, he is unable to prevent it. In the more marked degrees of weakness of the sphincter there is an involuntary trickling or an occasional discharge of urine, which the patient is unable to predict; this necessitates the constant wearing of some sort of receptacle; otherwise the patient is surrounded by such an ammoniacal odor that the incontinence is recognizable without any examination. If there is a combination of retention and incontinence, it manifests itself in the following manner: After long straining the urine is passed in a moderately strong stream, but this suddenly ceases, and can only be started again after renewed efforts. Sometimes, after the patient has strained in vain for a long time and has given it up in despair, the urine is passed involuntarily. These and many other facts of the same description are only to be discovered after careful and repeated questionings and examinations.

Sensory disturbances may manifest themselves (1) by more or less intense pain before and during the act of micturition, which may distress the patient greatly and make him dread to relieve his bladder (the "*crises vésicales*" of Charcot). The pain may be situated either in the hypogastric region or ex-

tend down into the urethra (*crises vésico-uréthrales*). Painful strangury, forcing the patient to urinate every half hour, when he only passes a few drops, has also been observed. On the other hand, (2) there may be a diminution in sensibility, so that, in consequence of the anæsthesia of the mucous membrane of the bladder and urethra, the flow of urine is not noticed, and the patients, especially when there is a weakness of the sphincter at the same time, do not know whether they are

Fig. 166.—A Case of Charcot's Joint in a Tabetic (personal observation).

urinating or not, and only become aware of the fact when they feel the chilly sensation proceeding from the damp clothes. A rather rare manifestation, which may be observed after violent bladder crises, is the appearance of hæmaturia, which must be attributed to capillary hæmorrhages into the bladder or urethra; the bloody character of the urine may be a source of new anxiety and worry to the unfortunate patient, already

greatly broken down by the agonizing pains. These hæmor-
rhages may be considered as analogous to the ecchymoses in
the skin occurring after the intense lancinating pains, which
we mentioned above on page 601.

The most troublesome rectal symptom is the very obsti-
nate constipation. Incontinentia alvi and anæsthesia of the
rectal mucous membrane, in consequence of which the patient
is not aware of the act of defecation, and so soils himself un-
consciously, are among the rarer occurrences.

The centre for the sexual functions, the seat of which is
also in the lumbar enlargement, is not only under the control
of reflex, but also of cerebral influences. If the path coming
from the psychical centres is interrupted, the performance of
the function is faulty; if the path from the inhibitory centres is
disturbed, the sexual reflex activity is increased and priapism
may occur. Pitres (Progr. méd., 1884, xii, 37), under the name
of "*crises clitoridiennes,*" has described in women conditions
which consisted of periods of voluptuous excitement accom-
panied by secretion, analogous to the violent erections and
spermatorrhœa found in men in the initial stages of tabes.
Such cases are, however, at least in Germany, exceptional.
Not infrequently tabetics have been known to preserve their
virility, and even after the beginning of the disease to beget
one or even several healthy children. Only later does the
sexual power, and with it the desire, become diminished, and
coitus lose its charm, so that it is undertaken more rarely, the
act being sometimes incomplete. A normal condition of the
nerves necessary for the erection of the penis, associated with
a paralysis of those going to the ejaculator seminis, so that
while coitus and orgasm are normal, the semen is not emitted
till later, and then very slowly—a condition which Bernhardt
has observed after injury (Deutsch. med. Zeit., 1888, 48)—has
been known to occur also in the course of tabes.

The vaso-motor and trophic centres in most cases are not
affected. In the majority of instances, symptoms of this char-
acter are entirely absent during the whole course. In some,
however, peculiar symptoms attract our attention, as, for ex-
ample, a local hyperidrosis, which Ollivier (Gaz. hebdom.,
September 7, 1883, xxx, 36), Raymond and Arthaud (Revue de
méd., 1884, 4, 5), and others have observed on the hands and
feet. In a case of tabes we have also seen the sweat secretion
on the hands so increased that we were able to note the forma-

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tion of small drops and watch them unite to form a steady dripping. In another case there was unilateral sweating, the hyperidrosis appearing after every meal on the left half of the head, face, and neck. I do not care to risk an opinion as to how far an assumption of an affection of the sympathetic would here be justifiable.

Greater practical importance must be attributed to the changes which are observed in the nails and teeth of those affected with tabes. The nails are either deformed, becoming twisted or marked by deep furrows, or fall out entirely from the fingers as well as from the toes, as Joffroy (L'Union, 1882, 106), Bonieux (Thèse de Paris, 1883, No. 237), Hay-Mar girandière (Thèse de Paris, 1883, No. 75), and others have observed. The loss of the nails (" *la chute des ongles* ") is not rare in tabes, and is in some cases to be attributed to the temporary cessation of growth of the nail matrix. In others an ecchymosis under the nail may be the exciting cause. Under certain circumstances the nail of the great toe falls off altogether, without pain, with only a slight itching sensation, and the newly formed nail, which is often rough and irregular, soon shares the fate of its predecessor.

It is occasionally observed that the teeth become loosened without any pain and fall out without the appearance of any symptoms of inflammation, the tooth itself being intact. This arises from some disturbance in the nutrition of the jaw, a rarefying ostitis which is connected with a lesion of the nucleus of the trigeminus (Vallin and Demange). In this way the patient may lose all his teeth in a few months. It is very interesting to note that this may be connected with laryngeal crises, a fact which would indicate that there may be some truth in the view advanced by Buzzard (British Med. Journal, February 19, 1886), according to which the centre for bone nutrition lies quite close to that of the vagus.

The so-called *mal perforant du pied* (perforating ulcer), which begins with the formation of a bleb and leads to abscess formation and necrosis of the tendinous and bony portions of the feet, is due to some trophic disturbance, and may become a source of great discomfort to the patient.

Affections of the bones and joints, which are also of trophic origin, belong to the more frequent complications of tabes. The bones become extraordinarily brittle and fractures frequently occur without pain, and one could almost say without

the knowledge of the patient. The seat of such fractures is most commonly in the femur, and, more especially in old women, in the neck of that bone. This remarkable fragility is of especial moment when it occurs, as it sometimes does, in the bones of the spinal column, and particularly in its lumbar portion, and gives rise to spondylolisthesis without it being possible to decide whether or not the cartilages and ligaments were first affected and the disease of the bones was only secondary (Kroenig, Zeitschr. f. klin. Med., 1888, xiv, 1, 2).

Among the joint affections which are not essentially different from those produced by arthritis deformans, the "*arthro-*

Fig. 167. Fig. 168.

Fig. 167.—Erosion of the Head of the Humerus in Tabes Dorsalis. Fig. 168.—Normal Humerus. (After Charcot.)

pathie des ataxiques" or "Charcot's joint," because it was first described by him, deserves particular mention. According to his description, there develops in the course of one night, without any appreciable cause and without pain or febrile movement, a swelling of a joint—for instance, the knee, shoulder, elbow, or hip. In the course of a few days there is noted a collection of fluid in the joint and in the periarticular bursæ, and on puncture a lemon-yellowish transparent serum can be withdrawn. In one or two weeks later one is able to make out more or less well-marked crepitation, due to changes in the joint surfaces. The joint becomes extraordinarily movable and luxations frequently occur, especially when the ends of the bone are worn away (Figs. 167 and 168).

Occasionally the tarsus is affected by the process. In such cases a marked swelling of the foot occurs in a relatively short
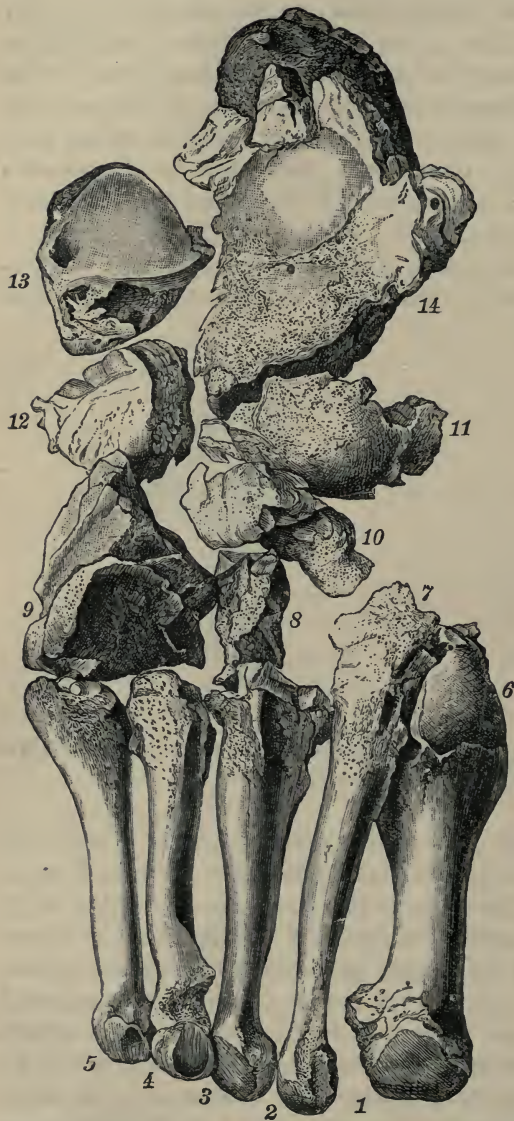
Fig. 169.—SKELETON OF A TABETIC FOOT. (After CHAR-COT.) (The original is in the pathological museum of Charcot's department in the Salpêtrière in Paris.) 1–5, metatarsal bones. 6, internal cuneiform bone. 7, middle cuneiform bone. 8, fragment of the external cuneiform bone. 9, cuboid bone. 10 and 11, fragments of the scaphoid bone. 12 and 13, the astragalus. 14, the os calcaneum.

time, the joints become affected in the way stated above, and at the post-mortem examination the tarsal bones are found to be altered in the manner represented in Fig. 169 ("tabetic foot").

The real cause of the affection is not yet known. While Charcot considered it due to an atrophy of the anterior ganglionic cells in the cord, Virchow pointed out that it might be due to a state of lowered nutrition of the bone in consequence of nerve influence. Oppenheim and Siemerling demonstrated a degeneration in the peripheral nerves, and according to Volkmann the analgesia produced by tabes creates a predisposition to the occurrence of the joint affection which he attributes to disturbances in the cartilages. Rotter divides the cases into three groups — true arthritides deformantes, primary

fractures of the joints, and a third class in which there are most pronounced changes, but in which we are unable to determine whether they are due to an arthritis or a primary fracture.

We may add that arthrectomy has lately been performed several times for tabetic affections of the knee joint, and has been followed by success (Wolff, Sitzung der Berliner med. Gesellsch., 7. März, 1888, Deutsche med. Ztg., 1888, 22, page 268).

For the last few years only we have known that the peripheral nerves play a large and important part in tabes; previous to this Türck and later Friedreich had reported alterations in the mixed nerves, but we were ignorant of their character until the publication of the work of Westphal (1878), which was soon followed by other articles, among them those of Déjerine, Pitres and Vaillard, Oppenheim and Siemerling, Sakaky and Pierret. The results of their work showed that the peripheral nerves suffer a parenchymatous degeneration, a destructive process, which, being associated with an increase of the perineurium, a proliferation of the nuclei, and extensive connective-tissue formation, leads to a final atrophy of the nerve elements. This peripheral neuritis is not necessarily followed by marked symptoms, but, according to our views, it is the main factor in the production of the analgesias which are often observed so early in the course of tabes, and to which O. Berger has already directed attention. Under certain circumstances this neuritis may produce deformities; thus if it involve the nerves which supply the muscles of the plantar surface of the foot these latter atrophy. The muscles concerned are those of the inner surface of the foot affecting the great toe, those of the outer surface to the little toe, the flexor brevis communis, and the interossei; the plantar aponeurosis retracts, and the toes become flexed and immovable (Fig. 170).

If larger nerves be affected by the process, the symptoms, which are characteristic of neuritis, and which have been described on page 386, make their appearance. They are chiefly pains, motor disturbances, and muscular atrophies. To this class belong the musculo-spiral paralyses caused by tabes, described by Strümpell (Berl. klin. Wochenschr., 1886, xxiii, 37), lesions of the median, described by Remak (ibid., 1887, xxiv, 26), and, lastly, lesions of the peroneus longus, as described by Joffroy (Gaz. hebdom., 1883, xxxii, 48). Lately Déjerine has

described a widespread muscular atrophy in tabetics, which has its origin in a peripheral neuritis (Neurite motrice periphé-rique des ataxiques, Revue de méd., 1889, 2). The observations of Remak seem to indicate that the muscles which are sub-jected to an unusual strain in the patient's occupation are par-ticularly prone to become atrophied. In confirmation of this I can add two cases of my own: (1) In a cigar-maker, who ex-erted particularly the first three fingers of the right hand in making the cigar-tips, atrophy developed in the muscles of

Fig. 170.—PLANTAR FLEXION OF THE TOES IN THE COURSE OF TABES (personal ob-servation).

the ball of the thumb supplied by the median. (2) A dentist with tabes, who overexerted the musculature of the hand in filling teeth and in other manipulations, came under my obser-vation on account of an atrophy of the hypothenar muscles supplied by the ulnar. Similar cases are not uncommon.

It is not at all rare in the course of tabes for the peripheral nerves to be attacked by neuralgias; the sciatic nerve calls for first mention, as it is usually affected early in the disease and very severely. We have already stated on page 370 that double sciatica is more particularly a frequent accompaniment of tabes. Branches of the pudic nerve may also be affected, and often recto-vesical neuralgia may be a source of great trouble (Neftel, Arch. f. Psych. und Nervenkrankheiten, 1880, 10); in this the patients complain of a painful burning sensa-tion in the rectum after each defecation, which is often fol-

lowed by marked depression of spirits, and the longer the interval between the acts of defecation and the firmer the consistence of the stool, the more intense becomes the suffering. After all, it is not easy to distinguish the peripheral from the above-described central affection, which may run a similar course.

We will now attempt to say something as to the relative frequency of the symptoms and the time of their occurrence, but, of course, such statements can not lay claim to accuracy, and can only serve to give an approximate idea concerning the points in question.

Among the most frequent symptoms belonging to the brain, are, as we have already shown, lesions of the cranial nerves, and particularly of the oculomotorius, by which transient diplopia and irregularities in the condition of the pupils (anisocoria, myosis) are produced; next come lesions of the abducens. Almost as frequently will one recognize disturbances of the vagus, among which the gastric crises deserve particular mention. Among the spinal symptoms belonging to this category the first to be mentioned are the manifold disturbances of sensibility, among them cutaneous analgesias, particularly in the lower extremities, then, the paræsthesias and the lancinating pains which occur more particularly in the legs; how far these symptoms in a given case are due to disease (irritation) of the posterior spinal roots or to lesions of the peripheral nerves can only be determined by microscopic examination. At all events, degeneration of the peripheral nerves in the most varied cutaneous areas is to be classed among the regular occurrences in tabes. The disappearance of the patellar reflex and some form of the various bladder troubles are almost constant accompaniments of the affection, and these, taken in connection with the symptoms just mentioned, must be considered as the foundation for the diagnosis.

Lesions, particularly atrophy, of the optic nerve, symptoms of irritation and paralysis in the domain of the fifth nerve, and ataxia of the lower extremities, are frequent but less regular occurrences.

Less frequently met with are the laryngeal crises, due to lesions of the vagus, and affections of the nerves of taste and of the accessorius; the same may be said of the psychoses, hemiplegias, and attacks of epilepsy observed in the course of

tabes. Certain disturbances of sensibility, the so-called rectal crises, cutaneous hyperæsthesias, neuralgias of the peripheral spinal nerves, paraplegia of the legs, the tremor, and disturbances in the sexual functions also belong to this category. The trophic disturbances, the muscular atrophy, the falling out of the nails and hair, the "*mal perforant du pied*," and Charcot's disease of the joints are also comparatively rare.

To the symptoms which occur only seldom, one might almost say exceptionally, belong those referable to the hypoglossal, the auditory, and the facial nerves; among the motor disturbances of spinal origin, the so-called associated movements and ataxia of the upper extremities, among the sensory disturbances, the so-called polyæsthesias, double sensations, and delayed sensations belong to this class; marked diminution in the muscular strength is also exceptional.

As to the time at which these various symptoms severally arise, it is even more difficult to give reliable data, since there exists no uniformity; still one can state with some amount of certainty that next to a feeling of slight weariness, particularly in the legs, the lesions of the oculo-motor and abducens are often the first to make their appearance; the disturbances of sensibility, particularly analgesia and paræsthesia, as a rule also occur early, while lancinating pains make their appearance at a later period. The gastric crises are observed relatively early, and bladder troubles are among the more frequent occurrences before the disease has advanced very far. The disappearance of the patellar reflex, as it usually constitutes one of the initial symptoms of the disease, plays an important part in the diagnosis, as we have already shown. Pronounced motor disturbances, particularly ataxia of the lower extremities, are often not observed until later in the disease, often only after years; and paraplegia of the legs, when it occurs at all, characterizes the last stages of the disease. Optic atrophy sometimes makes its appearance relatively early; in other instances it occurs only at a late period and comes on very gradually. For the time of its occurrence no definite rules can be laid down. Hemiplegias, epileptic attacks, and psychical disturbances, if they occur at all, manifest themselves sometimes earlier, sometimes later. As far as our own observations go, the trophic disturbances mentioned above, particularly the muscular atrophies and Charcot's joint affection, usually belong to the later stages.

The course of tabes is rarely markedly influenced by complications, and this will be readily understood when we consider that the affection presents such a manifold variety of symptoms, that it is almost impossible to add to it new ones, resulting from another and independent disease. On the other hand, one can readily imagine that a complication may exist and yet escape recognition. This would apply, for example, to hysteria, which may give rise to symptoms which, under certain circumstances, can not be distinguished at all from those of the disease under discussion. The same is true of syphilis, the nervous symptoms of which may be identical with those of tabes ; and, lastly, it may be applied to the progressive paralysis of the insane, as the manifestations of both diseases may be inextricably involved with each other. It is less difficult in the case of diabetes, which has also been observed as a complication ; yet the diabetic and tabetic symptoms may completely obscure each other, so that it becomes impossible to determine which are due to the one and which are due to the other affection. Should, however, the urine contain a large amount of sugar, the task becomes less complicated, for glycosuria is not frequent in tabes. Moreover, as Möbius holds, the Argyll-Robertson pupil should make us decide for tabes, as it is absent in diabetes (cf. Fischer, Centralbl. f. Nervenheilk., 1886, ix, 18).

If we consider tabes as an affection of the entire nervous system, we shall not be surprised if now and again other lesions in the cord are found to be coincident with the tabetic process, as, for example, lesions in the crossed pyramidal tracts, which Eulenburg has observed and reported (Deutsche med. Wochenschr., 1887, xiii, 35). The degeneration in the posterior columns, it is true, is the most common, but this would not exclude a simultaneous affection in other systems of fibres.

Some German and French writers have assumed aortic insufficiency to be a frequent complication of tabes, but the work of Leyden (Centralbl. f. klin. Med., 1887, viii, 1) and his lucid statements at the session of the Verein für innere Medicin in Berlin on the 29th of October, 1888, have shown this connection to be a mere coincidence (Deutsche med. Zeitung, 1888–'89).

Course.—About the general course of the disease the following remarks will hold good in a large number of cases : A middle-aged person who has become infected with syphilis some years previously, usually from eight to fifteen years be-

fore, begins to complain of slight fatigue on walking and occasional pains in the lower extremities. In spite of all treatment the pains continue to be troublesome, and occasionally become so severe that they disturb the patient's rest at night or even render sleep impossible. At the same time it appears to him that his vision is becoming affected, and he complains particularly that he sees double, and in consequence suffers from vertigo. The diplopia may last only for a few moments at a time. The vertigo, which at the onset of the trouble was insignificant, becomes more and more pronounced, more especially in the dark, so much so that it is almost impossible for the patient to pass through a dark room without help. He also discovers that he staggers or falls to one side in the morning when in washing he covers his face with the towel, and only regains his equilibrium when his eyes are free again. Finally, he complains that he is obliged to pass water more often than usual, and that he consumes more time and must exert himself more when urinating than previously. The objective examination shows that there is widespread anæsthesia, particularly analgesic areas, about the lower extremities, and a loss of the patellar reflex. He may be inconvenienced in this way for years without his condition becoming serious. He suffers more or less all the time, sometimes quite severely, but, on the whole, his existence is quite bearable. The state of his mind is hopeful, for the daily occupation has not yet been interfered with by the disease.

The aspect of affairs is quite different when the patient suffers from gastric disturbances. The appetite becomes poor, and occasionally—sometimes for weeks at a time—there is morning vomiting, which is quite profuse and occurs as soon as the patient awakes, when, without effort, watery, slimy masses are discharged. After lasting for a longer or shorter time this ceases, probably only to return later on. The appearance of the patient, which was previously natural, now becomes altered for the worse. The skin becomes yellow and wrinkled, and his friends and acquaintances, who have not seen him for some time, begin to inquire about his health. At the same time a new symptom makes its appearance, and he notices that his gait is becoming uncertain and that in walking he must invoke his eyes to aid his legs, which, instead of carrying out the movements he intends, are thrown out in a peculiar aimless manner, so that if he be not led or supported he runs the risk of

tumbling down. This trouble in walking, which is associated, perhaps, with occasional gastric and more rarely with laryngeal crises, may likewise continue for years; but if the ataxia implicate the upper extremities, as happens in a small proportion of the cases, it may so interfere with the patient's occupation that he may be unable to continue it. In the meanwhile the bladder symptoms become more prominent and are aggravated to such an extent that it becomes necessary for the patient to wear some sort of receptacle, while the marked contraction or inequality of the pupils is apparent even to the layman.

Gradually another change for the worse in the gait comes on. The legs, which, although thrown out in the characteristic manner, in other respects performed their duty and even enabled the patient to cover considerable distances, begin to be fatigued on the slightest exertion; they become heavier and heavier, and it becomes more and more difficult, and at last needs the greatest effort, to walk at all. The legs are so weak that they are no longer able to support their owner, who is forced to take to the invalid's chair, and in this he ends an existence, the last years of which are as wretched as could be imagined, especially if atrophy of the optic nerve has robbed him of sight and the lancinating pains make his days and nights miserable. When the disease progresses in this or a similar manner its duration varies from ten, fifteen, even to twenty years or more. It can, however, be considerably shorter. I have seen cases in which only from three to five months elapsed between the beginning of the affection, from the first appearance of the disturbances in the movements of convergence of the eyes, to the appearance of well-marked paralysis of the legs.

On the other hand, there are cases in which the course may extend over a space of thirty or more years; in these cases paralytic symptoms may not come on at all, and the ataxia continue to the end. There are tabetics who during their entire illness are hardly prevented at all from carrying on their work; they are always able to be up and about, and it appears as if the different symptoms never attained their full development in these cases. These are the so-called "*formes frustes*" of the French, analogous to those with which we have already become acquainted in Graves' disease and in multiple sclerosis. Again, in other cases, tabes sets in with brusque symptoms, such as apoplectiform attacks, disturbances of speech, and

lesions of the optic nerve, and then pursues a mild course for a long period—violent symptoms, such as laryngeal crises, intense neuralgias, etc., only occurring occasionally; these are the so-called atypical forms of the authors.

From what has been said it is evident how difficult it is to make a positive statement concerning the general course of the affection. Scarcely one case follows the same course as another, and it often requires a great amount of caution and experience to enable one to take a correct view of all that occurs.

Just as much uncertainty exists about the prognosis, which is influenced by various factors. One most important question is, of how long standing is the disease, for in recent cases in which there are no other symptoms than disturbances of sensibility and absence of the knee jerks, and the course of which has not as yet exceeded three or four months, and in which there is no ataxia, the prognosis is not at all unfavorable, and the disease is under some conditions curable. Advanced cases of tabes in which there are numerous spinal and cerebral symptoms offer a much more serious prognosis, but even here the possibility of cure is not excluded, though the highest percentage of recoveries is estimated at one per cent (Eulenburg). Of course, one can not expect that the anatomical changes will disappear, and at the autopsy a widespread degeneration of the posterior columns has been found in cases in which during life all symptoms had practically disappeared. In the majority of the so-called recoveries from tabes one is led to believe that there was a mistake in the diagnosis, and that these were cases of chronic nicotine poisoning, peripheral neuritis, hysteria, neurasthenia, etc. The prognosis of old cases with paraplegia of the legs, paralysis of the bladder, and so forth, is altogether unfavorable, and any attempts at cure are not only useless, but may even interfere with the comfort of the patient.

It is a matter of indifference, so far as the prognosis is concerned, whether one is able to demonstrate that the patient has at one time or other been infected with syphilis or not; a so-called specific or luetic tabes, especially when the infection has taken place ten or twenty years previously, does not afford a better outlook than the more rare idiopathic affection.

It is clear, then, that one must be very cautious in predicting the duration of the disease; one can not say definitely how many years a tabetic patient has to live, and just as little should

one attempt to make a positive statement as to how long the patient will be able to work. The condition may remain quite endurable for months or even years, and the outlook may appear quite hopeful, particularly in regard to the capacity for work, and yet suddenly a marked change may take place; pronounced ataxia, cerebral symptoms, or the like may manifest themselves, which render the patient incapable of following any occupation. The more cases one sees, the more cautious does one become in giving a prognosis, and the more distrustful of the reports of so-called cures—at least when old cases are concerned.

Diagnosis.—As one can readily see from what has been said, the diagnosis of tabes is sometimes one of the simplest possible tasks for the physician; in other instances it can not be made with certainty for a long time. Thus it may under certain circumstances be very difficult to differentiate between the disease under consideration and complicated cases of syphilis of the brain and spinal cord, diabetes, or hysteria. It seems perfectly possible for one to consider a severe case of neurasthenia for a long time as one of tabes, but the further course and final success of therapeutic measures will demonstrate the error. When in the course of tabes the sensory and bladder disturbances are only slightly marked, there may be question of the existence of a chronic anterior poliomyelitis, but usually the lancinating pains, the paræsthesias, the affection of the eye muscles, and the mere fact that bladder symptoms exist at all, afford sufficient grounds on which to base a diagnosis. In diseases of the vertebral column, in the course of which lancinating pains, "Westphal's sign," and bladder symptoms may be found, an examination will reveal that the vertebral column itself is affected, and the spinous processes are painful on pressure—a condition which is sufficient to settle the diagnosis. The mistake of considering a tabophobe, or a person who imagines he has tabes, as a real tabetic, can only occur when a careful examination is neglected, and the physician is afraid to adopt any energetic, psychical as well as somatic, treatment. As soon as this is instituted the tabetic symptoms will turn out to be mere hypochondriacal notions, and recovery will quickly follow.

It is of practical importance to note that the various symptoms occurring in tabes are also observed in other affections. In these cases there is much room for errors in diagnosis, the

most important of which we wish to bring to the reader's attention.

Paralysis of the eye muscles and pupillary symptoms are, as we have remarked, very common in the course of tabes. Another affection in which they also occur is multiple sclerosis. Here, however, diplopia as well as strabismus are rare, while, on the other hand, nystagmus is very frequent, and the pupillary reaction to light is preserved. Myosis occurs in this affection as well as in tabes, but whereas in the former the pupils contract still more under the influence of light, in tabes they usually remain immobile under the same circumstances.

Symptoms referable to the optic nerve—amblyopia, for instance—are also observed as the effect of different poisons (page 37). In such cases the history will be of great assistance to us in making the diagnosis. Amblyopia developing in the course of multiple sclerosis is not accompanied, as in tabes, by a contraction of the field of vision, nor does it steadily grow worse; but remissions occur, and the improvement may even last for a considerable time. It has already been shown on page 580 that the optic atrophy of multiple sclerosis differs in important points from that occurring in tabes. It should also be remembered that there is an optic atrophy in which the morbid process is confined to the optic nerve, and in which it is impossible to demonstrate any general nervous disease.

The various visceral "crises," in which tabes abounds, can likewise be produced by independent affections of the vagus. Here one must rely upon the more characteristic symptoms of tabes, particularly Westphal's and Romberg's signs. That "gastric crises" alone can not enable one to make the diagnosis is all the more to be insisted upon since Debove has recently observed them in neurasthenics (Soc. des hôp., séance 1888, xii, 28).

The motor disturbances which we find here, and of which the most important is the ataxia of the lower extremities, appear not only in the course of tabes, but also in other diseases in which one is unable sometimes to ascertain their anatomical basis. This is more especially true of the so-called functional ataxias (Gallard, Jaccoud), which develop sometimes with, sometimes without sensory disturbances, and are associated with no other symptoms. Ataxia has likewise been observed developing slowly or quickly after diphtheria (Berl. klin. Wochenschr., 1887, 49, p. 930), after quickly succeeding preg-

nancies, and in the course of diabetes; and the question must remain undecided whether it is to be considered as the expression of a severe general affection, of a faulty composition of the blood and an imperfect innervation dependent upon it, or as the result of a peripheral neuritis developing under the influence of an infectious agent. However, it can not be difficult in a given case to determine whether the ataxia is to be regarded as of spinal or tabetic, or as of functional or of infectious origin.

The lancinating pains also occur in affections of the vertebral column, e. g., in Pott's disease, when the posterior roots are irritated, but the deformity and the tenderness of the vertebræ upon pressure will make the diagnosis clear.

Other pains, following the course of various larger nerves, which can last for weeks or months without marked exacerbations, and be accompanied by paræsthesias, formication, numbness, etc., are observed not only in tabes, but also in peripheral neuritis, following, for example, the abuse of alcohol. If to these a temporary loss of the patellar reflex be added, we have the picture of what is called pseudo-tabes, and a cautious and often-repeated examination is necessary in order to make the differential diagnosis. The history and the further course of the disease, which in alcoholic neuritis may become favorable after the removal of the cause, should always be taken into consideration.

We have already pointed out on page 606 the circumstances under which Westphal's sign may be present, and we can not insist too strongly that it is an error, or at least a too hasty conclusion, to think only of tabes whenever the patellar reflex is absent. On the other hand, we must not imagine that its presence puts tabes out of the question, for the possibility of the existence of this disease is not at all excluded when the reflex is found to be normal.

Pathological Anatomy.—Considered from the pathological standpoint, tabes represents a degenerative process in which the entire nervous system takes part. The reason that we are unable in all cases to demonstrate the participation of all the nerves—that in many cases, for example, the cord seems to be the part most involved while the brain and its nerves appear less affected—lies in the fact that we have been accustomed to examine the cord with the greatest accuracy, while the brain and peripheral nerves were only considered of secondary im-

portance; and, secondly, in the fact that many cases are terminated by intercurrent diseases before the degenerative process could develop in all directions.

This degeneration, which consists principally in the death of the nerve elements and an increase of the connective tissue, presupposes a certain change in the nervous system, the nature of which we do not as yet know, and which is peculiar to the individual either as the result of hereditary influences or which has been acquired later through syphilitic infection. The congenital predisposition is not sufficient to produce an outbreak of the disease. For this some one of certain exciting causes, of which we shall speak later, is needed. On the other hand, the changes produced in the nervous system by a syphilitic infection are able of themselves to lead to the production of tabes. As to the manner in which heredity works in the production of these changes, we are not in a position even to hazard a conjecture, nor are we by any means certain of the precise mode of action of syphilis. In this latter case, however, it is, according to our idea, most probable that the changes are the result of a syphilitic affection of the blood-vessels. It is, in our opinion, less likely that a poison ("toxine"), which affects the nervous system, is developed secondarily, in which case tabes would have to be regarded as a post-syphilitic affection, just as paralysis of the soft palate is a post-diphtheritic affection (Strümpell); and it would be still harder to imagine that the syphilitic virus becomes localized in the nervous system, and, as such, later produces the disease (Rumpf). One could in the last case not help but ask how it is possible for ten, fifteen, or more years to elapse between the syphilitic infection and the appearance of the first tabetic symptoms, a circumstance which, on the other hand, could be easily explained by assuming the existence of anatomical changes which are due to a diminution in the blood supply and require a relatively long time for their development.

The degeneration begins probably always in the peripheral nerves. The terminations of the cutaneous sensory nerves may be the first to become affected. The admirable researches of Déjerine, Oppenheim, Siemerling, and others, have clearly demonstrated the participation of the peripheral nerves in the tabetic process, and there is no doubt but that they appear just as much degenerated as the posterior roots, in which the atrophy was shown to be most marked between the spinal ganglia

and the cord, while the peripheral portion was often relatively quite free (Déjerine, Compt. rend. de la Soc. de biol., 1882, page 215). The degree to which the several cutaneous nerves are attacked varies. Those of the legs are usually more affected than those of the upper extremities. No definite rule can be said to exist. Sometimes, and this often happens, the peripheral ends of certain of the cranial nerves are the first to become diseased—e. g., those of the optic, the oculo-motor, and the abducens—and then the symptoms described above appear in the initial stage. At any rate, the first symptoms develop in consequence of lesions of peripheral end organs.

The degenerative process in the cord, which occurs later, is the most prominent pathological feature at the autopsy, and formerly was considered the only, or at least the only characteristic lesion. This explains why tabes was and is still considered, by the majority of authors, as a disease of the spinal cord. According to our idea this is not true. It is rather an affection of the entire nervous system, in which the cord is not even the first part to become affected, but later is altered in such a characteristic and striking manner that we can not be surprised if the other, less marked, conditions were overlooked. Though the changes in the cord have long been recognized, the views as to their origin are still conflicting and the most varied interpretations have been put forward. We do not care to enter into an account of the controversies, but will only bring before our readers succinctly the conclusions arrived at as to the nature of the affection. It consists probably of a primary degenerative atrophy of the nerve fibres, which is followed by a secondary increase of the connective tissue. As the degeneration takes place slowly, few compound granular corpuscles are found, and only in older cases can corpora amylacea be demonstrated. The grayish discoloration of the posterior columns depends upon the destruction of the medullary sheaths. A marked degree of atrophy is to be noticed in the posterior columns, and in advanced cases the entire cord appears narrower and thinner than is normal. On cross section it is readily demonstrable that besides the posterior columns the posterior gray horns and the posterior roots also become atrophied. Moreover, it is of interest to note that certain portions of the cord seem, as a rule, to be spared, while others are almost always involved in the degeneration which affects both sides of the cord symmetrically.

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The lesion is of the character which we have learned to recognize in the so-called " combined system diseases "—that is, certain systems of fibres which have certain anatomical and physiological relations to one another become diseased, while others are unaffected.   It is also seen that not all portions of the posterior columns are implicated equally (Strümpell), but that the extent of the lesion differs according to its situation. For example, it is most severe in the lumbar region, in which only the anterior part is left intact, the middle and posterior portions being degenerated.   In the cervical region there are to be distinguished four fields on either side, of which, two, Goll's columns and a part of Burdach's columns, the so-called lateral root fields (into which direct fibres enter from the posterior nerve roots), appear degener-

Fig. 171.

Fig. 172.

Fig. 173.

Fig. 171.—Section through the Cervical Cord in a Case of Commencing Tabes. Fig. 172.—Section through the Lumbar Cord in Tabes. Fig. 173.—Section through the Cervical Cord in a Case of Advanced Tabes. (After Strümpell.)

ated, while two others, one anterior and lateral, the other posterior and external (the posterior outer fields of Strümpell), appear normal (Figs. 171, 172, and 173).   Such a distribution of the lesion is frequently observed, but naturally not found in all cases.   We have already mentioned that the posterior gray matter is involved in the process.   Lissauer deserves credit for having demonstrated (Arch. f. Psych. und Nervenkrankheiten, 1886, xvii, page 376) that here the affection of the fibres in Clarke's columns should be distinguished from that of the fine and large root fibres in the posterior horns.   Physiologically, this discovery can not as yet be utilized.

Of the lesions in the medulla oblongata and the brain, pro-

duced by tabes, the former affect the cranial nerves at their nuclei or in their peripheral course. Of the manifold symptoms produced thereby we have spoken before. On the other hand, we may have lesions of the cortex, an implication of which in many cases can not be called into question. We also said that some of the nuclei, particularly those of the eye muscles, of the vagus, and of the hypoglossus, are affected more often and more severely than others, while, for instance, the facial, the auditory, and the glosso-pharyngeal remain as a rule intact, a fact for which we have no explanation. According to Jendrassik's conception (Deutsches Arch. f. klin. Med., 1888, xliii, 6), the brain is the primary seat of the tabetic process, so that the sensory disturbances and the ataxia are to be considered as of cortical origin, and the degeneration in the posterior columns, and perhaps those of the direct cerebellar tracts, as secondary processes. Until the cortex has been examined microscopically in the initial stages of the affection, and some constant changes have been demonstrated in it after death, this theory, like all the others, will remain nothing more than a bare hypothesis, and can be neither contradicted nor yet accepted. Such a pathogenesis, however, is not impossible, though it is not difficult to bring forward objections to it.

Lastly, it should be mentioned that Basso (Ann. univers. di med. et chir., June, 1886) considers tabes to be an affection of the sympathetic system, under the influence of which the cerebro-spinal lesions develop. He thinks that the anatomical changes in the nervous system are at first caused by functional, and later by organic disease of the blood-vessels, and holds that when taken in time tabes is curable.

Ætiology.—In speaking of the ætiology of tabes, one must constantly distinguish, as is evident from the views expressed above, between the non-syphilitic and the syphilitic affection. In the first case one should above all take into consideration the hereditary conditions in order to comprehend the congenital predisposition which is necessary for the production of the disease. By this we do not mean to class tabes among the hereditary diseases in the ordinary sense of the word, for it certainly can not come under this category; on the contrary, we are justified in assuming that direct inheritance of it is quite rare. By heredity in this connection we mean a general neuropathic inherited tendency, or, in other words, that in the family of the patient all kinds of neuroses, not excluding psy-

choses, have occurred repeatedly. Not only the parents, but also more distant relatives, e. g., aunts, uncles, or grandparents, may have suffered with progressive paralysis of the insane, epilepsy, melancholia, hysteria, migraine, etc., and it is just this heredity which in the presence of exciting causes is sufficient to open the door to the tabetic process. The labors of Charcot (Arch. génér. de méd., September, 1883) and the comprehensive statistics of Ballet and Landouzy (Arch. de neurol., 1886, vii, 20) have thrown an interesting light upon this subject, and have brilliantly substantiated the view which Trousseau expressed at an earlier period, that tabes was (in the sense of the word as expressed above) hereditary. Among the German authors Möbius has occupied himself particularly with this subject (Allg. Zeitschr. f. Psych., 1883, xl, 1, 2).

The exciting causes which relatively frequently lead to the development of tabes (in those with hereditary tendencies) consist (*a*) in exposure to cold and wet, to sudden changes of temperature, and to prolonged living in damp lodgings; (*b*) in traumatic influences; (*c*) in certain factors due to the daily occupation, the most important of which is overexertion. The opinion that sexual excesses may lead to tabes, which has been expressed by various authors, must be given up as without proof.

I have never questioned but that exposure to cold, sudden changes of temperature, and, particularly, severe wettings, may play an important part in the ætiology of the affection; still, to me the following case was particularly convincing: The patient, a general agent for several hail-insurance companies, fifty-eight years old, had had syphilis thirty-nine years before, since which time he had been perfectly well. In August, 1885, while estimating the damage caused by a hailstorm, he was drenched to the skin, and was obliged to spend several hours in his wet boots. Three months later the first tabetic symptoms made their appearance—paræsthesia and anæsthesia of the legs, loss of the patellar reflexes, etc.; by Christmas, 1885, he was markedly ataxic, and in the spring of 1886 he was unable to pursue his calling. In the summer of 1886 he suffered with intestinal crises and intense lancinating pains, and eighteen months from the beginning of the affection he had paraplegia of both legs. In the early part of 1887 he died from an intercurrent attack of pneumonia. When tabes develops in one well on in the fifties, there must be some particular cause for it, and in this case

it was, without doubt, the wetting. Similar cases can easily be found if the history be carefully taken.

The *rôle* which traumatic influences play in the production of the affection is just as certain. In one of my cases, a government official of high position, who had been affected with syphilis twenty-nine years before, met with an accident on a glacier in the summer of 1884. He fell and slid some distance on a snow field with great rapidity, but no bones were broken and no dislocation occurred. A few months later the first tabetic symptoms made their appearance, and now the disease is well developed. Again, a fall from a height may be the cause (Oppenheim); Strauss reports numerous traumatic cases (Faits pour servir à l'étude des rapports du traumatisme avec le tabes. Arch. de phys., November, 1886). From his communication it is apparent (1) that years may elapse after the accident before the disease makes its appearance, and (2) that the traumatism may have an influence in determining the seat of the early symptoms, particularly of the lancinating pains, so that, for example, after a fracture of the lower part of the left leg the pains will first make their appearance at that point, and so forth. In an article by Spillman and Parisot (Traumatisme périphérique et tabes, Revue de méd., 1888, 3) there is a table which gives the different forms of injury which have been followed by tabes.

I have already pointed out, in my book on diseases of the laboring classes, that the occupation is not without importance, and more especially overexertion—for instance, at the sewing machine—and hard bodily labor in general, may be the cause of the outbreak of the disease in those who are predisposed to it. However, the percentage of such cases is not large. Hofmann gives an instance which may be classed partly with those cases in which the occupation, partly with those in which traumatism is the exciting cause. The patient was a laborer engaged in cutting tin plates, and in the course of his work his body was shaken from six to ten thousand times daily; under the influence of these shocks the disease developed (Arch. f. Psych. und Nervenkrankheiten, 1888, xviii, 2, 439).

Concerning the syphilitic tabes, which has been studied with the greatest care by Fournier and Erb, it is an undoubted fact that syphilis by itself is usually a sufficient cause for the disease, and that no other exciting factor is needed for its development.

We do not know what percentage of persons who have had syphilis become tabetic, but we do know for certain that the great majority of tabetics have had syphilis at some time or other—according to Erb, sixty per cent; according to Fournier, ninety per cent. Syphilis is more frequently followed by tabes than hereditary and exciting causes put together. Out of two hundred and forty-seven cases of tabes which I have seen in the last few years of my practice, in twenty-nine a syphilitic history was not obtained, while in the other two hundred and eighteen cases it was demonstrated with certainty, so that my figures almost correspond with the ninety per cent of Fournier. Minor points out in his statistics (Wyestnik psychiatri i nervipatologii, 1888, vi) that tabes is much rarer in Russia among the Jews than among the other Russians, which is simply due to the fact that the former are less frequently syphilitic. The communication of Nägel also deserves consideration. He found in 1403 cases of tabes forty-six per cent of syphilitics, and out of 1450 other patients only nine and one half per cent. The time which elapses between the infection and the first appearance of tabes varies from a few months to one, two, five, fifteen years or more. The severity of the syphilis does not appear to stand in any relation to the severity of the tabes; for one can observe very pronounced tabetic symptoms after an apparently trivial and quickly healed primary sore, whereas sometimes after the most severe form of syphilis the general affection of the nervous system only appears in its mildest form.

The influence which age and sex exert in the production of the disease can only be considered in the non-specific cases. It is, however, only of slight importance; for, although it is true that males are far more frequently affected than females (the proportion being seven to two), and although most of the patients are middle-aged, these facts can very well be accounted for by the nature of the several exciting causes, which make it comprehensible why men in the prime of life furnish relatively the greatest contingent of cases.

Lastly, it must be confessed that in a few cases, which, however, form an exceedingly small fraction of the whole number, no ætiological factor can be made out—neither hereditary predisposition, nor exciting causes, nor syphilitic infection. At present we can only acknowledge our ignorance of their pathogenesis.

Prognosis.—From what has been said, we may infer what the prognosis will be. Though it is not absolutely unfavorable *quoad vitam*, inasmuch as the patient may linger on for years, and sometimes tens of years, one should not forget that in general the course is unfavorable, that the patient will suffer greatly at times, and that the final lot of the tabetic is almost always a total inability to work or gain a living. In discussing the prognosis as to complete recovery, the question arises, Is tabes ever curable, or is there even a possibility of cure? This question is, with the proper restrictions, to be answered in the affirmative; it is possible to cure tabes, but only fresh cases of luetic origin. Advanced cases, in which degeneration in the cord has taken place, are incurable; we possess no means of bringing the lesion to a standstill or causing it to disappear. It is evident that the chances for the successful treatment of recent cases are increased the younger the patient and the better his general constitution. The prognosis is, *cæteris paribus*, less favorable in individuals with a neuropathic tendency, in whom the disease breaks out in consequence of some exciting cause, than in fresh specific cases. On the whole, one can say that out of two hundred and fifty tabetics one has a chance of regaining his previous health.

Treatment.—In taking charge of a case of tabes we must first see that we ourselves, as well as the patient, clearly understand how much can be expected from any treatment. If his is one of those exceptional cases in which the prognosis is relatively favorable, we may tell him so; but in most instances it will be our painful duty to make him acquainted with the seriousness of the situation, of which he will often be entirely ignorant. We must tell him with gentleness that a complete recovery can not be hoped for, and that all that it is possible to accomplish is to relieve some of his symptoms and to keep him in such a condition that he can as long as possible carry on his occupation. There is no disease in which it is more out of place to arouse in the patient vain hopes of recovery than in tabes.

The choice of the therapeutic measures themselves depends upon the stage of the disease in which we find the patient— that is, upon how long he has been sick. In old cases the greatest caution ought to be observed, and one should not forget that rash therapeutic interference may do more harm to the patient than good. The value of a treatment is often

quite problematical; its harmfulness is too often quite evident. Hygienic and dietetic measures, conscientious nursing and cleanliness, injections of morphine in severe attacks of pain, occasional cool baths, ever-repeated kindly encouragement, these constitute—if we leave out the suspension method, of which we shall speak later—the only treatment which old cases of tabes need, or, for that matter, can stand. But the recent cases also demand a great deal of care and forethought. In view of the duration of the treatment, protracted as it will probably be, all circumstances have to be taken into account— the constitution, the age, the occupation, and, above all, the pecuniary situation of the patient.

As syphilis is at the bottom of so many instances of tabes, the question whether we are justified in expecting anything from an antisyphilitic treatment should be mooted, but only in exceptional cases can we have such a hope—that is, only when either signs of syphilis are still present, or when the time that has elapsed since their disappearance is relatively short (not longer than a few months or at most a year). Such cases are very rare; generally years, perhaps twenty years, will have gone by during which the patient has been apparently perfectly well, and then the antisyphilitic treatment is of no avail. If, however, we wish to institute it for any reason, possibly because the patient himself insists upon it, bold doses ought to be given, four, six, even eight grammes (ℨ j– ℨ ij) of potassium iodide a day, and from three to six grammes (grs. xlv– ℨ jss.) of mercurial ointment rubbed in daily. In all, two or three hundred grammes (℥ vj– ℥ ix) of potassium iodide and the same amount of mercurial ointment ought to be used.

If we have resolved to try internal medicines, knowing, of course, that there is none which acts favorably upon the diseased nerve elements, we may begin with silver nitrate in doses of one centigramme (gr. $\frac{1}{7}$) in pill form three times a day for four or six weeks, after which time it may be combined with ergotin (arg. nitr., 0.3 (grs. ivss.); extr. secal. corn., 3, (grs. xlv); pulv. et extr. quass., q. s. ut f. pil. no. 30), of which also one pill is to be taken three times a day. Finally, a trial may be made with the salicylate of physostigmine, of which one milligramme (gr. $\frac{1}{66}$) in pill form may be given three times a day for a month, as recommended by Meyer in his paper on the Influence of Physostigmine upon the Patellar Reflex (Berlin. klin. Wochenschr., 1888, 2). With these drugs we may be fairly

confident that we are doing no harm, and often we may per-
ceive a distinct improvement in the condition of the patient,
although we are, of course, not able to definitely decide whether
this is actually to be attributed to the medicine or not. We
would recommend these remedies more warmly than any other,
even than strychnine, which has been administered subcutane-
ously in doses of from three to five milligrammes (gr. $^1/_{22}$–$^1/_{13}$),
gradually increased to one centigramme (gr. $^1/_7$) in twenty-four
hours, for repeatedly after these injections we have observed
the occurrence of pains, which had not been present for months.
In the treatment of the individual symptoms we must resort to
the same measures that we should adopt when these appear in
the course of other diseases or by themselves; for instance, for
the lancinating pains, as in other neuralgias, we shall be obliged
to give antipyrine and antifebrine, which have recently been
recommended by Lépine, Suckling, Germain Sée, G. Fischer,
and others, but we shall be driven to the conclusion finally,
that for the relief of these pains there exists only one drug by
the help of which the patient's painful existence may be ren-
dered at all bearable—viz., morphine, which here more than
in any other disease we are justified in using in large amounts.
Gastric and laryngeal crises, headache, etc., are to be treated
symptomatically.

In addition to the internal medication, it is especially the
electrical treatment which deserves consideration. This, if
used at a period early enough, may be followed by excellent
results, and may alone sometimes be capable of effecting a cure
or an arrest of the morbid process. On the other hand, if we
do not select our cases properly—for example, if we treat old
cases like recent ones—we may do more harm than good with
it. It may give rise to severe pains, and make the patients,
who until they were treated by electricity were in a fair con-
dition, begin to suffer terribly and soon lose confidence in the
physician. Electricity may also prove successful against the
motor disturbances, not so much against the ataxia as against
the weakness in the legs; also in combating anæsthesias and
paræsthesias in the hands and feet it may have some effect,
whereas it is usually of little avail against the lancinating and
rheumatoid pains. How to use the electricity, whether in the
form of the faradic or the galvanic current, it is impossible to
say in a few words. Every one forms for himself, in the course
of years of practice, his own technique, and gives preference

to this or that method; the one prefers the galvanic, another the faradic; again one will recommend the ascending, another the descending current through the spinal cord; the one believes in moist, the other in dry electrodes, especially the brush. Among all the different methods, besides the excellent general faradization advised by Beard and Rockwell, the faradic brush applied to the back, as recommended by Rumpf, has perhaps met with a more favorable reception than any other practice, and justly so. We prefer it, as far as electrical treatment goes, to all other modes. Details on the subject may be found in Erb's Handbuch der Elektrotherapie (Leipzic, Vogel). The treatment with dry cups, which has been urged by Lymann (Journ. of Nerv. and Ment. Diseases, July, 1887, xiv, page 409), is said to exert a favorable influence upon the nutrition of the muscles and the cutaneous nerves, by the rarefaction of the air, and thus to diminish the sensory disturbances. In recent cases this method may be made use of with good results.

In a large number of cases the cold-water treatment was found to be extremely beneficial. The action of the water on the peripheral nerve endings, the influence which cold douches, wet packs, moist ("Priessnitz's") abdominal bandages, cool baths, etc., exert upon the circulation in the vessels of the skin, and thus upon the terminal nerve twigs, is often so favorable that marked improvement during and after a stay in a hydrotherapeutic establishment is not rarely seen. Even in cases in which sensory and motor disturbances have attained to such a degree that but little can be hoped for, a carefully conducted cold-water treatment may be quite beneficial in improving the general condition of the patient and raising his spirits.

On the other hand, we would emphatically warn against the use of warm or hot as well as steam and sweat baths. As a rule they are of no avail, but often evoke the lancinating pains. Unfortunately, the physician is not always in a position to prevent this, since the patients, who believe implicitly in the rheumatic nature of their pains, use them at random without his orders often for months and years. There are a great many tabetics who during the course of their disease have taken many hundreds of steam baths, without perceiving the slightest benefit therefrom.

From the springs we can, on the whole, expect but little, and especially old cases with paraplegia and severe bladder troubles should be spared the trial. The disadvantages, the

overexertion attendant upon the journey, and the lack of home comforts, in the case of these patients especially, will far outweigh any good results obtained from the baths; nor should we, as we said above, leave out of sight the necessary cost which, even with the most modest pretensions, is not inconsiderable. One should never forget that the disease is likely to last a very long time, that the patient will soon be unable to earn any money, and that for him there can be no greater misfortune than to find that, heedlessly or yielding to overpersuasion, he has spent all his worldly goods of which now he stands in the greatest need. There exist not a few of such helpless patients in whose cases just this point was overlooked, and it is especially our younger colleagues who seem rather too prone to disregard it. If such and other objections do not exist, it is most advisable to recommend places where warm brine baths can be taken, as in Rehme-Oeynhausen, this place having become famous for the treatment of tabes especially, though it is my experience that patients get along there no better and no worse than at other springs of the same kind—e. g., Nauheim —and it only deserves to be warmly recommended owing to the excellent arrangements which we there find, particularly the facilities for moving helpless invalids from place to place. Chloride-of-sodium springs containing iodine and bromine— for instance, Königsdorf-Jastrzemb, Kreuznach, Goczalkowitz, Krankenheil—may be tried without fear of doing any harm; while the nonmedicated hot springs of Gastein, Teplitz, Johannisbad, Warmbrunn, Pfäffers, and the hot sulphur springs of Landeck, Aachen, Trentschin, Pistyán, Baden near Vienna, and Baden in Switzerland should be prescribed only with great caution, and the baths should never be taken too warm, never above a temperature of 80° to 90° F. Among the chalybeate springs, first Cudowa, then Pyrmont, Flinsberg, Schwalbach, and St. Moritz (Engadine) deserve to be tried.

The results of massage in the treatment of tabes are not satisfactory. There is no objection to giving massage in a careful manner so as to improve the nutrition of the muscles and to stimulate metabolism, especially in cases of young, comparatively robust patients; but we are hardly justified in building much upon such a procedure and in expecting to bring about a lasting improvement in the sensory or motor disturbances. I have known instances in which the general condition of the patient was influenced for the worse by massage, and in which

certain symptoms, especially the lancinating pains, appeared to be aggravated after its use.

Of only historical interest is the operation of nerve stretching, which, in the first half of the eighties, was by some claimed to be an excellent means in the treatment of tabes, the sciatic nerves being usually chosen for this operation. They were laid bare by cutting through the gluteal muscles and "stretched" according to different methods. The result was in many cases at first very striking. Pains, bladder disorders, and anæsthesias vanished, and the operation was undertaken comparatively frequently. Soon, however, it was found that what had been regarded as a success was of no long duration, and that the old troubles returned, and, finally, after it had been repeatedly demonstrated at the autopsy (Strümpell, Rosenstein) that the elongation of the nerves not only had not exerted the slightest beneficial effect upon the morbid process in the spinal cord, but that several times at the place where the nerve had been stretched a neuritis had developed and extended to the substance of the spinal cord, giving rise to a myelitis, the practice was given up, and can be looked upon to-day as having been definitely discarded.

Fig. 174.—SUSPENSION APPARATUS USED IN THE TREATMENT OF TABES.

Finally, after a warm recommendation by Charcot, a method has recently again been taken up which some years ago had been written about by Motschukowsky (Wratsch, 1883, 17–21). The patient is suspended by means of an apparatus (cf. Fig. 174), as he would be if he were going to have a plaster-of-Paris jacket applied, and allowed to remain so for a few minutes.

The results obtained with this mode of treatment in the Salpêtrière were favorable enough to induce many clinicians in

Germany, England, and America to make further trial of it, and at present we possess quite an imposing array of articles treating of the " suspension method " and the results obtained by it. According to some authors the cerebral, according to others the spinal symptoms are improved by it. The procedure is said to be without danger, but in one instance the immediate consequences were fatal; yet we should add that in this case the suspension was undertaken without the physician's supervision (Gorecki, Lyon méd., 1889, 20). Althaus (cf. lit.) has attempted to give an explanation of the mode of action of this treatment. According to his opinion, the meningitic adhesions over the posterior columns are loosened, so that the nerve fibres, especially the superficial ones, gain in power of conduction, the sclerosed, thickened neuroglia becomes looser, and the pressure upon the nerve tubes is thus diminished. He also thinks that suspension should only be used in older cases, because in recent ones it might lead to inflammatory conditions. The possibility that this loosening does take place, as Althaus claims, can not be disproved, but this is certain, that for those instances in which improvement is said to have shown itself after only one, two, three, or ten suspensions, this theory affords no explanation.

We have tried this method on one hundred and three patients in private practice and in eleven cases in the hospital wards, all of whom had tabes. Among the one hundred and three private cases there were twenty-one women and eighty-two men; among the hospital patients there were four women and seven men. The number of suspensions in the different cases varied from three to eighty-seven, the duration of each being from one to five and a half minutes, and the whole treatment comprised from three to a hundred and sixteen days. Among the patients some were older, some more recent cases. The earliest period after the onset of the disease at which the treatment was begun was fourteen months, the latest, seventeen years.

In no single instance was I able to note any marked or lasting improvement. In no case was either the general condition of the patient or the course of the disease influenced for the better by it, nay, even in the individual symptoms no decided improvement could be perceived. Transient diminution of the ataxia, transient relief from the pains or bladder troubles could be remarked, but these ameliorations were of no

duration, and soon everything was again just as it had been before. The account which intelligent and unprejudiced patients gave of themselves after the thirtieth, fiftieth, and eightieth suspension corresponded almost exactly to that which they had given prior to the institution of the treatment. In opposition to Althaus, then, it is my conviction that anatomical changes are not produced by suspension, but that the transient improvement has to be referred to the influence of suggestion. The patients hear of a new treatment for their incurable disease, they subject themselves to it with much pleasure and confidence, and by autosuggestion produce an improvement in some functional impediment (for example, in the ataxia), which may be quite marked, but which is never lasting. Four times during the act of suspension I myself met with rather unpleasant accidents; in two cases the patients lost consciousness and had to be rapidly taken down, and were only then with some difficulty recalled to life; in two other instances severe laryngeal crises appeared, so that the procedure had at once to be stopped. Such accidents, of course, make a very bad impression upon the patient, and bring the results, which are in any case doubtful, still more into question. Careful examination is necessary before the suspension is used, and if there exists a disease of the heart or of the vessels it should under no consideration be undertaken. We need, of course, hardly add that while the patient is suspended he should be carefully watched. A final judgment as to the usefulness of the method can, of course, not as yet be ventured upon, because our time of observation has been too short; the impression, however, which I myself have gathered, is that suspension will be in vogue for a time, only to fall again into complete oblivion.

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CHAPTER III.

DEMENTIA PARALYTICA PROGRESSIVA—GENERAL PARALYSIS OF THE
INSANE—GENERAL PARESIS—SOFTENING OF THE BRAIN.

WHILE in tabes we have learned to recognize a disease of
the general nervous system, in which the spinal cord chiefly is
the affected portion, we find that in dementia paralytica, on the
other hand, the brain is above all the part attacked, whereas
the spinal cord and the peripheral nerves do not suffer so regu-
larly nor to so marked a degree. In its distribution the lesion
of the cord is either diffuse or, as in tabes, confined to the pos-
terior columns. With regard to the affections of the spinal
nerves a more careful study is still necessary, and more espe-
cially this question needs to be answered whether here, as
seems indeed very probable, primary degenerative processes,
analogous to those of tabes, can also be demonstrated.

Ætiology.—The manifold points of resemblance between
general paralysis and tabes, to some of which we have drawn
attention above, are found first in the ætiology of the two dis-
eases. In the former as well as in the latter hereditary tenden-
cies are of much significance. A person belonging to a family
in which nervous diseases of any kind have been prevalent is
more prone to develop general paralysis than a member of a
healthy family. However, this factor is in the majority of in-
stances not sufficient in itself to bring about the disease, certain
exciting causes being also necessary, and experience has taught
us that it is chiefly overstrain of the nervous system, and more
especially of the brain, which favors its development. Such
excessive demands which are made upon the brain are numer-
ous. Chief among them is mental overstrain, caused by too
close attention to work and worrying over business—too much
scheming, calculating, etc.—so that we are not surprised that
bookkeepers, accountants, bankers, stock brokers, authors,
actors, etc., form the relatively largest contingent of cases.

Besides this, deep emotions, repeated or long-lasting sorrows or care, the struggle for existence, disappointed hopes, baffled ambitions, and fright, may obtain ætiological importance. On hearing the history of a general paralytic, one at least of these factors will hardly ever be absent. Bodily overstrain, forced marches, excesses *in Venere* and the like, can be made responsible to a less extent. Excesses *in Baccho*, the habitual abuse of alcohol, only rarely lead to general paralysis, but sometimes a condition is produced by such excesses, the " pseudo-paralysis a potu," which resembles general paralysis, but which is quite distinct from it, and belongs to chronic alcoholism. There is no question but that the occupation may furnish causes for the disease ; thus, those which entail at once bodily and mental work, or those in which the workers must for a long time remain in very hot rooms, and again the working in poisons, especially in lead (Snell, Vogel, and others), are particularly dangerous. Of great interest, finally, is the fact that, like tabes, general paralysis may be caused by traumatism either to the head or the back, so that we have a traumatic progressive paralysis which is quite analogous to the traumatic tabes. In this latter category we must also place the insolation, sunstroke (heat stroke) which has been known to lead to general paralysis (Bonnet and Paris, Ann. méd.-psych., November, 1884, 6, S. 12).

Besides the congenital, however, there exists also an acquired predisposition, which differs from the former, inasmuch as no other exciting causes are needed for the production of the disease, but that, just as is the case in tabes, it alone is sufficient to bring about general paralysis. We refer, of course, to syphilis. The same highly important *rôle* which it plays in tabes it plays here too. An individual who has had syphilis has much greater cause to fear general paralysis than one who has never been infected. According to the statistics of Rieger (cf. lit.), the one is sixteen or seventeen times as liable to the disease as the other. These figures correspond about to those founded on my own experience. Out of two hundred and fifty-seven paralytics a hundred and seventy-one had been syphilitic, and out of two hundred and sixty patients with other diseases only fourteen. Heredity and all the exciting causes taken together do not give rise to as many cases of paralysis as syphilis alone ; but here again, as in tabes, we must leave the question open as to how syphilis acts, whether, as we

think ourselves, the syphilitic arterial disease is responsible, or whether we are dealing with a toxic action (cf. page 624), so that general paralysis has to be regarded as a post-syphilitic affection. Whatever we decide about this, the fact that general paralysis may be the result of syphilis is universally acknowledged, and the numerous writings which we possess on the subject are all without exception in favor of this view.

The influence which has been ascribed to age and sex can usually be explained by that of syphilis. Males are more frequently attacked than females, the ratio being seven men to two women. Those in the prime of life furnish the largest contingent.

Symptoms.—The symptoms of the disease are partly psychical, partly somatic, and this will not surprise us when we learn that the seat of the affection is preferably in the brain, and more particularly in the psycho-motor region of the cerebral cortex. The psychical manifestations differ very greatly, and it is more especially in the prodromal stage that these variations are most noticeable. This is a feature equally well marked here as in tabes, and the main difference between the two consists in the fact that in general paralysis the clinical picture of the prodromal stage is dominated by the psychical manifestations. The patient becomes unable to concentrate his mind for any length of time. He gets easily fatigued when he has exerted himself mentally, he becomes forgetful, and is no longer able to comprehend and deal with matters which he previously understood perfectly. He is found to be indifferent in the performance of his duties and careless in keeping his appointments; he becomes unreliable and absent-minded. When writing, he makes mistakes in spelling, and presents a slowness in thinking and a general dullness of intellect which are quite foreign to him. At the same time his disposition presents alterations. Previously tolerant and kind, he becomes now ill-humored, moody, and irritable; on the slightest provocation he loses his temper and may even be inclined to violence. His character is not the same as it was; his will power becomes weak; he loses his energy and his moral individuality; he allows himself to be influenced and overpersuaded by anybody, and even thus early does things for which he can give no clear motive; he gradually loses all consideration for others in his social intercourse; he neglects his appearance, his dress looks

untidy, he becomes indecent, commits nuisances on the open streets, tells obscene stories before his children, and so forth. In exceptional cases the patient himself is to a certain extent conscious of these changes which are going on in him. They surprise him, and he speaks about it to his most intimate friends and expresses a fear that some serious disease is coming on; but in the great majority of cases, he does not in the least appreciate his condition, which worries and troubles his family so much. Months, even years, may thus pass and no new manifestations make their appearance. It is only the occurrence of certain somatic symptoms which gives to the clinical picture a different aspect. Among these latter there are especially two on account of which the physician is consulted, namely, insomnia and the alteration in speech. The former is all the more striking because the patients often by day and at their work are overpowered by sleep, while at night they lie awake for hours without being able to rest. The latter manifests itself by a difficulty in pronouncing certain words. The patient stutters, misplaces letters and syllables, leaves syllables out, in a word, presents the group of symptoms known as "syllable stumbling" (*Sylbenstolpern*). At the same time the voice loses its usual timbre; it becomes harsh and its former modulation is gone.

For the examining physician, the associated movements in the facial muscles, the fibrillary tremor and twitching of the lips, and the tremulousness of the protruded tongue are sufficient to lead him to the diagnosis, and the inequality of the pupils which may appear at this stage is an important sign. The motor disturbances further manifest themselves in a change in the handwriting and in the gait. The writing shows uncertainty and irregularity; the letters, which are usually larger and written more awkwardly than before, become tremulous; the paper is covered with blots; the words are incorrectly written, inasmuch as letters or entire syllables are omitted or misplaced. The gait becomes awkward and clumsy and the patient "shuffles along"; he is one-sided, and small obstacles in his path are apt to cause him to fall.

This initial stage, which in its duration varies from a few months to one, even two or three years, is followed by a stage which is mostly characterized by a rapid increase in the psychical excitement ("maniacal exaltation"). The patient—previously quiet, sullen, apparently occupied with his own

thoughts—now becomes noisy, talkative, all the time restless and in a state of excitement; without noticing his surroundings and his friends, he lives with a sense of perfect comfort; he is young, handsome, extremely strong, and immensely rich; he has studied all sciences; he occupies himself with absolutely preposterous but to him feasible schemes; he is going to dry up the Atlantic Ocean, he is the Emperor of China, he is Napoleon, Christ, he is the chief among the gods, etc. In the dreamlike play of his imagination all these fantasies arise, but the patient is not able to give them any logical connection. Without critical faculty he stands out a pitiable victim of the most bizarre delusions of grandeur. At the same time his memory rapidly fails him, especially for recent events; what he did to-day, yesterday, the last visit of the physician, etc., he does not remember, whereas the reminiscences of long-past years can still be called up. He does not know the day of the week or even the name of the month and the season in which he is at present. People with whom he used to deal in business he no longer recognizes; he confounds them with other persons, etc. The lack of judgment of the patient has, of course, a decided influence upon his actions; he buys things recklessly, squanders his money in a most foolish manner, he makes debts, commits easily discoverable frauds, which he denies with the utmost calmness when he is found out. Assaults of which he may be guilty, misdemeanors against the public order, offenses against the public morals, etc., not infrequently lead to trouble with the authorities and to the arrest of the patient.

In by far the smaller number of cases the above-described initial stage is followed, instead of by the maniacal exaltation, by a stage of depression. The patient believes himself persecuted by everybody, and his life menaced; he hears voices, and he is always troubled with a presentiment that something terrible is going to happen. He cries, laments, begs for help, and so forth. In other instances hypochondriacal delusions gain the upper hand. The patient imagines that he is made of glass, that he can not eat, that he is unable to urinate, that he has no head, and the like. The lack of all power of criticism in these delusions, and the inability to systematically elaborate them, and the usually rapid course of this stage, distinguishes the general paralytic from the insane patient.

Quite gradually in the course of time the general aspect

changes, the excitement abates and disappears, and the intellectual impotency increases. The patient spends his days without a thought or care, writing and reading become to him lost arts, he forgets his own name, and his social position, he becomes oblivious of his family, and in general takes no interest whatever in the outside world. This is the stage of dementia. He becomes uncleanly in his habits, his eating and drinking must be watched, and step by step the psychical life approaches more and more its extinction; the patient no longer lives, he vegetates.

It is of great practical importance and interest to study the somatic disturbances which occur in the course of the disease associated with the psychical ones, and which are caused by the simultaneous affection of the spinal cord (and peripheral nerves). The diminution of sensibility, that of the skin as well as of the nerves of special sense, particularly of the opticus, the absolute inactivity of the pupils, the decrease in the perception of pain, the changes in the electrical excitability of the muscles, which at first is increased, later diminished, the (not regular) loss of the tendon reflexes, the appearance of trophic disturbances, the tendency to bedsores, the perforating ulcer of the foot (*mal perforant du pied*, cf. page 610), all point to a participation of the spinal cord in the morbid process. Sometimes, quite early, peculiar attacks occur, which, associated with loss of consciousness, are either accompanied by transient hemiplegias or convulsive movements, and which therefore either deserve the name of apoplectiform or epileptiform seizures. They are designated as "paralytic attacks" *par excellence*. Under certain circumstances they appear very frequently, from ten to fifty times in one day, and they may then keep the patient in an almost constant condition of unconsciousness. The elevation of temperature which accompanies these attacks is not considerable, the occurrence of albumen in the urine not constant. Among the affections of the cranial nerves which have been but little studied in their connection with general paralysis, there is particularly the optic atrophy, which is seen in ten per cent of all cases. The nerves of the ocular muscles also frequently become attacked, the implication of the trigeminus and of the facial being less common. Of the nuclear affections of the vagus coming on in the course of this paralysis nothing definite is known.

The duration of the disease varies much. In the "galloping

form," in which, owing to the sleeplessness and inability to take
sufficient food, the strength rapidly fails, it may require only a
few months to bring about a fatal issue.   At other times the
disease may last two, three, five, or even more years, out of
which no small proportion is liable to be spent in an asylum,
as it is positively impossible to keep the patient at home, in
spite of all the care and devotion possible on the part of the
family.

Pathological Anatomy.—The questions as to the anatom-
ical nature of the disease have unfortunately not been as yet
answered satisfactorily, and there is still a great deal of diver-
sity of opinion among the authorities on this point, although
the macroscopical appearances are usually very characteristic,
the atrophy of the brain, especially in the anterior regions,
being very striking.   Although no one can doubt that the con-
volutions are diminished in size, that the frontal and the parie-
tal lobes weigh less than in a normal brain, yet the precise
mode in which this atrophy comes about, what are the micro-
scopical changes in the nerve elements of the cortex, and what
is the primary process in all this, is not as yet decided, but re-
mains the subject of much controversy.   According to Tuczek,
there is a marked primary atrophy of the fine medullated nerve
fibres, particularly in the outer layers of the cortex, in the
tangential " association " fibres, which run parallel to the sur-
face.   The gyrus rectus is said to be relatively the earliest
attacked, later, the remaining frontal brain and the island of
Reil, then the temporal, but the occipital lobes never.   This
view, according to which the atrophy is the primary process,
is in all probability correct, although it is still combated by
many authorities (Mendel), who look upon the death of the
nerve fibres as the secondary, upon the increase of the con-
nective tissue, the thickening of the vessel walls, and the ap-
pearance of spider cells, as the primary process (" encephalitis
interstitialis ").

Analogous changes in the ganglionic cells have frequently
been noted (Binswanger, Mendel, Gudden); a peculiar aggre-
gation of nuclei associated with disease of the vessels, changes
in the bodies of the cells in the large pyramids of the para-
central lobule, changes in the nucleoli and nuclei, and sclerosis
and atrophy of the cells are not uncommonly found in this
connection.

But, besides the cortex, the deeper regions also are the

seat of alterations, and the manifold changes, which the white matter of the hemispheres may undergo, have been studied among others by Friedmann. He describes four different forms of atrophy of the fibres of the white matter, the number of the fibres diminishing in a manner analogous to that which has been shown by Tuczek to be true for the cortex. The central ganglia of the brain do not remain exempt, nor, as Westphal has shown, are the pyramidal tracts or the posterior columns of the spinal cord spared. Fascicular degeneration occurs in them also, a fact which probably accounts for a not inconsiderable part of the motor disturbances.

The condition of the pia varies. Frequently it is adherent over large areas of the underlying cortex, so that it can not be stripped off without loss of substance ("decortication"). In rare instances, although it is nowhere adherent, in places it is thickened, of greater consistence than normal, and contains variable amounts of fluid in its meshes. Whether the latter condition is only a later stage of the former—that is, whether adhesions only exist at first, but later disappear—is not definitely known.

A case recently reported by Rey (cf. lit.) shows that exceptionally all the symptoms of progressive paralysis of the insane may be observed during life, and yet at the autopsy no change be found. The same thing, as we have mentioned, has been known to occur in connection with multiple sclerosis.

Diagnosis.—The diagnosis may present some difficulty, inasmuch as in certain forms of chronic alcoholism the egoism may also be exaggerated as in general paralysis, and inasmuch as cerebral syphilis, brain tumor, senile dementia, finally, chronic meningitis and multiple sclerosis, may more or less resemble general paralysis in their course and their symptoms. In alcoholism the hallucinations are wont to be a prominent feature, the speech disturbances are less marked, and the ideas are worked out in a more connected manner. The tremor and the history in cases of chronic alcoholism will also assist us in our diagnosis. In cerebral syphilis also the history as well as the age of the patient (who is, as a rule, younger than the paralytic) must be taken into consideration. Brain tumors present a similarly progressive course, but the stage of exaltation is absent and the characteristic delusions of grandeur do not occur; in place of them we have stupor and somnolence. Senile dementia, of course, occurs in people of advanced age, and is

characterized by a tendency of the process to remain stationary for some time.

In meningitis we have febrile symptoms; the choked disks, which are found comparatively frequently here, and the delirium which occurs early will guard us against errors. In multiple sclerosis, finally, we have the scanning speech and the intention tremor, and in cases where the disease is well developed, it can not be mistaken for general paralysis. In certain forms, however, the differentiation may be impossible. The most important points to be remembered in the diagnosis of general paralysis are, then, the following: The pronounced psychical weakness, which even in the initial stages is the most prominent feature of the disease; the constantly progressive course; the motor as well as the sensory changes, the former of which give rise to more or less marked alterations in the speech, the hand-writing, and the walk, the latter to changes in the impressionability to external stimuli and to marked interference with the functions of the nerves of special sense—the cutaneous sensibility, the sense of taste, hearing, and smell. With this in mind we shall make a correct diagnosis at least in a good many cases; to avoid errors completely will be impossible even to the most experienced.

Prognosis.—We need hardly say much about the prognosis. From the above description we can well infer how unfavorable it must be. Almost all cases prove fatal in a few years, and the outlook for complete recovery is worse here than in tabes. To be sure, it has been claimed that such may occur in progressive paralysis (Wendt, Voisin), but, in the instances in which it was observed, the possibility that the case was not one of dementia paralytica, but rather one of the so-called pseudo-paralyses, such as are known to occur after the abuse of alcohol, can not be excluded with certainty.

Treatment.—In the treatment of the disease we must chiefly endeavor to keep away all excitement from the patient, and, since this is best and most easily accomplished in an asylum, it is the first duty of the physician, after he has once made the diagnosis definitely, to urge the family to transfer the patient to some such institution. Only then is it possible to guard the patient as well as the family against all the accidents and fatalities to which he is otherwise necessarily exposed. This step must be taken as early as possible, not with the idea that the patient will be cured, but with the conviction that only in an

institution is he safe, and that there alone it will be possible to secure for him the proper care and nursing so necessary for one in his condition. Where there is a history of syphilis, the treatment with inunctions must of necessity be given a trial, however slight may be the prospect of success. Once decided upon, let the antisyphilitic treatment be pursued with vigor; at least three to four hundred grammes (℥ ix– ℥ xij) of mercurial ointment should be used altogether, to which must be added from two to three grammes (grs. xxx–xlv) of the iodide of potassium daily for a good while. To meet the outbreaks of exaltation and the insomnia the usual hypnotics, which are, however, of little avail, should be tried. Sulphonal in doses of two or three grammes (grs. xxx–xlv), methylal in doses of from five to eight grammes (grs. lxxv– ℥ ij), by the mouth (Mairet and Combemale), morphine, from one and a half to three centigrammes (grs. $\frac{1}{4}$–$\frac{1}{2}$) hypodermically, chloral, paraldehyde, possibly also hyoscyamine, should be tried in turn. The cold-water treatment and baths, also galvanism to the brain, are decidedly contraindicated. All such procedures are likely only to increase the excitability of the patient, to give him all kinds of unpleasant sensations, and to make his troubles worse, without being in any way of benefit to him or relieving his condition.

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CHAPTER IV.

IN different places in our book, in the chapters on diseases of the brain as well as in those treating of affections of the spinal cord, we have had occasion to point out the *rôle* which syphilis plays as an ætiological factor in various diseases. We have also shown that tabes and the progressive paralysis of the insane are to be regarded as the main representatives of affections of the general nervous system depending upon syphilitic infection. It only remains, therefore, in the present chapter to add some general remarks to what has already been stated.

No part of the nervous system, whether of the brain or of the spinal cord, is exempt from the chance of becoming implicated in the syphilitic infection, and remembering how the blood-vessels are affected by syphilis this is easily understood. Clinically, it is especially interesting if we are able to recognize diseases of the cerebral cortex and symptoms—e. g., monoplegias—resulting therefrom, as syphilitic, but the corona radiata and the basal ganglia, the pons, the medulla oblongata, and the cerebellum, all may become the seat of syphilis, and syphilitic affections of the base of the brain are relatively common. In many cases it is difficult to make a certain diagnosis, especially if the patient denies the primary sore and no trace of it can be found, for the clinical symptoms, of course, are the same, whether the brain lesion depends upon syphilis or not.

Symptoms.—Among the manifold symptoms which occur in brain syphilis we may mention polyuria and polydipsia (cf. Buttersack, lit.), but these may, it seems, occur also primarily. If focal symptoms are present it is easier to make a diagnosis than in their absence. In the latter case it may sometimes be impossible to decide upon the diagnosis of brain syphilis; we may be dealing with a case of cerebral neurasthenia.

With reference to the spinal cord the matter is somewhat

more simple, because syphilitic disease here, which does not implicate also the brain, as in tabes and progressive paralysis of the insane, is rather rare. It is not a common thing to find disease of one system of fibres or disease of several systems combined depending upon syphilis, and the cases where lateral sclerosis, for example, was attributed to this have been published as rarities. In the work of Rinecker (cf. lit.) all the references on this subject will be found. It is of pathological interest to note, as Buttersack has also pointed out, that the root bundles usually present a marked and extensive participation in the process. In a case reported by Siemerling (cf. lit.) there were gummatous growths of the pia, which, although they had extended into the substance of the cord, had not attacked any "system" in its whole extent, so that, as is often the case, the spinal symptoms here also were not at all prominent.

We can not assume that the spinal nerves, either motor or sensory, ever become diseased alone, but we must rather look upon their implication as a partial manifestation of a general affection. If in exceptional cases we find a neuritis of the sciatic or of the musculo-spiral nerve, etc., which we have to regard as of syphilitic origin, perhaps because it rapidly passes off under antisyphilitic treatment, the manifestations of cerebral and spinal syphilis have either existed previously and have not been recognized or their presence later has to be looked for.

Diagnosis.—The diagnosis is based first upon the history of the patient and the presence of signs of the primary sore. If these are established, it is relatively easy; if not, we must look for other signs to help us. Secondly, the other organs—for instance, the skin, the visible mucous membranes—all must be examined for the possible existence of syphilitic lesions. Repeated and careful search may sometimes clear up much that is obscure, although the patient's account may be imperfect. Thirdly, it must be remembered that the symptoms of cerebral syphilis are extremely changeable, and are rarely ever of long duration. To-day, matters may look as if the patient's life were in danger, while to-morrow he is apparently perfectly safe again. The rapidity with which the changes in the condition follow each other, just as in hysteria, the extraordinary circumstance that apoplectiform attacks occur in younger and epileptiform attacks in older persons, in doubtful cases are in favor of the diagnosis of syphilis of the nervous system. In every instance we shall do well to pay careful attention to the

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condition of the eyes, and be on the lookout not only for a possible ophthalmoplegia, but also for iritis. Finally, the therapeutic test is of some value, inasmuch as the successful antisyphilitic treatment makes the existence of syphilis almost certain, although a failure does not warrant the contrary conclusion.

Prognosis.—The prognosis must, above all, be influenced by the time which has elapsed since the primary infection and before the first appearance of the nervous symptoms. The longer this period of incubation the worse is the prognosis. According to my own experience, from five to nine years is the most common time. Occasionally the infection of the nervous system manifests itself earlier, and in quite exceptional instances two years, or even one year after the primary lesion; but often the interval which elapses is longer than the time above given. Cases in which the spinal or cerebral symptoms did not appear till after twenty or twenty-five years I have never seen get well. The second question of importance is, how long the nervous symptoms have existed before energetic antisyphilitic treatment was commenced. Often as it remains without effect, a trial of it is still indicated if not more than two or four months have elapsed after their appearance. If they have existed for half a year or longer, nothing can be expected from any such treatment, and it need not, therefore, be begun. In such cases the prognosis, of course, is worse than in the others. Thirdly, a good deal depends upon the kind of symptoms by which the infection of the nervous system manifests itself. General symptoms, headache, vertigo, epileptiform attacks, allow, *cæteris paribus*, of a more favorable prognosis than focal symptoms, such as monoplegias, hemiplegias, and paralyses of certain nerves. The worst outlook is afforded by those instances in which the brain and the spinal cord are equally severely attacked, as in tabes and progressive paralysis of the insane.

Treatment.—The manner in which the treatment is to be conducted must be made to depend upon the individual case, the age, the nutrition of the patient, and so forth, and no rule applicable to all cases can be laid down. Only one remark, which has repeatedly been made before in this book, we wish here again to emphasize, namely, that if we have once decided upon adopting the antisyphilitic treatment we must do so energetically, giving iodide of potassium, one to six or eight

grammes (grs. xv– ℥ jss.– ℥ ij) daily, in one or two doses in hot milk, continued for from six to ten weeks, and inunctions of blue ointment, from three to five grammes (grs. xlv–lxxv) a day, continued for from four to six weeks. All necessary precautions are self-evident. Finally, we should not neglect to familiarize ourselves with the progress which has been made in the modern treatment of syphilis, and consider whether the subcutaneous use of mercury is advisable, and, if so, what the exact mode of its administration then should be.

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APPENDIX.

IT would be beyond the scope of this work to treat here at length of the pathology of the intoxication paralyses, which form a well-recognized part of the poisonings and the diseases in general due to the various trades and occupations; we only wish at the end of this book to point out, that working in poisons may under certain circumstances lead to diseases which affect the general nervous system, and which therefore can well be treated of here in an appendix to the preceding chapters.

The intoxication paralyses come on either acutely or gradually; their symptoms and course are influenced and modified (*a*) by the nature of the poison; (*b*) by the manner in which the poison enters the body, whether through the organs of respiration or digestion or through the skin, either when the epidermis is intact or when there is a loss of continuity in places. In how many ways the work itself—that is, the different manipulations, etc.—favor the entrance of the poison into the system can not here be dwelt upon. In the third volume of my Krankheiten der Arbeiter this subject is fully discussed.

The substances which are here concerned are inorganic as well as organic; gaseous poisons may also be injurious. Among the inorganic substances it is especially certain of the heavy metals, which first attract our attention. Every year hundreds and thousands of people are occupied in elaborating them so as to adapt them for their different uses, and the number of persons whose health is thereby damaged is considerable, in spite of the measures of precaution established by law.

The motor disturbances which develop under their influence are more particularly paralyses, but before the occurrence of these symptoms, and also during their existence, certain signs of motor irritation may be observed, which manifest themselves frequently in tremor, or more rarely in spasm. Certain

metals—for instance, mercury—seem to be particularly apt to give rise to symptoms of irritation, whereas others, like arsenic and lead, seem more rarely to have this effect; in cases of poisoning from the latter, symptoms of paralysis are more marked. Sensory disorders are rare, and only in certain intoxications—for instance, in lead poisoning—do they constitute an annoying and prominent feature of the disease. Lead, more than any other of these substances, it is which produces the severest disturbances in those who occupy themselves with it, and it is safe to say that the brain as well as the spinal cord and all the peripheral nerves are equally exposed to its injurious influence.

Lead Palsy, the affection which interests us more than any other belonging to the present category, is confined chiefly to the extensors of the wrist and fingers, the triceps and supinator longus remaining exempt from the morbid process. There are cases in which the extensor region is intact, but in which the deltoid and biceps, possibly also the small muscles of the hand, are affected. However, the extensor paralysis alone is to be regarded as typical of lead poisoning. The investigations of Möbius more particularly, have shown that the muscles most exerted become affected alone, or at least more seriously than others. Here may also be mentioned the interesting observation of Manouvriez, that in left-handed people the left arm is more affected than its fellow. As a matter of fact, both arms are frequently attacked to the same degree. The time at which the paralysis sets in varies greatly. It is very unusual for it to appear only a few days after exposure to the poison, and the case of Hérard (cf. lit.), where a working man developed extensor paralysis of both arms the same day on which he had filled a barrel with white lead, is decidedly unique. Usually some weeks elapse before the paralysis manifests itself, and often it is preceded by one or more attacks of lead colic. The course is tedious; if the poison continues to act, the affected muscles undergo atrophy relatively rapidly and to a very marked degree, and in such cases recovery becomes improbable. On electrical examination, we generally find the reaction of degeneration; with the diminution in the number of the muscle fibres the excitability decreases and becomes lost completely and forever in grave cases. The anatomical seat of lead palsy is unquestionably to be looked for in the peripheral nerves, possibly also in the muscles. It is a peripheral atrophic paralysis with a degeneration of the musculo-spiral and, to a

lesser extent, of the median and ulnar nerves. In the spinal cord and the anterior roots, in cases of this class, gross and striking changes are not demonstrable (Vierordt). Possibly the paralysis may also be explained by a change in the ganglionic cells of the spinal cord, which takes place in consequence of the peripheral irritation in the manner lately described by Ssadowski in his Inaugural Dissertation (Petersburg, 1889). According to his opinion, the accumulation of the stimuli affecting the peripheral nerves brings about an increased activity of the corresponding gangli, and with this a disturbance in the nutrition of the nerve cells, which leads to an atrophic degeneration.

Besides the peripheral, we have also central lead palsies : (*a*) those which depend upon spinal, (*b*) those which depend upon cerebral lesions ; the former, the significance of which E. Remak more particularly has emphasized, are connected with diseases in the ganglionic cells in the anterior horns of the spinal cord, which are affected according to their anatomical position, so that groups of muscles are attacked which in their functions are connected with one another. The changes found in the spinal cord consist of numerous small hæmorrhages, particularly in the cervical enlargement, in the middle portions of the anterior horns, and in the existence of small atrophic, in places pigmented, ganglionic cells, which are often found to have lost their nuclei (Oeller, cf. lit.). The saturnine cerebral palsies manifest themselves as hemiplegias which appear suddenly and resemble in every detail the ones described above (page 225 *et seq.*). It is not clear how their development should be explained. Disregarding the few cases in which the hemiplegia was connected with chronic nephritis and heart hypertrophy, the action of the poison has either to be regarded as giving rise to vaso-motor changes or else we must suppose that certain parts of the central nervous system are directly thrown into a state of irritation (Heubel, Malassez, Harnack).

The sensory saturnine paralyses—e. g., the hemianæsthesia which occasionally accompanies the hemiplegia, must also remain as yet unexplained. The problem is by no means solved by calling this toxic hemianæsthesia hysterical, and the question whether the group of symptoms which we call hysteria can ever be the consequence of the occupation of the patient, and more particularly the consequence of lead poisoning, must still remain open. Possibly various symptoms which for the

sake of convenience are called hysterical, in reality depend upon anatomical changes. The appearance of epilepsy, and of eclampsia, as also of certain states of excitement associated with hallucinations, also of the habitual headache (encephalopathia saturnina), of the amblyopia with consequent amaurosis, all point to an affection of the entire nervous system of a degree which is not found in poisoning from any other inorganic substance.

Recent investigations have made it probable that zinc in which among other impurities a certain amount of lead is found chiefly owes its poisonous action to this latter. Pure zinc, as it seems, has no injurious effect upon the nervous system, and the cases formerly described as intoxications due to zinc (Schlockow, Deutsch. med. Wochenschrift, 1879, v, pp. 17, 18) must be regarded as cases of lead poisoning (Laffter). The possibility that some injurious action may also be attributable to an admixture of copper with the zinc we have mentioned on a former occasion, in our work Krankheiten der Arbeiter, iii, 1885.

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    workers. Brit. Med. Journ., December, 1888.

Arsenical paralysis may set in from five to ten days after
the symptoms of an acute intoxication have made their appear-
ance, sometimes not until some weeks later. It commences
with pains and paræsthesias in the extremities, which persist
for a variable period of time, together with a marked weakness
of the lower extremities, which soon makes walking and stand-
ing difficult for the patient. The extensors are the muscles
most seriously attacked. Dorsal flexion of the foot and the
toes becomes difficult, the walk is pronouncedly ataxic; the
legs are raised a good deal during walking in order to keep
the drooping foot from touching the ground. The presence of
Romberg's sign, together with the gait, may lead us to think
of tabes if the history of arsenical poisoning is, for some reason
or other, not clear. The affection of the upper extremities
consists of paresis of the extensor muscles, of those of the
thenar and antithenar, which, although the elbow and shoulder
joint remain intact, gives rise to a considerable disturbance of
function in the hands. To this motor disorder there is soon

added, as we have seen to be the case in lead poisoning, a pronounced atrophy of the muscles, which is more particularly marked in the lower extremities, while it is less noticeable in the arms. The electrical excitability is here also, at least in the grave cases, completely lost. Vaso-motor changes do not occur, with the exception of occasional œdema about the ankles. The whole disease lasts for from a few months to three, four, or five years. Although complete recovery is not common, as a rule some improvement is noted, and the patient regains some movements. Arsenical paralysis may be mistaken more particularly for syringomyelia, for multiple neuritis, and for tabes; however, the history of exposure to arsenic, the peculiarity of the motor disturbances, which are more marked in the lower than in the upper extremities, the nonimplication of the sphincters, and the rarity with which the cranial nerves are attacked, will, as a rule, be sufficient to allow of a correct diagnosis. That the anatomical seat of the arsenical paralysis must be sought for in the peripheral nerves, that we are dealing with a peripheral neuritis, and more particularly with a toxic form in the sense of the term used by Leyden, has been shown by Strümpell and Falkenheim, and quite recently again by Alexander (cf. lit.).

The pathogenesis is, according to the same author, to be connected with the fact pointed out by Silbermann (Deutsche medicinische Wochenschrift, 1888, p. 504), who claims that under the action of arsenic an obstruction of numerous capillaries can be demonstrated in the different organs, especially in the lungs, the gastro-intestinal tract, the liver, and the kidneys. Similar circulatory disturbances in the nervous system Alexander claims to be the cause of the arsenical paralysis.

There is no doubt but that arsenic may have a noxious action not only upon the peripheral nerves, but also upon the central nervous system, although, according to the statement of Alexander, Scolosuboff and Popow could not find, in their examination, any changes in the spinal cord after arsenical poisoning. Pathologically, the action of arsenic upon the brain has not been demonstrated either, yet certain clinical facts seem to point to an affection of this organ also. Instances where workers in arsenic suffer from headache, psychical depression, apathy, and sleeplessness, although now, owing to the better prophylaxis, much less frequently met with, certainly do occur, and prove that the brain may also be affected

by the poison.   Yet this influence is less marked and less constant in the case of arsenic than in that of lead.

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Mercury may also give rise to paralyses, but they are far more uncommon and develop much more slowly and gradually than those due either to lead or arsenic.   I have repeatedly

Fig. 175.—Specimen of Handwriting of a Patient (hat-maker) with a Mercurial Tremor.

seen paresis of the upper and lower extremities in those working in quicksilver, but these did not attract the attention of the

observers much because they came on, without exception, during the presence of or even long after the first appearance of another symptom, which, as it were, dominated the clinical picture, namely, the tremor. In those working in mercury this tremor is seen even much more frequently than the typical ex-
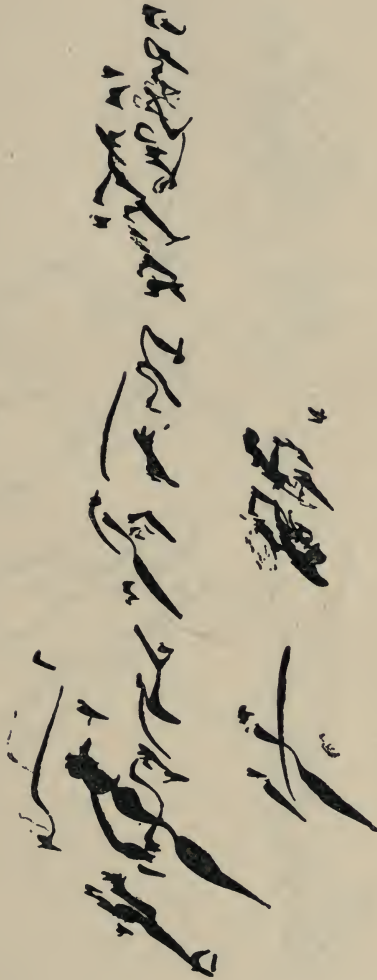
Fig. 176.—Specimen of Handwriting illustrating the Tremor produced in a Case by the Combined Action of Alcohol and Mercury. (V. Finkenstein, 4, 86.)

tensor paralysis in lead-workers. It begins usually in the face or the tongue, and gradually invades the upper and then the lower extremities. Its grade of intensity is different in different cases. From a slight tremor, resembling that of paralysis agi-

tans, every stage, up to the strongest shaking movement, even convulsive jerkings passing through the whole body, and similar to those of a severe chorea, may be observed. They cease during sleep, but in the waking stage are aggravated by every
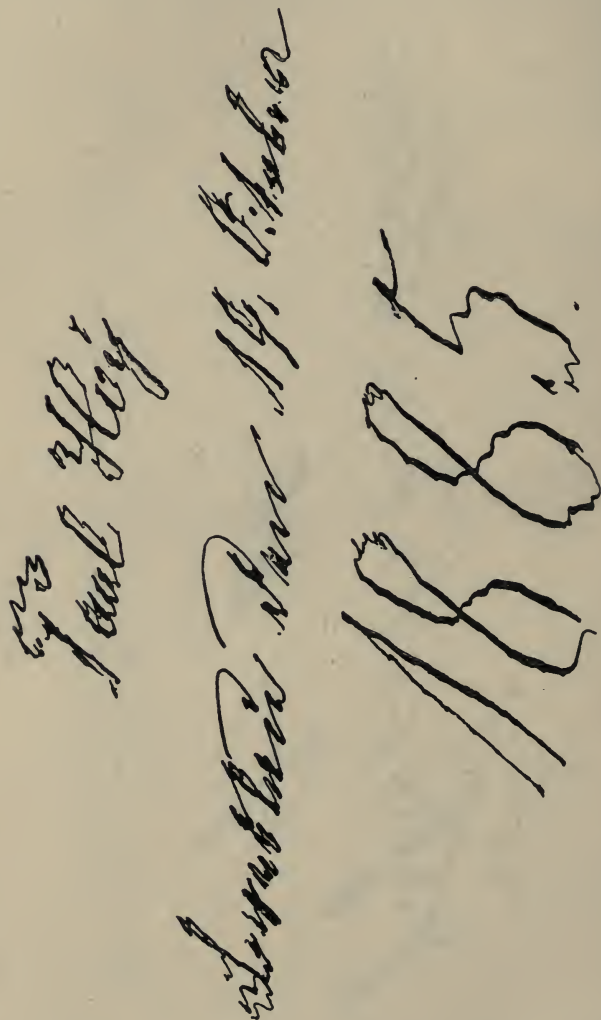
Fig. 177.—Specimen of Handwriting illustrating Alcoholic Tremor.

movement, so that they remind one of the tremor of multiple sclerosis. After this has lasted for years, more or less pronounced muscular weakness appears, but paralyses without tremor do not occur as a result of quicksilver poisoning. Ac-

cording to the investigations of Letulle (cf. lit.), we are here again dealing with a peripheral disease, the myeline sheath becoming degenerated while the axis cylinders remain intact. To this latter circumstance the possibility of recovery from
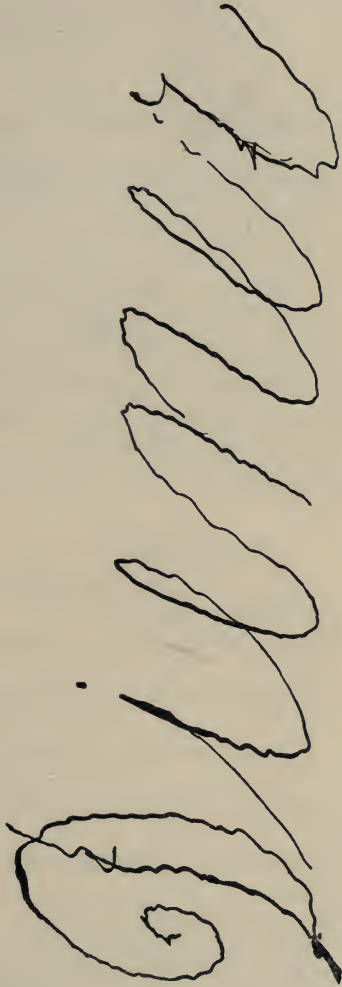
Fig. 178.—Specimen of Handwriting illustrating Tremor Senilis.

mercurial tremor may be attributed. Besides the tremor there are seen circumscribed areas of anæsthesia and hyperæsthesia. The occasional amblyopias, the diminution in acuteness of hearing and smell, the gradual dulling of the intellectual facul-

ties, which may pass into pronounced dementia, all point to an implication of the entire nervous system.

The belief which lately has been repeatedly expressed (Letulle, Guinon), that in connection with mercurial poisoning hysterical symptoms may appear, so that we should be justified in speaking of a mercurial hysteria, must be held subject to this reservation, namely, that simultaneously with this intoxication, hysteria may occur just as it is known to accompany neurasthenia, tabes, and many other nervous affections. To regard this, however, as a toxic hysteria which has its own peculiarities and course is, according to our opinion, unquestionably not justifiable. Hysteria will remain hysteria no matter whether it develops under the influence of mercury, alcohol, or lead, etc. If, as may be the case, speech disturbances and nystagmus occur in the course of mercurialism, care must be taken not to mistake it for multiple sclerosis. Wising (cf. lit.) has considered the question of the differential diagnosis between the two. The difference between the mercurial and other forms of tremor with regard to the oscillations is shown in the specimens of handwriting which are represented in Figs. 175 to 178, all taken from observations of my own.

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Further, bisulphide of carbon must be mentioned in this connection, under the influence of which motor paralyses and

anæsthesias have recently been observed. Certain authors have also designated the symptoms produced by these gases as hysterical, and have coined the name "bisulphide of carbon hysteria." We have just expressed our opinion on such a conception.

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Finally, a few words may be devoted to the highly interesting disease of the central nervous system attributable to the ingestion of ergot. Clinically, as Tuczek (cf. lit.) has observed, the brain and spinal cord take an equal part in the affection, but as a rule anatomical changes in the latter only could be demonstrated. They were similar to those found in tabes, consisting, namely, of a degeneration of the posterior columns, so that we were tempted to call the disease "ergotine tabes." The whole course of the disease, however, which is not progressive, and which differs greatly from that of tabes, does not justify this designation, which besides would only be liable to give rise to misunderstandings.

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