Dr Adams

A

CLINICAL TREATISE

ON THE

DISEASES

OF THE

NERVOUS SYSTEM

BY M. ROSENTHAL

Professor of Diseases of the Nervous System at Vienna

WITH A PREFACE BY PROFESSOR CHARCOT

TRANSLATED FROM THE AUTHOR'S REVISED AND ENLARGED EDITION

BY

L. PUTZEL, M.D.

Visiting Physician for Nervous Diseases, Randall's Island Hospital. Physician to the Class for Nervous Diseases, Bellevue Hospital Out-Door Department, and Pathologist to the Lunatic Asylum, B. I.

NEW YORK

WILLIAM WOOD & COMPANY

27 GREAT JONES STREET

1879

TROW'S
PRINTING AND BOOKBINDING CO.,
205-213 *East 12th St.*,
NEW YORK.

CONTENTS.

II.—*DISEASES OF THE CEREBRAL PARENCHYMA.*

CHAPTER II.

CHAPTER III.

CHAPTER IV.

CHAPTER V.

CHAPTER VI.

CHAPTER VII.

CHAPTER VIII.

CHAPTER IX.

CHAPTER X.

CHAPTER XI.

CHAPTER XII.

CHAPTER XIII.

CLASS II.

DISEASES OF THE MEDULLA OBLONGATA.

CHAPTER XIV.

CHAPTER XV.

CLASS III.

DISEASES OF THE MENINGES AND PARENCHYMA OF THE CORD.

I.—*DISEASES OF THE SPINAL MENINGES.*

CHAPTER XVI.

CHAPTER XVII.

II.—*DISEASES OF THE PARENCHYMA OF THE CORD.*

CHAPTER XVIII.

CHAPTER XIX.

CHAPTER XX.

CHAPTER XXI.

CHAPTER XXII.

CHAPTER XXIII.

PREFACE.

I WAS acquainted with the present treatise long before a French translation was contemplated, and, mindful of the services it has rendered me in my teachings, I have accepted, with pleasure, the task of presenting it to its new circle of readers. In any work of recent date upon the pathology of the nervous system we expect to find, in accordance with a very widespread penchant, theories regarding physiological mechanism predominate over the descriptive portions, and even dilated upon at the expense of clinical and anatomo-pathological details—those true foundations of all solid work in this department. But the Vienna School is not open to the reproach of having succumbed to these tendencies; and, as far as concerns Dr. Rosenthal's work more particularly, it appears to us to justify, in great part at least, its title of *Clinical Treatise*. It is very certain that the materials placed before us, as well as the spirit of the work, have been derived by the author from a long career devoted to the study of disease; the care bestowed upon the symptomatic descriptions is sufficient testimony to this fact. It is undoubtedly true that the subject could hardly have been arranged more harmoniously, nor could the pathological descriptions appear in more vivid and striking colors; but it would be difficult to push further, than has been done in this work, the constant habit of examining questions from all points of view. This is not done by departing from the clinical spirit but by appealing to information furnished by experiments upon animals, when they are legitimately applicable to man; or, still further, by invoking at each step the revelations of pathological anatomy. We need not be surprised that the latter study is an object of special attention in a work written in Vienna and dedicated by the author to Prof. Rokitansky.

The erudition displayed in Dr. Rosenthal's treatise does not recognize the boundaries of country, though it is but natural that the author should chiefly refer to the works of his compatriots. We will find in this work, which is not deficient in original observations, a concise but complete exposé of important researches upon various questions in nervous pathology by L. Tuerck, Benedikt, Meynert, and other authors of the Vienna School. Among works of this character, which are, in my opinion, too little known among us, I shall restrict myself to calling attention to those which discuss the anatomical lesions of the nervous centres in hydrophobia, the lesions of the arteries in cerebral syphilis, and, finally, the observations which have established, according to Dr. Voigt, the relations of cutaneous anæsthesia and hyperæsthesia with the distribution of the nerves to the integument.

The study of foreign works upon nervous diseases has led me to pay attention to certain studies in comparative pathology, which may be applied to the investigation of the changes which morbid types of this class experience, according to climate, nationality, race, etc., but without losing their autonomy. As a rule, we are only able to point out slight shades of difference in a study of this character. But the deviation may sometimes be characterized by more or less profound modifications, even in adjacent countries and under very similar parallels of latitude.

Thus—in order to mention an example which I have very recently had under observation, and which I propose to discuss in detail at some future period—the hysterical neurosis in England differs markedly from that observed in France in certain symptomatic features. Complete hemianæsthesia, among other peculiarities worthy of mention, and the hystero-epileptic grand mal, which are so common in our country, are observed very rarely on the other side of the Channel, while permanent contracture of the limbs and other symptoms of this character (sometimes called *local hysteria* by our neighbors) are, on the other hand, of frequent occurrence. It does not appear that the same remark can apply, in this particular case, to Austria, since the clinical history of hysteria in Dr. Rosenthal's work is almost identical with that observed in our own midst. In other respects the differences of soil are perhaps more strongly marked. Is it on account of

local peculiarities that the author is inclined to belittle the importance of sexual excesses in the etiology of locomotor ataxia, and to lay especial stress upon exposure; and that he considers the prognosis of cerebro-spinal meningitis as relatively favorable, in fact almost benign, an assertion which cannot fail to cause some surprise among the epidemiologists of our country?

The portion devoted to treatment is not the least interesting. The author has been known to have long paid especial attention to the study of medical electricity, and useful indications are found in his work, both for diagnosis and treatment, even to the citizens of a country which numbers Duchenne, of Boulogne, among its representatives. But we should pay especial attention to the rules which are laid down for the employment of hydrotherapeutic measures. As it is presented to us by Dr. Rosenthal, that is to say, divested of those disturbing agents which have roused so much prejudice against it, and reduced to simple and always moderate measures, in accordance with the doctrines long since advocated in France by L. Fleury, Dr. Lubanski, Sr., and some others, hydrotherapeutics should enter more and more into the treatment of chronic diseases of the nervous system, in which it must be regarded as one of our most powerful resources.

I believe that I have shown with sufficient clearness that Dr. Rosenthal's work presents itself to our notice with strong claims to our esteem. It is called upon to fill a gap in our literature. For although original works on diseases of the nervous system have, for the past few years, rapidly followed one another in France, we do not, at the present time, possess any special work in which the data recently acquired upon the various subjects which compose this large department of pathology are collected in a didactic form.

J. M. CHARCOT.

PARIS, *Sept. 15th*, 1877.

AMERICAN TRANSLATOR'S PREFACE.

I have been led to present Dr. Rosenthal's work to the profession in this country chiefly for the reason that it is in my judgment the best practical treatise on nervous diseases hitherto published here. Since 1870 the book has run through two large editions in Germany and Austria, and the French edition of 1878 has met with exceedingly favorable criticism at the hands of French neuropathologists. Dr. Rosenthal's work is emphatically a clinical treatise, and is particularly adapted to the wants of the practitioner; although not by any means neglecting the physiological pathology of the subject, especial attention has been paid to the more practical departments of symptomatology and therapeutics. In this respect we desire more particularly to call attention to the advocacy of hydrotherapeutic measures in chronic nervous diseases which has been entered into so enthusiastically by the author. Hydropathic treatment appears to be steadily gaining ground, both in France and Germany, as a valuable therapeutic agent, and the present treatise does good service in calling attention to this agent, since its employment has been almost entirely neglected by American physicians.

Both the German and French editions were published without plates. I have introduced a number of woodcuts (copied from various foreign works), and hope that their evident utility will atone for the liberty taken with the original.

L. PUTZEL.

252 E. 48th St., N. Y.

CLINICAL TREATISE

ON

DISEASES OF THE NERVOUS SYSTEM.

GENERAL CHARACTERISTICS OF CEREBRAL DISEASES.

No method of investigation can embrace, in all their exuberance, the characteristics of the normal activity of the brain. We can only make conjectures concerning them, with the imperfect means of exploration at our command. Experimentation seeks to interpret the disorders which are produced during life, by artificially destroying the integrity of the brain.

This interpretation is not always correct and safe, since lesions, which cannot possibly be controlled, may complicate and aggravate the results of the experiment. We must especially exercise great caution in applying the results of experiments on animals to the higher organization of the human brain.

On the other hand, clinical observation and pathological anatomy also furnish data, which are explained by physiology, and thus receive scientific endorsement.

Experimentation upon the brains of mammalia—whose organization is so much inferior to that of man—leave many questions unanswered with regard to the human brain. Thus, in disorders of the higher mental faculties peculiar to man, and in disturbances of speech, sensibility, and the special senses, we must be guided by the large number of pathological observations which have been carefully collected, aided by the latest developments in histology. It is in this manner that the clinical study of nervous diseases has, during the past few years, been filling up the gaps in our physiological knowledge.

Central lesions are revealed by symptoms in which the observer recognizes the signs of a disturbance which has occurred in the functions of the brain. Here belong the large group of so-called cephalic symptoms, in addition to disorders of the different cranial nerves, of motion and sensation on the opposite half of the body, and, finally, of the larger portion of the organic functions.

The series of cerebral symptoms peculiar to diseases of the brain or its meninges, generally begins with cephalalgia. This may gradually increase, may be diffuse or circumscribed, as in febrile processes, or may assume

the character of a fixed, intermittent, or only remittent pain, as in tumors. The cephalalgia is often accompanied by vertigo, roaring in the ears, photophobia, nausea, and vomiting. Consciousness is only blunted in slight cases, but, in severe cases, it is entirely lost.

The intellectual faculties are involved in a variable manner, in different diseases of the brain. All stages of disordered intelligence are observed, from simple excitement, incoherence, hallucinations and melancholia, to a condition of mania or low forms of apathy, idiocy, and imbecility.

The faculty of language is often affected in cerebral diseases. The disorders of speech may have a motor origin, as when the lesion involves the fibres of the roots of the hypoglossal nerves (in diseases of the pons varolii; it is then called alalia or anarthia by Leyden), or when it occurs in the course of the ascending fibres, as in localized lesions near the central ganglia. In other cases, language is involved, with preservation of the movements of the tongue, and of intelligence, as in aphasia. This occurs in cerebral embolism, especially when the superficial or deep anatomical lesions exist in the central organ of articulate speech—in the island of Reil and the parts which connect this with the frontal lobe, and with the central and parietal convolutions.

Disorders of special sense must also be placed in this category, and they constitute, in central lesions, complications of equal frequency and importance. In the first rank, we must mention changes in the eye which, in cerebral embolism, and especially in tumors of the brain, may be determined by ophthalmoscopic examination, and furnish a firm foundation for diagnosis. The changes in the retina due to optic neuritis, cause impairment of sight (amblyopia) or total loss of vision (amaurosis). We shall discuss this subject, at full length, under the head of cerebral tumors. Hearing, smell, and taste are affected in chronic inflammations and in lesions of certain parts of the brain (especially of the base of the brain, the pons, the cerebellum and peduncles).

Irritation of the motor functions is manifested by stiffness or spasms in the muscles of the face or limbs, and, in more serious conditions, by tonic, clonic or choreiform convulsions (with disorders of co-ordination), or even by tremor of a limb or one half the body. Contractures, general convulsions, and all the baleful symptoms of an epileptiform attack are not unfrequently manifested. The characteristic group of cerebral paralyses are less often produced by hyperæmia or anæmia of certain regions, than by localized lesions of a chronic character (hæmorrhage, encephalitis and its consequences, sclerosis, tumors, &c.). Sometimes primary lesions of the tissues may occur, causing destruction of the site of motor impulses and of their paths of transmission; sometimes secondary lesions occur in the same situation, from diseases of the cranial bones or cerebral meninges, from the growth of tumors, from chronic hydrocephalus, and from anæmia due to arterial embolism, all of which causes may give rise to paralyses.

The hemiplegic type of paralysis upon the side opposite to the cerebral lesion, is due to disease of the motor ganglia (corpus striatum and lenticular nucleus), or to localized lesions of the cerebral lobes, involving the peduncles and pons varolii. The paralysis of one half the body generally attacks the muscles of the limbs, especially the extensors, and often with contracture of their antagonists. When hemiplegia occurs upon the same side as the cerebral lesion, a foyer is usually found in the motor ganglia of the opposite side, with alteration in the cerebral lobes of this side, and softening or œdema in the parenchyma of the other hemisphere. The muscles upon the paralyzed side generally undergo no noteworthy change

with regard to electrical sensibility. At the onset of paralysis from cerebral inflammation or apoplexy, the farado-muscular reactions and the galvanic excitability of the nerves, may be increased. Cerebral hemiplegia is nearly always accompanied by paralyses of the cranial nerves, whose varied characteristics may offer valuable indications as to the situation of the cerebral lesion. If the lesion is situated in the system of fibres between the cerebral ganglia and the lower nuclei of the gray centre (Hirnstamm), and in the motor ganglia, paralysis of that portion of the fibres of the facial nerve which passes through the foot of the cerebral peduncle, will occur upon the same side as the hemiplegia, and the paralysis will therefore only involve the inferior or respiratory muscles of the face. In foyers of hæmorrhage or encephalitis, the paresis of the ocular muscles is accompanied by deviation of the eyeball, with rotation of the head to the side opposite the hemiplegia. In isolated lesions of the cerebral peduncle, incomplete paralysis of the oculo-motor nerve occurs upon the same side, that is, upon the side opposite to the hemiplegia, with incomplete paralysis of the face and tongue. In lesions of the pons, alternate hemiplegia is almost always produced, that is to say, facial paralysis upon one side, and paralysis of the limbs on the opposite side. According to Brown-Séquard, when the lesion is situated above the decussation of the fibres of the facial, the facial paralysis is observed upon the same side as the hemiplegia. It is alternate, on the other hand, when the lesion is situated below the decussation. As we shall demonstrate at a later period, in the chapter on tumors of the pons, it is more reasonable to believe that a lesion, situated above the nucleus which gives rise to decussation of the facials, will produce alternate facial paralysis; that the compression of the roots of the facial will cause its appearance on the same side; and that, finally, lesion of the inferior facial nucleus will produce incomplete facial paralysis on the opposite side. Alternate facial paralysis is generally complete, and, as in peripheral facial hemiplegias, the faradic contractibility of the muscles and nerves is abolished, the galvano-muscular contractility is increased, and the galvanic reaction of the nerves is diminished or lost. Lesions of the pons may also give rise to paralysis of motion and sensation in the domain of the trigeminal nerve, and to paralysis of the muscles of the eye and tongue, and of the acoustic and optic nerves. Cerebral paraplegias usually result from the coexistence of two distinct hemiplegias, and are caused by symmetrical foci of disease in the ganglia or in the cerebral peduncles, by circumscribed lesions in the median portion of the pons, by aneurisms at the base, or by more extended processes in the pons and medulla oblongata. The association of these paraplegias with multiple paralyses of the cranial nerves, is a characteristic symptom. Disturbances of co-ordination, of an ataxic character, are found in diseases of the pons and cerebellum.

The sensory disturbances, which are at first produced by cerebral diseases, consist of vague pains, formication or a sense of heaviness in the limbs upon the affected side, trigeminal neuralgia (especially in tumors), and increased reflex excitability. At a more advanced stage, more or less severe disturbances are produced in hæmorrhage, softening or tumors (upon the side opposite to the lesion), from interruption in the conducting paths of sensation. The central termination of the latter is situated in the medullary substance, behind the lenticular nucleus, as far as the occipital lobe, the histological demonstration of which statement we shall adduce in the article on cerebral apoplexy. Alterations of sensibility also occur when the lesion involves the intermediate points of the route followed by

the sensory impressions, in the external portion of the foot of the cerebral peduncle and in the pons varolii and medulla oblongata. In tumors of the pons, sensory disorders (crossed with regard to the motor paralysis) often occur.

This topography of lesions of sensibility must, in the future, be carefully examined and verified by histology, in a large number of cerebral diseases. These investigations are of greater utility in human pathology than experiments made upon animals (like those lately begun by Veyssière, Arch. de Physiol., 1874), which are always less exact and trustworthy.

Among the remote complications of cerebral diseases, we may mention strabismus, irritative contraction of the pupils, their final dilatation, their insensibility, and disorders in the functions of the heart and in the respiratory movements. From the united results of experimentation and pathology, it appears that increase of the cerebral compression at first causes slowness and then excessive acceleration of the pulse (at first, irritation, then paralysis of the origin of the pneumogastrics). The respiratory changes also depend on increase of the cerebral pressure. It is, at first, frequent and irregular, and, when coma develops, it becomes slow and deep.

This review of the symptomatology of cerebral diseases may be terminated by a few remarks on vaso-motor disorders, and trophic lesions. Acute diseases of the brain (meningitis, encephalitis, apoplexy), are recognized, in general, by early rise of temperature. Maintenance of the temperature at a considerable elevation (41° C. and even 42° C. in meningitis) or rapid elevation after it has been lowered for a short time (as Bourneville has observed in apoplexy) are usually indicative of a fatal termination. The temperature is lowered in old hemiplegias, in which continuous retardation of the circulation and passive hyperæmia are present.

Among the trophic disturbances we may mention bed-sores (acute decubitus) occurring upon the paralyzed side, in the first few days after an attack of apoplexy, or other cerebral disorders and arthropathies of the lower or upper limbs in hemiplegias of an apoplectic origin. The alterations of nutrition in the muscles may be absent or insignificant in cerebral paralyses which have lasted several years, since the trophic centres of the muscles have not been positively located in the brain. Charcot has observed a case of progressive muscular atrophy (with loss of electrical contractility) in a left apoplectic hemiplegia, in which the limbs had been contractured at the beginning. At the autopsy, descending sclerosis was found in the left lateral column of the cord, and extending to the corresponding anterior gray horn, with spots of atrophy of the nerve cells. The slightest pressure upon the skin of the paralyzed limbs sufficed to provoke an eruption of bullæ, which were promptly transformed into eschars.

Various pathological conditions may cause the spinal cord to participate in cerebral diseases, thus effacing the lines of demarcation which are theoretically drawn between the nervous centres. In several forms of basilar meningitis, tubercular products may develop upon the meninges of the brain and cord, or inflammatory exudations and pus may pass through the foramen magnum from the brain to the cord, as in cerebro-spinal meningitis. In multiple sclerosis of the nerve centres, the brain, medulla oblongata, and spinal cord undergo a fibrillary transformation in places. The peri-encephalitis or, according to others, the chronic interstitial lesions of progressive general paralysis are often complicated by gray degeneration of the cord.

Not infrequently, morbid foci in the brain are the starting-point of

secondary lesions of nutrition in the paths of transmission in the spinal cord. As Tuerck has first shown, foyers of apoplexy in the cerebral ganglia and their vicinity may lead to atrophic degeneration of the antero-lateral columns. The severe sensory disturbances which are found in hemiplegic limbs (as in apoplexy) are due to anatomical changes in the corresponding organs of conduction, from the medullary layer of the cerebral ganglia to the fibres of the posterior spinal columns. Compression of the medulla oblongata by tumors of the pons or cerebellum, and this secondary degeneration of the spinal paths in case of tumor, may give rise to paraplegia and disorders of sensation. This phenomenon is much more infrequent in syphilis of the brain or cord. Finally, double cerebral emboli and thrombosis of the vessels of the pons, which, according to Duret, are supplied by the trunk of the basilar artery, may induce paralysis of all the limbs.

The diagnosis of cerebral diseases has made considerable progress of late years, thanks to important achievements in physiology, clinical observation and pathological anatomy. The more critical investigation of the symptoms of localized lesions, the more rational grouping of those phenomena which indicate the situation of the lesion; the more complete study, especially of hemiplegias, of their relations with paralysis of the cranial nerves, of the disturbances of the psychical faculties and special senses, of early retinal lesions (recognizable with the ophthalmoscope), of characteristic disturbances of speech, of anomalies of reflex sensibility and of the sensibility to the electric current—these facts have contributed a great deal towards clearing up certain obscure symptoms, and towards bringing symptoms of extremely variable character into relation with their real point of origin.

The clinical study of tumors, encephalitis, cerebral embolism, cerebro-spinal sclerosis, etc., has only been attended with successful results in the last few years. We hope that the difficulties which still encompass the localization of symptoms, on account of the multiplicity of the diseased processes, may be gradually cleared up by a more profound study of the cerebral functions in health and disease, and by the combined efforts of experimentation and clinical observation.

CLASS I.

DISEASES OF THE MENINGES AND PARENCHYMA OF THE BRAIN

I.—DISEASES OF THE CEREBRAL MENINGES.

CHAPTER I.

From their position and relations, the membranes enveloping the brain are frequently subject to disease. They may participate in lesions of the cranial vault and they are also frequently the starting-point of diseased processes.

A.—Diseases of the Dura Mater.

Hyperæmia of the dura mater, accompanying congestion of the subjacent membranes, offers merely an anatomical interest. We need only consider, from a clinical standpoint, inflammations of the dura mater, viz., perimeningitis or pachymeningitis. Following Virchow, we recognize two forms of inflammation, that of the external surface and that of the internal surface of the dura mater. The two surfaces are sometimes simultaneously involved.

a. *External Pachymeningitis.*

Inflammation of the external layer of the dura mater usually follows traumatic lesions of the skull-cap, diseases of the bones, caries and suppuration of the vertebræ and neighboring ligaments.

The *anatomical lesions*, in acute and slight cases, are characterized by the development of vascular rosettes upon the external surface of the membrane, which is red, softened and covered with fine layers of exudation. In the more serious cases, ecchymoses are found in the dura mater which is also thickened by exudation or infiltrated with pus, and friable.

The superficial portions of the dura mater, especially those which are bare (as under an opening made by a trephine), readily suppurate and furnish abundant granulations projecting across the loss of substance. In serious affections of the bones, as in caries, the pus accumulates between

the cranium and dura mater, detaching the latter little by little and threatening the bone with necrosis. The inflammation may also extend to the sinuses; their walls become thickened, their inner surface reddened, and a purulent or ichorous thrombus is found within the sinus. All these lesions may be limited to the dura mater, but if they extend deeper, general meningitis will result.

In the chronic forms of external pachymeningitis, the dura mater becomes considerably thickened, is adherent to the skull, and the new-formed false membranes are transformed into osseous lamellæ.

Pathogenesis.

External pachymeningitis is rarely produced by syphilitic or rheumatic affections of the pericranium or by erysipelas of the scalp. It more frequently follows thrombosis with suppuration of the sinuses of the dura mater, but especially of the transverse and petrous sinuses. In insanity, Foerster has found the dura mater thickened, of a cartilaginous consistence, anæmic and sometimes pigmented. More frequently, the process is secondary to injuries to the skull, either with or without fracture, and to caries of the internal ear, of the cribriform plate of the ethmoid, or of the first cervical vertebræ.

Symptoms and Course.

The symptoms are not always sufficiently clear to permit a diagnosis during life. There are, nevertheless, etiological conditions, such as traumatism, internal otitis, and caries of the upper cervical vertebræ, by means of which we may diagnose this condition when symptoms of inflammation appear. Intense and circumscribed cephalalgia, vertigo, nausea, vomiting, and slight convulsions are the first symptoms. Recovery is, nevertheless, possible, as is proved by the fact that partial thickenings and cicatrices are found in the dura mater.

Increase of the headache in extent and intensity, and also of the somnolence, immobility of the face, of the inequality of the pupils and of the acceleration of the pulse, testify to the increase of the process. The final slowing of the pulse, coma, irregular chills and paralyses are of evil augury and indicate suppuration and compression of the brain.

Treatment.

At the first signs of inflammation, absolute rest, restricted diet, and powerful antiphlogistic measures are indicated. Cold compresses to the head and neck, application of leeches to the mastoid processes, and powerful derivations to the intestines are the most efficient means. In comminuted fractures with compression, trepanning may, according to the individual case, be promptly followed by relief. When the disease is due to an internal otitis with otorrhœa, we may employ tepid injections, cataplasms, local bleedings and narcotics, but the latter must be only used when there are no symptoms of cerebral compression.

b. *Internal Pachymeningitis.*

This process and its sequences were not unknown to our predecessors (Baillarger, Pons, Oesterlen, etc.). The pathogenic relations of the sanguinolent effusions and false membranes found between the dura mater and arachnoid, were often wrongly interpreted, and Virchow first thoroughly recognized their anatomical character (Wuerz. Verh., Bd. VII., 1857).

Pathological Anatomy.

The internal surface of the dura mater is covered with a yellowish exudation, and strewn with ecchymoses; or a thin layer of dense fibrin is present which, with care, may be separated from the underlying membrane. At a later period, a very thin fibrous membrane, abundantly supplied with capillaries, forms at these spots, either on one or both sides of the brain, and especially at the convexity. In consequence of the duration and extent of the inflammation, numerous layers (10–20) are superimposed upon the false membrane. Delicate blood-vessels develop in large-meshed plexuses, which often rupture and give rise to hæmorrhages either between the layers of false membrane or between the latter and the dura mater (hæmorrhagic pachymeningitis of Virchow). These hæmorrhages, which vary in volume, being more profuse at the centre and thinning off at the edges, are united into circumscribed foyers (simple or circumscribed) which adhere more or less firmly to the adjacent dura mater and arachnoid, and constitute Virchow's hæmatoma of the dura mater. These rounded cysts contain blood or serous fluid in varying proportions, and are found especially at the convexity of the hemispheres; more frequently in the anterior and middle regions than posteriorly, and sometimes in the cerebral fossæ. The hæmatoma occupies either one or both sides, and causes cerebral compression. This prolonged compression leads to partial atrophy, softening and discoloration of the cortex, with thickening of the meninges.

Etiology.

Pachymeningitis is nearly always chronic and is usually found in old age. The male sex is more predisposed to its development than the female. Kremiansky (Virch. Archiv, B. XLII. pp. 129–321, 1868) and Neumann have shown, by experiments on dogs, that pachymeningitis may be produced artificially by chronic alcohol poisoning, in consequence of prolonged congestion and dilatation of the arteries. In man, also, chronic alcoholism furnishes the majority of cases of spontaneous pachymeningitis, and, according to Kremiansky, the process is usually situated at the vertex.

Injuries to the dura mater, caries and degenerations of neighboring parts are also causes of secondary pachymeningitis. It may complicate recurrent fever (Kremiansky), typhoid and typhus fevers, pleuro-pneumonia (especially during pregnancy), acute articular rheumatism, febrile exanthemata and puerperal diseases. It also occurs in the last stages of chronic diseases, such as tuberculosis, organic cardiac affections, diseases of the heart, diseases of the brain in the infant, and general paralysis of the insane.

Symptoms and Course.

The initial symptoms of the acute inflammatory period usually simulate meningitis. The symptoms are: periodical headache limited to one side, like migraine, and gradually reaching an extreme intensity; weakness of memory, apathy, somnolence, delirium, slow pulse, complete anorexia, constipation with considerable meteorism, and an instinctive tendency to clasp the hand to the affected side of the head.

In the *chronic forms*, which may last weeks and months, the symptoms begin slowly and are interrupted by remissions which may readily lead to error. Dull headache, weakness of the intellectual faculties, feebleness of the limbs, unsteady gait, imperfect and incoherent speech, and visible change in nutrition, prove the latent progress of an encephalic inflammation.

Recovery is possible, both in the acute and chronic stages, especially when the collection of fluid is of a serous nature. This fact has been demonstrated by a large number of positive clinical observations, as well as by autopsies, in which lesions of pachymeningitis were accidentally found several years after recovery therefrom, and when death had followed some other acute disease. In a case of Cruveilhier, the recovery was complete, and also in Goschler's patient (Allg. Wien. med. Zeit., N. 647, 1865), who died six years later of tubercular peritonitis with ascites and eclamptic attacks. The hæmatoma often becomes encysted (hygroma of the dura mater, of Virchow) or its contents undergo calcification (Rokitansky and Foerster).

When the disease takes a favorable turn, the symptoms of cerebral irritation subside, the mental faculties revive, and, little by little, recovery takes place. At other times, life is preserved, but the patient remains imbecile (Schuberg's case). The fatal termination is heralded by the progressive failure of the mental faculties and physical forces, loss of consciousness, and difficulty of deglutition and respiration. At other times, an apoplectic seizure occurs towards the last, and convulsions or partial contractures may precede the attack of paralysis. The gradual advance, and slight improvement followed by sudden relapses, are characteristic of hæmorrhages in the hæmatoma. According to Pons, chronic internal pachymeningitis begins with headache, difficulty of speech, absence of "stilted ideas," and with development of the symptoms of general paralysis of the insane upon one side of the body. The *treatment* in the acute variety must be chiefly antiphlogistic and derivative. In chronic cases we must endeavor to produce absorption and build up the general condition.

In severe apoplectiform seizures, the treatment is the same as in cerebral hæmorrhages, of which we shall speak later.

c. *Inflammation and Thrombosis of the Sinuses of the Dura Mater.*

The venous reservoirs known as sinuses, which receive blood from the meninges, the veins of the brain, the diploë of the cranial bones, and, in part, from the organs of special sense, are frequently exposed to disease.

Pathological Anatomy.

Inflammation of the sinuses is rarely primary, but more frequently follows lesions of the skull, and presents the symptoms and course of suppu-

rative or adhesive phlebitis. Usually the inflammation of the sinus, after bony suppuration, occurs from infiltration of its wall with pus, giving rise to necrosis. A purulent exudation fills the cavity of the sinus and causes the formation of obstructing clots and thromboses in the corresponding veins.

Inflammation of the transverse sinus may arise from inflammation of the cells of the mastoid process (Troeltsch); or from a perforation of the sinus with fatal extravasation within the skull and hæmorrhage through the external auditory meatus (Wreden). Phlebitis of the superior petrous sinus may give rise to the passage of pus into the vein of the mastoid petrous canal, followed by pachymeningitis and abscess of the brain (Troeltsch), and suppuration of the sinus may cause the formation of deposits at the base, between the arachnoid and pia mater, and be propagated thence to the jugular vein (Lebert). In a case of this kind recently observed, the brain was discolored and softened at the base, the dura mater adherent, and the sinuses enclosed in a pigmented cellular tissue, with destruction of the auditory bones. In Stokes's case (osteitis of the mastoid process and internal otitis) phlebitis of the petrous and cavernous sinuses was found, with basilar meningitis and softening of the superficial parts of the anterior and inferior portions of the right cerebellar hemisphere. Extension of thrombosis of the superior longitudinal sinus may cause inflammation of the veins of the pia mater at the convexity of the brain, and often gives rise to cerebral hæmorrhage (Rokitansky).

Etiology.

The most frequent causes of phlebitis of the sinuses are inflammations and purulent deposits within the osseous portions of the ear. Among forty cases of phlebitis collected by Lebert, there were nine of suppuration of the sinuses. In five cases, the disease of the sinus started from an internal otitis with caries of the temporal bone. But cases are known in which, without disease of the bones or of the cavity of the tympanum, the sinuses have become diseased from the proximity to other organs. An inflammation may be propagated by contiguity to the dura mater through the agency of the numerous canals in the temporal bone.

Inflammation of the sinuses is observed in injuries to the skull, when the dura mater has been contused in the neighborhood of the vascular canals, when it has been lacerated, or when splinters of bone have torn the sinuses. Spontaneous inflammation of the sinuses, with suppuration and pyæmia, have been observed in the puerperal state by Castelnau and Ducrest, and by Foerster. In erysipelas or anthrax of the face, the purulent infiltration of the cellular tissue of the cheek, as I have seen in one case, may be propagated, by the inflammation of the facial veins and ophthalmic vein, to the transverse and cavernous sinuses. Cachectic thromboses of the sinuses (especially the longitudinal and transverse sinuses) are met with in old age, in chronic cachexiæ, and in cholera infantum. In insanity, disease of the sinuses may develop, and, according to Guentz, it originates either in the general condition or in the local cerebral lesions.

Symptoms and Course.

The symptoms present a typhoid character. The patients complain of intermittent headache; the temporal region of the affected side is painful,

either spontaneously or on pressure; in otitis interna there are painful throbbing sensations in the deeper portions of the ear, distressing buzzing, otorrhœa (the discharge being often mixed with blood), and enfeebled hearing; sometimes an abscess forms in the mastoid process. In two cases of inflammation of the sinuses, Lebert saw, in the one, an abscess form within the orbit, with violent periorbital pain, and almost continuous corrugation in the frontal and palpebral regions; in the other case, an ulcerated keratitis, like that following section of the trigeminal nerve, occurred on the affected side. At a more advanced stage, the patients are seized with high fever, delirium or drowsiness, with vomiting, muscular tremor, partial convulsions, and subsultus tendinum. Despite the existence of these typhoid symptoms, the characteristic signs of typhoid fever, according to Lebert, are absent. Although well-marked stupor is present, we can rouse the patient and obtain correct responses. The evident loss of hearing, the discharge from the ear, the disease of the tympanum, and the other symptoms which we have mentioned, permit a diagnosis during life.

When the disease advances, an intense delirium alternates with the coma. Convulsions supervene with hemiplegia, inequality of the pupils (which are at first contracted, then dilated), and dysphagia. Finally, the fever and loss of consciousness increase, and then give place to chills and fatal collapse. When internal otitis exists, and convulsions and paralyses indicate serious cerebral disease, recovery is very rare. Some instances, however, have been published by Wilde, Lallemand, Canstatt, and Griesinger. The fatal termination is usually due to our inability to relieve the cerebral organs from the effects of the bony lesion and from the contact with pus.

Thrombosis of the sinuses frequently accompanies cerebral affections which are consecutive to diseases of the middle ear. In addition to convulsions and the symptoms of a localized lesion (facial paralysis, paralysis of ocular muscles, ptosis), the characteristic phenomena are those of a localized arrest of circulation within the skull, viz.: circumscribed cyanosis, and partial dilatation of the veins, of acute origin. In thrombosis of the transverse sinus (which occurs with the greatest frequency), when the process extends to the mastoid process through the posterior auricular veins, we sometimes observe, according to Griesinger (Archiv f. Heilk., 1862), as important diagnostic signs, a painful circumscribed œdema behind the ear, and a limited phlegmasia alba dolens. In inflammation of the cavernous sinus and thrombosis of the ophthalmic and facial veins, we find venous congestion of the fundus of the eye, ecchymoses of the conjunctiva, projection of the eyeball forwards, œdema of the eyelids and orbital region, or pseudo-erysipelas of the integument of the face. Change or disappearance of these symptoms of compression and of the arrest of circulation characterize thrombosis, according to Wreden, while their persistence appertains more to phlebitis. According to Gerhardt and Huguenin, the external jugular vein of the affected side is often less full than the other, in the cachectic thrombosis of the sinuses in children. In many cases, however, this symptom is absent, especially when the vascular canals are obstructed on the two sides, or when the flow of blood through the jugulars occurs in diminished quantity, but regularly. According to Wreden (Petersb. Zsch., Bd. XVIII.), who has collected 151 cases of diseases of the sinuses, the clinical symptoms accord with the experimental results obtained by Panum, Billroth, and O. Weber, and permit a differentiation between thrombosis and phlebitis of the sinuses.

In thrombosis (with intact condition of the wall of the sinus and

tendency of the thrombus to become organized, or to break up and become removed) fever is wanting; there is considerable stasis in the afferent vessels; hæmorrhages are frequent; inflammation of the brain and meninges is rare, and pyæmia does not occur. *Phlebitis* of the sinuses, on the other hand (with purulent infiltration of its walls and tendency to ulceration and perforation), is accompanied by high fever; there are few symptoms of stasis; the inflammation often extends to the brain and meninges; metastatic abscesses are common, and the disease usually assumes the character of septicæmia or pyæmia.

Thrombosis of the transverse sinus, following diseases of the ear, is often followed by recovery, which is confirmed by the early development of collateral circulation (Griesinger, loc. cit.). Nevertheless, the majority of cases terminate in capillary hæmorrhages or meningitis. In cachectic thrombosis of the sinuses, the prognosis is absolutely fatal.

Treatment.

In thrombosis of the sinuses connected with internal otitis, we must prescribe absolute rest from the beginning, antiphlogistic measures, and intestinal derivatives. The otorrhœa should be treated with lukewarm injections, and, when the opportunity is favorable, the mastoid process should be trephined. We must avoid, especially in children, the use of opiates and bleeding. In the first stages of the diseases, stimulants may, perhaps, be indicated.

d. *New Growths of the Dura Mater.*

Epithelial tumors, growths of varying consistence following chronic pachymeningitis, and the psammoma and osteoma of Virchow, possess merely an anatomo-pathological interest. Syphilomata of the dura mater (Wagner) usually accompany gummata of the pericranium, or analogous lesions in the cerebral substance or in the nerves. Cancer, which develops very readily in the dura mater, may be recognized in some cases during life.

I have seen, in Ulrich's surgical service, a woman, aged 70 years, operated upon for cancer of the left breast. Nine months afterwards she began to suffer from intense pain in the head, vertigo, a sensation of compression of the skull, with hyperæsthesia of the scalp. In the absence of all symptoms of irritation or paralysis, carcinoma of the dura mater and skull were suspected. The patient died two months later with icterus and delirium. At the autopsy, the left side of the skull was found thickened, ploughed by channels and infiltrated with an encephaloid substance. At the height of the superior longitudinal sinus, the dura mater was thickened and strewn with tumors, from which oozed an opaque fluid. Similar tumors existed on the inner surface of the middle cerebral fossa.

B.—Diseases of the Arachnoid.

Opacity and thickening of the arachnoid from repeated congestions are usually met with in old age. In a large number of autopsies made in Vienna upon suicides, considerable hypertrophy of the arachnoid and adjacent meninges was noted. The subjects, who belonged to the intelligent classes of society, had been afflicted during life with frequent cerebral congestions, periodical and intractable insomnia, melancholia, fixed ideas, and distrust.

Inflammation of the arachnoid, or arachnitis, is only revealed upon autopsy, by thickening and by the presence of false membranes or osseous plates upon the arachnoid and by its adherence to the dura mater. L. Meyer (Virchow, Archiv, 17. Bd., p. 209) has found epithelial products on the superior surface of the arachnoid, in cases of chronic cerebral irritation. Sometimes the Pacchionian bodies are transformed into connective-tissue products, stretching along the falx cerebri and even into the depressions on the inner surface of the skull. These are also the results of repeated hyperæmia and inflammatory exacerbations occurring in drinkers, epileptics, and in insane patients who are subject to attacks of excitement. Diffuse parietal arachnitis is observed in caries of the internal ear.

Arachnitis is always accompanied by meningitis, so that it is impossible to distinguish the two affections clinically.

Hæmorrhages into the cavity of the arachnoid are caused chiefly by exudations and inflammatory new formations of the dura mater. In many cases, however, meningeal hemorrhages may be produced independently of pre-existing false membranes. In new-born babes and infants, hæmorrhages of the pia mater or brain cause imbibition of the arachnoid with blood.

New growths (tubercle, cancer, syphiloma) do not originate upon the arachnoid, but start from the pia mater or dura mater.

C.—Diseases of the Pia Mater.

Congestion of the pia mater, which usually accompanies cerebral congestion, is found in acute forms of insanity and in many diseases of childhood, especially the exanthemata. They may prove fatal when they give rise to an effusion of serum into the subarachnoid space or to a hæmorrhage in the pia mater. Repeated hyperæmia causes thickening of the pia mater and arachnoid, and œdema and hypertrophy of the Pacchionian bodies. In old age, thickening and pigmentation of the pia mater with adhesions of its folds are frequently observed as consequences of previous hæmorrhages.

Inflammations of the pia mater (the meningitides) are of much greater clinical importance. The usual classification depends upon the character of the exudation and upon its chief situation. According to Rokitansky, purulent pseudo-membranous meningitis may attack different portions of the brain, but especially involves the convexity of the hemispheres; on the other hand, tubercular meningitis is limited to the base. But this distinction cannot be rigorously maintained. Tubercular meningitis sometimes extends to the Sylvian fissure and to the convexity of the hemispheres. On the other hand, severe meningitis of long duration may extend from the convexity to the base and even to the pons varolii and cerebellum. The different forms of exudation may also be combined in varying proportions.

From a clinical standpoint, it is advisable to distinguish three forms of cerebral meningitis. They are, 1, simple primary meningitis; 2, meningitis involving chiefly the base of the brain; 3, an epidemic form, in which the exudation is general and extends into the spinal canal.

1. Simple Meningitis.

Pathological Anatomy and Experimental Investigations.

In the *acute form,* the pia mater is strongly injected and infiltrated with a purulent exudation, especially upon the convexity of the hemi-

spheres towards their internal portions. In other cases, the inflammation is limited to the anterior lobes, to the fissure of Sylvius or to the median fissure.

The exudation is yellowish and thick, formed of pus globules and finely granular fibrin, and fills the subarachnoid space (according to Klob, the pus is furnished by the epithelium of the lower surface of the arachnoid). The exudation is often especially abundant along the vessels. In the severer cases, the inflammatory products follow the fissures to the base, and extend both anteriorly and posteriorly. According to Bednar, the cerebellar pia mater may be affected in very young infants.

The arachnoid is frequently opaque and covered with pus. The cortical substance is usually softened, discolored, infiltrated with pus. It is very frequently strewn with capillary hæmorrhages, and adheres so closely to the exudations which have formed upon the inner surface of the pia mater that it is often impossible to detach them without tearing the brain.

In the *chronic meningitis* of idiocy, general progressive paralysis, epilepsy and syphilis, we find thickening and fibrous changes in the arachnoid and pia mater, and adhesions in spots to the reddened cortex. According to L. Meyer (Centralb. f. d. med. Wiss., No. 8 and 9, 1867), progressive paralysis is characterized by the abundance of new-formed vessels in these membranes, and by endogenous proliferation of the nuclei of the medullary substance. According to Westphal, these lesions are also found in the meninges and substance of the spinal cord. When the inflammatory process is chronic, it causes atrophy of the cortical substance, and sometimes of the entire brain, the ventricles being dilated and filled with serum.

Foerster has seen cheesy degeneration of the exudation within the sulci and the formation of a cyst-wall of connective tissue.

The experimental data recently obtained, concerning the symptoms of increase of cerebral compression, should be taken into consideration in the discussion of the pathology of the meninges. According to the manometric researches of Leyden upon trephined animals (Virch. Archiv, Bd. 57, p. 519), and the experiments of Pagenstecher (Heidelberg, 1871) and of Jolly (Wuerzburg, 1871), upon cerebral compression, we should especially take into consideration the degree of pressure which the brain undergoes, and which is proportionate to the quantity of the injected matter which produces the compression. Although the experiments on animals do not furnish the diversity of symptoms observed in man, they are none the less instructive. Pain first appears and is manifested by groans and cries. Loss of consciousness is shown by stupor, somnolence, and complete coma; the spasms indicate irritation of motor portions.

Spasm of ocular muscles, inequality, and, in severe cases, dilatation of the pupils are observed. Increase of cerebral pressure first produces slowness, and then great acceleration of the pulse (irritation and paralysis of the vagus, Leyden). Respiration is also affected; it is at first irregular and more frequent, and later, especially in coma, it becomes slow and profound. In severe cases, the temperature is lowered until death. Clinical observation is in perfect accord with these results of experimentation on animals.

Etiology.

Simple primary meningitis (leptomeningitis or meningitis of the convexity) is not a frequent affection, if we exclude the epidemic exacerbations of certain years. It may occur at all ages, but, according to Bier-

baum (Die Meningitis simplex, Leipzig, 1866), it chiefly attacks infants under two years, and appears in the convulsive form more often towards the middle than towards the end of infancy. In childhood it is much rarer, and diminishes in frequency towards puberty. In youth and adult life, it again increases in frequency. At a more advanced age, the acute variety is infrequent, but the chronic form, especially if we include the insane, is much more common. It is more frequent in males than in females.

Simple primary meningitis follows cerebral irritation and concussion (wounds of the head, effects of cold, sunstroke, mental effort, violent excitement). According to J. Rosenthal, the basilar meningitis following a gunshot wound of the spine is due to escape of cerebro-spinal fluid, and to the concussion thus communicated to the base of the brain. According to Fischer, traumatic meningitis is due to irritation of the brain and its meninges by splinters of bone. *Secondary* meningitis appears in the vicinity of pachymeningitis, in cerebral syphilis (Griesinger), inflammation and thrombosis of the sinuses, caries of the cranial bones, internal otitis and *chemosis* of the eyeball (Leyden, Foerster).

Meningitis sometimes complicates other inflammatory diseases, such as bronchitis, pneumonia, pleurisy, pericarditis, acute exanthemata, erysipelas, and acute articular rheumatism. Finally, meningitis may appear in the course of Bright's disease, pyæmia, endocarditis, puerperal diseases, phlebitis, typhoid fever, dysentery, and carcinoma.

Symptomatology.

Meningitis usually begins with fever and cerebral symptoms. The patient complains particularly of a feeling of heaviness in the head and violent cephalalgia. The headache, which is either diffuse or circumscribed, is often intermittent, and develops so slowly that the patient attends to his occupation for a certain length of time, until the advent of fever, weakness, vomiting, and increased cephalalgia indicate the onset of a serious disease. In many cases, the headache increases very rapidly under the form of shooting pains. The patient becomes insensible, and often carries the hands to the head. In infants, there are frequent and sudden cries. Among the most common initial irritative symptoms, we may mention buzzing in the ears, photopsia, photophobia, and hyperæsthesia of audition.

Fever is soon added to these initial symptoms, and it is often preceded by chills, which may however be absent. There is a prompt rise in temperature and in the rapidity of the pulse, and these maintain very high figures (Wunderlich, Rosenstein, and myself) for a long time (pulse, 120–130, and more; temperature, 40°–41° C.). The respirations may be increased to 30–40 per minute.

From clinical observation and the result of autopsies, the fever is found to correspond to the intensity and extension of the exudative process, and the variations of the pulse correspond with analogous variations of temperature and respiration. Consciousness and the intellectual faculties are more or less disturbed from the beginning. Restlessness, irritability, slowness of ideas, difficulty of speech, tendency to apathy, somnolence and delirium, precede the loss of consciousness. Strabismus and partial rotation of the eyeball are not infrequently observed. The pupils are usually contracted at first, or they are unequal, with frequent variations on either

side. Later, in severe cases with fatal termination, they remain dilated and become insensible. Frequently there are no characteristic symptoms on the part of the pupils.

As disturbances of sensibility, we may mention, in addition to the cephalalgia, a characteristic cutaneous hyperæsthesia. In patients already comatose, we may observe the production of pain and increase of reflex excitability, when the hand is passed over the surface of the body. According to Trousseau, erythematous tachês are produced when the skin of infants, affected with meningitis, is lightly touched. The irritative motor symptoms are as follows: contraction of the muscles of the neck, convulsions of the upper and lower limbs, more rarely, general convulsions (except in infants), tonic or clonic contractions of the muscles of mastication (mumbling, grinding the teeth, trismus—especially in children); finally, subsultus tendinum, and tremor of the hands. In young infants, when the open fontanelles are distended by abundant effusion, pressure upon the fontanelles may produce convulsions. Paralyses appear with the increase of cerebral pressure. They are usually localized, affecting chiefly the facial muscles, and more rarely those of the extremities. Well-marked and complete hemiplegia, paralysis of the sphincters, preceded by retention of urine (often by intense albuminuria, according to Rosenstein), are rare symptoms which appear when the exudation is complicated with cerebral œdema. Towards the last, the paralyses and coma become more profound.

The *pulse*, full and rapid at the onset, becomes irregular and compressible at a later period. When the cerebral pressure increases, it is markedly slowed, and then again becomes frequent, and in the last stages it is extremely rapid (140 or more). The respirations are also very much accelerated (60 or more).

The *duration* of the disease is very variable. In slight cases, in which the diagnosis is always doubtful, the serious symptoms may rapidly mend But more frequently, when the termination is fatal, the duration of the disease is short. In adults, it may terminate during or at the close of, the first week, and in infants, within a few days. In certain cases, death only occurs after two or three weeks, or even after several months, if the meningitis has become chronic.

Diagnosis and Prognosis.

In the absence of well-marked initial symptoms, it is hardly possible to arrive at a diagnosis. But, on the other hand, the sudden appearance of cerebral symptoms in the midst of health, and their violence and rapid increase, often allow a very early diagnosis. This is also true of meningitis due to cerebral irritation or concussion, as well as of the secondary form which sometimes follows caries of the cranial bones or otorrhœa. In these latter cases, however, experience teaches us that inflammatory processes in the meninges, even when they appear to be of a serious nature, may undergo prompt retrogression. Meningitis is not readily mistaken for violent gastro-intestinal irritation, broncho-pneumonia, or the outbreak of acute exanthemata. Typhoid fever is recognized by the characteristic appearance of the tongue, by the regularity of the pulse and respiration, and by the diminution of cutaneous sensibility. The diagnosis becomes very difficult, and even impossible, when, as happened in Loeschner's and Steiner's cases, meningitis develops during the course of typhoid fever.

Cerebral hyeræmia is distinguished from meningitis by the absence of premonitory symptoms on the part of the mental faculties. We do not find either the rapid increase of fever (which is frequently ushered in by chills) or convulsive movements tending to become generalized, the remarkable pallor of the face, or the distortion of the features found in cases of meningitis. The differential diagnosis between simple and tubercular meningitis will be discussed at a later period. The meningeal form of cerebral rheumatism is not characterized either by vomiting or convulsions. It is recognized by the coexistence of polyarthritis, and by the disappearance of the latter when the cerebral symptoms become marked; later, psychical disturbances become manifest, especially melancholia. In a case which I have published (Wochenbl. d. Ges. d. Aerzte, 17 and 18, 1863), a profound state of melancholia developed during acute articular rheumatism. Following this condition, I observed complete abolition of electro-muscular contractility and sensibility in the lower limbs, which disappeared very slowly during convalescence, after the resorption of the intracranial exudation and the removal of the cerebral œdema. Eclampsia of children differs from convulsive meningitis (which occurs most frequently in them) by the usually shorter duration of the convulsions ($\frac{1}{4}$–$\frac{1}{2}$ hour), by the more frequent remissions during the day (if the disease, as it does rarely, continues longer than a day), and by the rapid course of eclampsia. The existence of an accidental cause, of an hereditary disposition, or the periodical return of the affection, are elements in diagnosis. Uræmic encephalopathy is distinguished from simple meningitis by the dropsies which usually precede the cerebral symptoms for a long time, and by the presence of blood and albumen in the urine. Its course is more rapid, and there is no fever or acceleration of the pulse. According to Voltolini (Monatsschrift f. Ohrenheilkunde, No. 1, 1867) acute inflammation of the labyrinth in children may assume the characters of meningitis. In fact, inflammation of the labyrinth is accompanied by loss of consciousness, vomiting (obtained experimentally by Czermak in lesions of the labyrinth), delirium, and fever. We are aided in the diagnosis of these cases, by the rapid course and unusual violence of the symptoms, by the absence of paralysis, by the loss of hearing, coexisting with an intact condition of the external ear and tympanum, and followed by loss of speech and deaf-mutism.

The *prognosis* of meningitis depends upon the symptoms and severity of the affection. Simple primary meningitis is followed, in a very small number of cases, by recovery, as is shown by the traces of inflammation found upon the meninges after the lapse of several years.

The proportion of recoveries is greater in adults than in nurslings and young infants. They are much more frequent in the period of irritation than in that of exudation. Complications render the prognosis more grave. It is unfavorable in the secondary forms, except in rare exceptions.

The favorable termination is ushered in by a rapid lowering of the temperature and pulse, by a deep sleep, and by the return of consciousness. Violence and continuance of delirium, persistence of profound coma, continuous elevation of temperature to 41° C., and progression of the symptoms of paralysis, lead us to give an extremely grave prognosis. Towards the close of life, when the vagus centre begins to be paralyzed, the pulse increases in frequency, the temperature reaches the highest degree ever observed during life (as high as 42.8° C., according to Rosenstein), and after death it may mount beyond 43° C. The fatal termina-

tion is hastened by coexisting inflammations of the cortex, by ventricular dropsy, by the continuance of the exudation, and, more rarely, by effusions between the meninges.

As more remote sequences of meningitis, we may mention fibrous thickening of the pia mater and arachnoid, adhesions of the latter to the atrophied cerebral convolutions, to the dura mater and even to the skull.

These inflammatory lesions, which are usually referred to *chronic meningitis*, are found in epilepsy, acquired imbecility, and general paralysis of the insane. Judging from the periodical rise of temperature observed in this disease by L. Meyer, and from the cortical encephalitis frequently noted by Meschede and Mettenheimer, it is extremely probable that the anatomical lesions attributed to chronic meningitis are due to an inflammatory process, which is nearly always latent, and to febrile exacerbations, which are widely separated and are therefore often misconstrued. To the stage of irritation belong the headache, vertigo, convulsions, hallucinations and cerebral excitability. The stage of depression is characterized by the failure of the mental powers, the disturbances of speech, and the various changes in sensation and motion. When the disease lasts several years, the secondary lesions may extend to the cord, and, according to Westphal, the spinal lesion is primary in many cases.

Treatment.

In the beginning, we should employ all those measures which tend to diminish the pressure of the blood in the vascular system. The patient should be immediately placed in a cool, dark, quiet spot, and the head be placed as high as possible and subjected to cold applications, preferably in the shape of an ice bag. In the first few days, local bleedings are also indicated (leeches to the temples or mastoid processes): the amount of blood to be withdrawn varies with the constitution and vigor of the patient. Often, after the bleeding, it is advisable to employ cold compresses, which should be interrupted when signs of syncope appear.

Cool ablutions and affusions are strongly recommended by Trousseau, and Barthez and Rilliet. Cool affusions in a warm bath, cool baths, or, better still, the use of wet blankets several times a day, lower the temperature 1° or 2° C. But it again rises after a few hours, and the hydropathic measures must be repeated methodically and for a long time, at the beginning of the febrile exacerbations. They often exercise a good influence, but do not succeed in preventing a fatal termination in severe forms.

Mercury is still employed from time to time in children. It is used in the form of frictions to the neck or sub-maxillary region, or calomel is administered internally in large doses. The calomel is said to cause derivation to the intestines and biliary passages, diminish the amount of blood passing to the brain, and retard exudation. Infants, it is also said, tolerate the use of calomel better than adults. All these statements are without any positive, scientific basis. Large doses of calomel not only produce salivation with buccal and pharyngeal ulcerations, or diarrhœa followed by enteritis, but they are also incapable of diminishing the amount of blood flowing to the brain. Less can be said against moderate doses (.020–.05 every three hours), but no benefit is positively known to follow their employment.

Derivate measures are much more important. In the stage of excitement, employ sinapisms or blisters to the trunk or extremities. The ap-

plication of tartar emetic ointment to the head of infants who are hopelessly ill, merely increases their sufferings. Intestinal derivation is produced by irritating enemata and purgatives, but emetics and drastics should not be employed.

Narcotics should be reserved for those cases in which convulsions are a serious symptom, or in which a maniacal form of delirium supervenes, or when the serious symptoms have resisted cold applications, bleeding and derivatives. In these cases, small doses of opium or morphine (some prefer aconite or belladonna in infants) may diminish the symptoms of excitement and cause a favorable termination. (According to Gscheidlen and Mendel, the use of opiates in animals lowers the temperature of the head.) It goes without saying that drowsiness, coma, and collapse are contra-indications to the employment of opiates.

When there is a tendency to syncope, we should employ active stimulants, such as cold affusions to the head during a warm sitz-bath, the use of musk, ammonia, and camphor. When a bath with affusions does not promptly restore the patient, we may recommence the operation, but the chances of success are then reduced to a minimum.

In *chronic meningitis*, the use of tonics, iodide of iron, a prolonged sojourn in the country and appropriate regimen, are of considerable benefit. Friction with wet cloths and cool baths also tend to restore the strength. If paralysis persist, the electrical current should be applied.

2. Basilar Meningitis.

Basilar meningitis presents distinct characteristics which must be examined with care in order to differentiate it from closely allied conditions. We may distinguish, from a clinical point of view, the following varieties: First, simple basilar meningitis (circumscribed or diffuse), with its peculiar symptoms and anatomical lesions; secondly, the tubercular forms, embracing tubercular basilar meningitis, properly speaking, and acute meningeal tuberculosis of the hydrocephalic type.

a. *Simple Basilar Meningitis.*

Meningitis may give rise to localized inflammatory products at the base of the brain. In two cases of this nature which I have observed, traces of chronic meningitis were found limited to the base, with adhesions of the meninges and compression of the oculo-motor nerve (which, in one subject, was discolored and atrophied upon one side). During life, the patients suffered from continuous headache, dizziness, ptosis, paralysis of ocular muscles, paresis within the distribution of the facial nerve, and, in one case, paresis of the left lower limb. The irregularity of the ocular paralyses, their disappearance (either spontaneously or after the use of galvanism), the slight affection of the facial or trigeminal nerves, the more or less complete integrity of motion of the limbs, are factors in the diagnosis of localized inflammatory processes at the base of the brain.

But when basilar meningitis is more diffused and involves several cerebral nerves, and when there is considerable difficulty of motion in the limbs, it is hardly possible to distinguish meningitis from a tumor at the base. In a case observed by Benedikt, the meninges were adherent from the sella turcica to the foramen magnum, and several nerves of the base

were enclosed in retracting connective tissue. During life, the patient presented the appearances of labio-glosso-pharyngeal paralysis. In an analogous case of Graefe (paralysis of the common and external motor-oculi, and of the pathetici, without pain or fever, but with dysphagia and dyspnœa at the end), Virchow and Klebs discovered, at the autopsy, an osteo-periostitis at the base of the skull.

Basilar meningitis is more frequently diffused and general. The base of the brain is then covered in great part or even entirely by a purulent exudation which fills the subarachnoid space from the optic chiasm to the pons varolii and medulla oblongata. The cerebral fossæ also contain a large quantity of fluid, and the ventricles are distended by an opaque, flocculent, purulent or pseudo-membranous effusion which may measure 100–150 gr. The ependyma is thickened or softened, and the choroid plexuses are infiltrated with exudation or strongly congested, and the surrounding cerebral tissues and commissures are softened and œdematous. The cortical tissue in contact with the exudation is sometimes discolored or of a red hue, and strewn with small hæmorrhages. The convexity of the brain is usually free from exudation. When the ventricular dropsy is considerable in extent, the convolutions are flattened and pressed against one another. Here and there we find traces of past inflammation in the form of false membranes, thickening and adhesions of the meninges.

Basilar meningitis may be primary, but it more frequently follows other cerebral affections, such as lesions of the cranial vault, fissures at the base of the brain, phlebitis of the sinuses, tumors, localized inflammations, abscess and softening of the brain. The primary disease often masks the symptoms of the secondary meningitis. In some cases, the latter may manifest itself by violent headache, stiffness of the neck, retraction of the abdomen, slow development of the disease with moderate fever, and by the appearance of paralysis of several cerebral nerves in addition to the other signs of meningitis.

b. *Tubercular Forms of Basilar Meningitis.*

Clinical observation has not been able to draw any distinction between the anatomically distinct tubercular basilar meningitis and miliary tuberculosis of the pia mater. Anatomically, tubercular basilar meningitis develops in the midst of an inflammatory process. In the gelatinous or yellowish exudation occupying the subarachnoid space at the base of the brain, tubercular granulations are found upon the meninges and sometimes extending towards the convexity and Sylvian fissure. The cerebral tissue in their vicinity is softened and discolored. This exudation is absent in acute miliary tuberculosis of the pia mater. The tubercles appear as small grayish granulations, which are often seen only after careful inspection of the pia mater at the base, the membrane being examined in transmitted light after detaching it from the brain. Similar granulations may also be seen in the fissures of the convexity between the cerebral convolutions, and upon the choroid plexuses. According to Weisbach (med. Yahrb., Bd. XVI., 1868), the proportion of water in the brain is very much increased both in simple and tubercular meningitis. According to the latest researches of Bastian (Edinb. Med. Journ., April, 1868), the granulations in tubercular meningitis are due to a proliferation of the nuclei contained in the walls of the hyaline vessels of the pia mater. In Magnan's case (Gaz. Med., No. 15, 1870), in which epileptiform attacks, irregu-

lar muscular contractions and fibrillary spasms were present, the autopsy proved the existence of a tubercular affection of both the cerebral and spinal meninges. Liouville has observed the same fact in tubercular meningitis of childhood.

The numerous transitions which exist between the two forms of tuberculization of the meninges, their extension from the base to other parts of the brain, the tuberculization of other organs occurring in both varieties, and their analogous appearance in the ventricles, prevent us from discriminating clinically between them, and we are obliged to describe their pathognomonic symptoms under one category. We prefer the term tubercular meningitis to that of acute hydrocephalus, which is so frequently employed, because this only comprehends a part of the symptomatology of meningitis and because it may exist in many affections entirely foreign to tuberculosis.

Etiology.

Tubercular meningitis, according to the concurrent observations of Bennet, Rilliet and Barthez, is rare in the first years of life. It is most frequent from the second to seventh years, and then diminishes in frequency, occurring very rarely after the age of ten. In the adult, it occurs with the greatest frequency from the twentieth to fortieth years, and is more frequent in the male sex. Large densely populated cities exhibit a greater mortality than small towns and rural districts. The disease is more frequent in bad seasons of the year, but it never appears as an epidemic. Heredity is of importance in many cases, but the tubercular diathesis may also be acquired. Poor hygienic conditions favor its development. Children who are not nursed at the breast, receiving instead defective and insufficient nutriment, and who live in damp lodgings, deprived of air and light, and remain pale and lean, often have glandular enlargements and carry the germs of tuberculosis even when their general condition does not, at first, lead to any suspicion of danger. When the constitutional affection is thus developed in a latent manner, the cerebral symptoms may appear suddenly. Their outbreak may be incited by slight causes, such as toothache, cold, and excitement. It also occurs in certain debilitating diseases, such as whooping-cough, diarrhœa, measles, and scarlatina, which incite the proliferation of the tuberculous germs situated in the brain. Chronic exanthemata of the scalp and skin, and otorrhœa, whose sudden disappearance is regarded as a cause of meningitis, are rather the first manifestations of the dyscrasia. In adults, there are other causes of debility. Precocious and excessive mental labor, physical fatigue, prolonged sorrow, and the material cares of life may give rise to hydræmia, disturbances of cerebral circulation, and to congestion of the venous plexuses followed by serous effusion. Similar phenomena occur in other organs, as in the lungs and their serous coverings.

Symptomatology.

The symptoms of tubercular meningitis present numerous varieties, depending upon the age, previous constitution, and method of development of the exudation. The symptomatology varies according as the brain was previously intact or already affected by congestions and effusion. The exudation may develop suddenly or slowly. Great importance must

be attached to the lesions which the exudation or serous effusion produce in the cerebral tissues by inflammatory irritation, by mechanical compression or by softening.

The disease may begin suddenly, especially in adults, with fever and cerebral irritation. But usually, in the majority of cases, the cerebral symptoms are preceded for a long time by a condition of malaise, a feeling of constriction in the head, disturbances of digestion, sleep, and general nutrition. These phenomena are probably attributable to the slow progress of the meningeal exudation.

The pathognomonic symptoms only appear distinctly when the disease begins acutely. The cephalalgia of the onset is sharp, paroxysmal, and presents intermissions. It is usually situated in the frontal region, and it is often accompanied by dizziness, not only while walking, but also during the recumbent posture. The features are changed, the brow contracted, gaze fixed, especially in infants, the expression dull, and the pupils more frequently contracted than dilated. Speech is markedly embarrassed and slow, intelligence is disturbed, and there is a tendency to sleep. Among the initial symptoms, we must also mention vomiting, which occurs more or less frequently even in the first few days; marked hyperæsthesia of the skin and senses (unusual sensitiveness to light and sound); obstinate constipation, with distention and sensitiveness of the stomach; scanty urine. The fever is moderate in the beginning. The pulse is slightly accelerated as a rule, but in adults it may be retarded and intermittent from the beginning. In infants, according to Rilliet, the pulse is always wiry. The temperature usually varies between 38.5° and 39.5° C., but in acute tubercular meningitis it may suddenly rise to 40° or 41° C., and remain at this point for a long time. Respiration is very slow and superficial in children, and often irregular and interrupted by deep sighs.

While the disease is developing slowly and irregularly, deceptive appearances of recovery often occur. The psychical faculties change more and more, somnolence and delirium become more frequent, and alternate with grinding of the teeth and strabismus; the eyelids are half closed, the eyes directed inwards and upwards; the patient bores his head into the pillow, in consequence of tonic spasms of the neck, and gives utterance to repeated cries. The hydrocephalic cry of Coindet, regarded as characteristic of infantile tubercular meningitis, is also observed in other acute diseases, but it has great diagnostic significance when preceded by spasms or followed by convulsions.

The pupils at this time are more often dilated than contracted. As Cohnheim, and later, Bouchut, have shown, we can recognize, with the ophthalmoscope, certain varicosities of the retinal veins, retinal hæmorrhages, a serous peri-papillary infiltration (signs of obstructed circulation at the chiasm), and white miliary granulations on the choroid and retina. It is undoubted that in many cases, when a cerebral or pulmonary affection of doubtful character exists, the determination of tubercles in the choroid will render the diagnosis certain. But the absence of choroidal tubercles and of retinal lesions does not disprove the existence of tubercular meningitis. According to Galezowski (Union Med., 1867), ocular disturbances are absent in tubercular meningitis if there are no granulations upon the chiasm.

Digestion is poor, and the appetite usually disappears from the second week of the disease. There is great thirst, the tongue and buccal mucous membrane are dry and cracked, and the abdomen is retracted in the shape of a boat (contraction of the intestines from irritation of the centres of in-

testinal innervation, Traube). The pulse, at first a little accelerated and irregular, soon becomes markedly slower (irritation of the centre of the regulating cardiac nerves). It beats 40–45 per minute in the adult, and 60–65 in children. The respirations become slower, irregular, and superficial. The skin is cool, and covered with a cold sweat.

In the last period, or *stage of paralysis*, the paralyses of the muscles of the face, eyes and limbs, which were at first limited and transitory, become more complete and persisting, and certain muscles (jaw and neck) become contractured. Cutaneous and reflex sensibility, which were at first increased, become entirely lost. The pulse, small and soft, is very rapid (as in section of the pneumogastric, Traube); the abdomen is tympanitic; the bladder and bowels are evacuated involuntarily; deglutition is impossible; the skin is cold and discolored, and covered with a viscid sweat; the patients sink into coma, which, in children, is often interrupted by convulsions.

The duration of the disease should be dated from the appearance of the first well-marked cerebral phenomena. The uncertain prodromata should not be considered in the evolution of the symptoms.

The average duration is from two to three weeks. If the disease is superadded to general tuberculosis, it terminates in the first week, rarely in the second. But if tubercular meningitis attacks individuals who appeared to be previously healthy, it may last several weeks, sometimes even two or three months, or may terminate in a few days.

Diagnosis and Prognosis.

Vague prodromata, consisting of general malaise without noteworthy cerebral disturbances, will not lead us to suspect a tubercular affection of the brain, especially when the patient has previously enjoyed good health. In such cases, we cannot make a diagnosis except after a long-continued observation, or after the appearance of serious cerebral symptoms and constitutional disturbances, such as persistent headache, vomiting, fixed look, somnolence, and obstinate constipation.

Acute pulmonary and bronchial affections may be recognized by careful and repeated physical explorations. The differential diagnosis offers great difficulty when there is a simultaneous tubercular affection of the lungs and meninges, and we must be aided, especially in children, by the history of similar disease in the parents or other relatives. The symptoms of cerebral excitement which precede the eruption in acute exanthemata, or which, especially in children, are due to gastric irritation or the presence of worms, do not give rise to any doubt after the lapse of a few days, when the eruption has appeared or the worms have been passed, after appropriate remedies.

The diagnosis between typhoid fever and tubercular meningitis is usually not difficult. The former is characterized from the beginning by the increasing elevation and exacerbations of the temperature, and the acceleration of the pulse; lively delirium is rarer and appears later; the abdomen is usually tympanitic, and diarrhœa is almost always present; if constipation exists, it is readily overcome by laxatives; in the coma of typhoid fever, the patient is insensible to impressions upon the skin and senses.

In tubercular meningitis, on the other hand, the temperature is usually moderately elevated, the pulse and respirations slightly accelerated, and

furious delirium sometimes appears in the second week. Other characteristic signs are furnished by the "boat belly," the obstinate constipation, the repugnance of the patient to external impressions, and the secondary slowing of the pulse and respiration.

The differential diagnosis between simple and tubercular meningitis is determined by the aggregate of the symptoms. Simple meningitis attacks adults and infants in good health, and has very few prodromata, or complicates other affections which have no relation to tuberculosis. It begins with rapid rise of temperature, acceleration of the pulse and respirations, and somnolence; delirium and convulsions soon appear. The constipation is moderate, and the appearance of the abdomen is not modified. The situation becomes more and more grave, and terminates in a period varying from several days to a week. Tubercular meningitis usually attacks adults who have previously presented evidences of tuberculosis, or feeble infants with a scrofulous or tuberculous diathesis, either hereditary or acquired. The meningeal affection commences with slight rise of temperature and acceleration of pulse and respiration. Somnolence and delirium only appear at an advanced stage of the disease, and convulsions towards the close. The constipation is obstinate, and the abdomen is boat-shaped. The disease does not appear to be serious in the beginning, and may continue for several weeks, with deceptive appearances of improvement.

The *prognosis* of tubercular meningitis is very grave. In some cases, in which the exudation is not abundant, it may be absorbed and the miliary granulations of the meninges may disappear, as has been seen in tuberculosis of other organs. But this favorable termination forms a very rare exception, and does not diminish the gravity of the prognosis. The large proportion of recoveries reported by former observers (Formey and Goelis) are open to doubt as to the accuracy of the diagnosis. More modern authors, such as Hahn, Rilliet and Barthez, have seen the convulsive symptoms disappear at the end of several weeks, the pulse recover its force, and sleep restored after a profuse sweat and alvine evacuation. But these observations do not convince us of a permanent recovery, since relapses occur after these remissions.

Prof. Politzer has seen one case of recovery from basilar meningitis (Jahrb. f. Kinderheilk., VI., 1863). The child remained very emaciated, and died in a relapse three years later. At the autopsy, recent meningitis was found at the base, and an old exudation was observed upon the pons varolii.

Tubercular meningitis usually terminates in death, which occurs with irregular action of the heart and respiration, and with convulsions and coma. Idiocy, epilepsy, and chronic hydrocephalus may follow tubercular meningitis of childhood.

Treatment.

Since treatment is of little avail when tubercular meningitis has become established, therapeutic measures should be chiefly directed against the first manifestations of the dyscrasia. A rational prophylactic treatment, begun early and continued methodically, may succeed, in quite a number of cases, in checking the germs of scrofula and tuberculosis, and in preventing the occurrence of meningitis. Infants with an hereditary predisposition should be provided with a good nurse and reared in the country for a long time. It is well to habituate them early to cool baths; they should

have sufficient clothing without enervating the body; the sleeping apartments should be large, cheerful, and not too cold; excitement should be avoided if the children are impressionable, and their mental faculties must not be stimulated too rapidly. These precautions, together with the use of iron and cod-liver oil, give positive beneficial results in a considerable number of cases.

When the meningitis has already begun, the treatment is purely symptomatic. On account of the dyscrasic nature of the affection, antiphlogistic measures must be employed with caution. This is also true of leeching, the prolonged employment of which favors serous transudations. Local applications of ice or cold affusions are not indicated, and frequently renewed cold compresses satisfy all the indications. Active cutaneous revulsives may be applied to the neck and extremities, but it is useless cruelty to shave the infant's scalp in order to apply a blister or tartar emetic ointment.

Derivation to the intestines may be established by irritant enemata and saline purgatives, but we must not produce a debilitating diarrhœa. The use of iodide of potassium and iodide of iron is more extended. Niemeyer states that he has obtained gradual recovery by the long-continued use of iodide of potassium, pushed until toxic effects were produced. Other observers are less positive in affirmative statements in this regard. When the first signs of collapse and coma appear, the stimulants and tonics, to which we have referred under meningitis, should be employed. If the disease is prolonged, the patient should be kept absolutely quiet, the air of the sick-room should be frequently changed, and we should administer nutritious but easily digested food, combined with a small quantity of wine.

3. Epidemic Cerebro-spinal Meningitis.

This form of meningitis attacks, by preference, the pia mater of the brain and cord. The affection has already traversed a portion of Europe for several decades, as Hirsch, of Berlin, has positively proven, and its recent epidemic appearance has given rise to numerous observations and researches. All the epidemics present great differences with regard to the intensity, number, and duration of the cases.

Pathological Anatomy.

Let us first examine the changes in the central nervous system.

The meninges and sinuses are usually strongly congested, and the arachnoid is dry, injected, rarely infiltrated with fluid, and still more rarely covered with a plastic exudation, causing adhesion to the dura mater. *The real seat of the purulent meningitis is in the pia mater.* In very recent cases, this membrane is merely injected, but later, it becomes opaque, roughened, and covered with a gelatinous, transparent exudation, which sometimes presents a milky appearance, and is tinged with blood (Hirsch). Thiersch has found fœtid pus in the meninges. The exudation is sometimes so abundant that the arachnoid is completely detached. While Niemeyer considers the vascular sheaths as the sole source of the pus, according to Buhl, the cerebral substance is also softened, infiltrated with granular corpuscles and amyloid bodies. Merkel, of Nuremberg, has found in the ependyma and adjacent portions of the brain, and upon

transverse sections of the meninges and cortical substance, *a nuclear proliferation in the vessels, extending from the cerebral meninges to the spinal cord.*

The brain as well as the cord sometimes increases in volume, causing the convolutions to appear obliterated and dry. Sometimes the portions of the brain in contact with the exudation are softened (mechanical softening of French authors). It is rare to find pus within the ventricles. Klebs supposes that pus may enter the ventricles along the velum interpositum, by following the prolongation of the plexuses, or along the cerebellar choroid plexuses (Virch. Arch., XXXIV. Bd., 3 Heft, 1866). The pus is especially abundant at the base in the subarachnoid space, between the infundibulum and pons varolii, around the optic chiasm and medulla oblongata, and in the parietal region. It is found in less abundance along the convexity. In places in which the pus is not collected in considerable quantity, but in which the pia mater is opaque and roughened, according to Klebs, a more or less abundant cellular proliferation is noticeable. According to him, the process at the base of the brain is primary, and that in the cord is, at the most, synchronous with it, but will remain latent until the spinal arachnoid is invaded by the pus.

The posterior surface of the cord is more affected. The spinal dura mater often takes part in the process, a fact which is of rare occurrence in the brain. At first, recent, delicate false membranes are found on the dura mater, and, at a later period, very delicate adhesions develop between the dura mater and pia mater. These adhesions are found, especially on the posterior surface of the cervical and dorsal regions. In acute cases, according to Klebs, the subarachnoid exudation is viscid, fibrinous, yellowish or greenish, and it is crowded with round, granular cells containing a single nucleus. It contains mucin and albuminate of soda.

The most profound changes are found in the inferior cervical and lumbar regions. The layers of pus are sometimes irregular, and appear reticulated, and according to Klebs, their distribution, especially in the cord, depends upon the action of gravity. The largest quantities of pus are found in the dependent portions, and above any obstacles which bar its passage. We must take into consideration the spontaneous movements of pus globules demonstrated by Recklinghausen, and their passage through the intact walls of the capillaries shown by Cohnheim. The greater mobility of certain portions of the spinal column prevents any considerable collection in these places. Klebs has observed two kinds of change in acute purulent meningitis; viz., those of purulent encephalitis, properly speaking, and those of consecutive softening. The first is frequent in the white substance of the brain. It consists of small extravasations, usually in the sheaths of the arteries, with white or yellowish softening of the neighboring parts. The other variety of softening consists in an œdema of the white substance of the cord, more pronounced in the posterior columns, and especially in those places in which the pus has collected in the largest quantity. This softening plays the chief part in the symptomatology of the disease.

In a case cited by Klebs, the pus was disseminated, and since mitral endocarditis coexisted, we are led to suspect an embolismic origin of the spots of encephalitis.

Other organs are also often affected in epidemic meningitis. We may find broncho-pneumonia; bronchitis, acute pulmonary œdema, pleurisy, and pericarditis are rare; Buhl has found beginning fatty degeneration of the heart. The spleen is usually small; but, in some cases, it is enlarged at

the beginning. Klebs has found slight changes in the liver and kidneys, (granular, albuminoid or fatty degeneration, affecting chiefly the secretory elements). The viscera are rarely increased in size, but are usually softened. The intestinal mucous membrane presents catarrhal thickening, with enlargement of the agminated glands, which project like millet seeds above the mucous membrane, and present ulcerations in some places. The lymphatic glands nearly always present a reddish appearance.

The muscles are dry, soft, brownish-red, and atrophied. In recent cases, the muscular fibres present fine granulations. The cardiac muscle presents the same changes as the voluntary fibres. Klebs has found the condition of the blood very variable; in rapidly fatal cases, it is fluid, and the clots are soft and scanty. The blood contained in the vessels was dark, as in typhoid and typhus fevers. The ventricular fluid contained chloride of sodium, phosphate of soda and ammonia, and a large proportion of oxalate of urea (Meschede). Petechiæ and rubeolar eruptions are often found on the skin.

Etiology.

Defective hygienic conditions exercise an undoubted effect upon the development of the disease. Of 47 epidemics occurring in France, Hirsch attributes 46 to the military population. It raged with the greatest violence among the armies in the Netherlands, Russia, and Spain, and the soldiers were more affected than the officers. The crowded barracks, the accumulation of animal products and vegetable detritus, caused the production of the miasms which determine the appearance and rapid extension of the disease. In Sweden, the greatest mortality occurred among the filthy, crowded houses and narrow streets of the poorer classes. The better classes of society enjoy a remarkable immunity in nearly all countries. The mortality among infants, in certain epidemics, is due to other accidental causes, perhaps to the action of miasmatic emanations upon the impressionable organism of the child. Winter (in which all antihygienic factors are at their maximum) and spring (at which time organic decomposition is most active) furnish the greatest proportion of cases of this disease (about $\frac{3}{4}$ of the whole number).

The evacuation of the barracks is almost always followed by the disappearance of the epidemic. Better ventilation, cleansing, or evacuation —at least partial—of crowded tenements, and closure of schools, have promptly diminished or arrested the epidemic progress of the affection. All these facts prove that the disease is of an infectious nature, but opinions differ widely as to its contagious character.

Childhood and middle life are most subject to the affection. Rudnew and Burzew state (Virch. Arch., XLI., 1 Heft, 1867) that in Russia individuals of fifty and sixty years are often subject to the disease. The greater mortality observed among negroes in America is due less to a predisposition than to unfavorable hygienic conditions.

Symptomatology.

Epidemic cerebro-spinal meningitis, also called apoplectic or cerebral typhus, encephalo-rachidien meningitis, cerebro-spinitis (Chaussard), and epidemic spasm of the neck, presents a great diversity in its sympto-

matic forms. Even in the prodromic period, certain disturbances are no ticeable in the cerebro-spinal system. The patients complain of headache, dizziness, numbness in the limbs, tension and distressing stiffness in the neck and limbs, occasional chilly sensations and flashes of heat. These first symptoms may be so slight as to escape observation, or of such short duration that they almost coincide with the real onset of the disease.

It usually begins during the night with high fever, a violent chill followed by burning heat, intense headache and vomiting. To the ordinary symptoms of cerebral hyperæmia (turgor, hallucinations, delirium, strabismus, tremor of the limbs) are soon added tetanic spasms of the muscles of the neck and back, with considerable elevation of temperature. The patients often complain of pains radiating from the back into the limbs. Tetanic spasms, varying in intensity and situation, appear under the form of pleurosthotonos, opisthotonos, tetanic contractions of the limbs, and convulsions. Trismus, and at a more advanced stage, paralyses, may appear, but the latter do not last long. There is general hyperæsthesia, which renders all movements and contact insupportable. This symptom may be present in such severity that the patient, although partially stupid, is thrown into convulsions by mere contact with the skin. If improvement takes place, the skin, which was hitherto cold, becomes slowly warmer and perhaps even hot. The face, which was pale, becomes flushed, and the eyes regain their brilliancy. The temperature varies from 39°–41° C., and when it passes beyond 42° C., a fatal termination usually results. The pulse, at this stage, is generally small; the urine often contains albumen, is poor in chlorides and rich in urates; the alvine dejections cease for a long time. Polyuria and melliturıa (observed by Ziemssen, Mannkopff, and Hasse) are exceptional.

This period does not last more than 12–24 hours, but, in rare cases, it may continue three days. It is followed by the *stage of depression*, during which the patient remains in dorsal decubitus, sometimes with tremor of the limbs. The pulse is slower, the face pale, the pupils sometimes dilated, sometimes contracted. In addition to the persistent stiffness of the neck, an eruption often appears in this stage either like rubeola, scarlatina, or herpes, and extends from the eyes and ears downwards to the chin and neck. Extravasations and petechial spots rarely occur (they are met with, on autopsy, in the serous membranes). In fatal cases, the coma rapidly deepens and becomes complicated with paralytic symptoms, ptosis, strabismus, paresis of the limbs. The skin is cold and covered with a profuse sweat, and the pulse and respiration become irregular and intermittent. According to Hirsch, the skin at times is cyanosed as in the asphyctic stage of cholera. Death sometimes occurs at the beginning of the second stage (fulminant meningitis), at other times, it occurs at the close of the second or during the course of the third stage.

When the patient has passed through the stage of depression, the disease terminates in convalescence (which is usually prolonged). The most frequent termination is in recovery, which may occur in the first stage (abortive form) or after all the stages have terminated. The type of the affection, which we have depicted, is subject to very many pathological variations, depending upon the rapidity with which the exudation forms, upon its abundance and position, and, finally, upon the more or less prompt appearance of the poisonous action of the pus. The average duration is two or three weeks. The mortality varies in different epidemics from 30 to 80 per cent.

As secondary affections, we may mention intestinal catarrh, pleurisy,

pneumonia, bronchitis, pericarditis, parotitis, and suppurative irido-choroiditis, with detachment of the retina. Malarial and typhoid fevers, sometimes, also, rubeola, scarlatina, and (in two cases of Botkin) recurrent fever occur as complications.

Diagnosis and Prognosis.

The diagnosis is easy when the disease is epidemic, but sporadic cases are often confounded with other miasmatic or contagious diseases presenting analogous symptoms. The opinion of Boudin that the epidemic variety alone exists, is positively contradicted by Hasse and other observers.

Pernicious intermittent fever with its fulminant manifestations, cases of typhoid with pains in the neck, scarlatina beginning with violent cerebral symptoms, the cerebral affections which, in children, are often complicated by hyperæsthesia and pain in the neck, and finally, acute spinal meningitis, have many symptoms in common with the disease under consideration and may lead to error in diagnosis.

We may consider as characteristic signs of cerebro-spinal meningitis, the sudden invasion, fever, headache, vomiting, disturbances of sensation, constipation, small pulse, tetanic contractions affecting chiefly the muscles of the neck, and sometimes occurring in the thorax and abdomen until complete opisthotonos is produced. But, in many cases, we can arrive at a diagnosis only after prolonged observation and by way of exclusion, though usually the diagnosis may be made after the lapse of several days. Hirsch has emphasized the fact that we often have to deal apparently with the first symptoms of true epidemic meningitis, but, with appropriate treatment, recovery takes place after the return of warmth and profuse perspiration.

The *prognosis* is not unfavorable in a large number of cases. Buhl believes that when it terminates in recovery, the exudation was merely of a sero-fibrinous nature. The rapid formation and abundance of the exudation give rise to the symptoms of central irritation, followed by great depression and profound coma with small pulse, which constitute the alarming symptoms. But the danger may disappear if this condition has not lasted a long time. We may hope for recovery when the coolness of the skin gives place to warmth, when the face resumes its natural color, the eyes recover their brilliancy, the pulse becomes stronger and the tetanic spasms disappear. On the other hand, the appearance of petechiæ and extensive extravasations, drowsiness and deep coma, paralyses of the cranial nerves already mentioned, elevation of the temperature to 42° C., profuse sweats, with lividity of the skin and intermitting pulse and respiration, are the terminal symptoms indicative of approaching death.

Treatment.

Therapeutic measures must be employed for two purposes, first: to sustain the nervous system, diminish the violence of the fever and quiet the pain; and, secondly, to counteract the first symptoms of depression by the use of stimulants.

The *repeated bleedings* practised by French physicians have an injurious effect and should be proscribed, nor do the moderate bleedings, re-

cently recommended by Hanuschke and Rémy, give satisfactory results. *Local abstraction of blood* by the application of leeches behind the ears and of wet cups to the neck or vertebral column, are more useful and fulfil the indications better. They diminish rachialgia, but exert no influence upon the final termination in the severe forms of the disease.

Cold applications are especially recommended by the majority of observers. Cold compresses to the head diminish the cephalalgia and are demanded by patients who have not lost consciousness. Ziemssen has obtained more or less favorable effects by cold applications to the neck, and by pouring ether or chloroform, drop by drop, upon the occiput (production of cold by evaporation).

Cold applications to the spine, according to Wunderlich, are more disagreeable to the patients than useful. In certain more recent epidemics, methodical hydro-therapeutics has achieved good results. Ziemssen, Hirsch, Griesinger and Mannkopff, have obtained benefit from the use of quinine, both at the onset of the disease as well as in the more advanced stages with phenomena of intermittence. During convalescence, it may be employed as a tonic combined with small doses of iron.

Calomel is given in doses of 0.15 to 0.20 every two or three hours (Frentzel, Niemeyer, Dotzauer, etc.), either alone or with mercurial frictions, or with other purgatives. But the majority of authors have recognized the fact that recovery does not coincide with the appearance of the dejections produced by the calomel, and this circumstance does not speak in favor of the remedy which, in any event, must be employed with caution. Iodide of potassium has been employed by Wunderlich, Rollet, Pfeiffer, Rummel, etc., to cause absorption of the inflammatory products. It is to be preferred, at all events, to the local application of tincture of iodine to the vertebral column (Rémy).

The question as to the value of the iodide of potassium must, however, be decided by new and more careful observations.

Narcotics possess at least the advantage of procuring temporary sleep for the patient. When obstinate constipation exists, opium is advantageously replaced by its derivatives. Extract of cannabis indica (0.10–0.20) is considered by Mannkopff, Rummel, Hirsch, etc., as a useful sedative and palliative against the violent nocturnal delirium, the jactitation, opisthotonos and insomnia. Subcutaneous injections of morphine (0.12–0.15 at night for several days in succession) are of advantage in combating the insomnia, headache, rachialgia, and convulsions. Great caution must be employed in the hypodermic administration of atropine, as well as in the use of belladonna and of chloroform, which Wunderlich recommends in inhalations.

The digitalis recommended by Rummel, the oxide of zinc by Kirchof and Heuschkel, and the bromide of potassium vaunted by Prussian physicians, may be useful in combating certain symptoms, but they do not constitute an efficient medication, especially in severe cases. When symptoms of depression are manifested, recourse must be had without delay to excitants and cold affusions (repeated even several times a day).

II.—DISEASES OF THE CEREBRAL PARENCHYMA.

CHAPTER II.

CEREBRAL HYPERÆMIA.

At the close of the last and beginning of this century, Monroe, Kellie, and Abercrombie maintained that the adult skull contained an unvarying quantity of blood. This statement was seriously attacked by Burrows, Donders, and Berlin. The latter, after trephining the skull and removing the dura mater, saw the pia mater (through a watch-glass) assume a vivid red color, when respiration was arrested by closing the nose and mouth of the animal (Nederl. Lanzet, March and April, 1850).

The more recent experimental investigations have demonstrated the variability of the cerebral circulation. The retina forms, in some sort, a window, opening into the brain, and enables us to observe the cerebral circulation during life, and to estimate the quantity of blood in the cerebral and meningeal vessels, from the condition of the retinal capillaries. Certain nervines, such as belladonna and ergotine (according to Nicool, Mossop, etc.), produce, in man, pallor of the fundus of the eye from irritation of the vascular nervous centre, while alcohol, on the other hand, produces hyperæmia of the retina from paralysis of this centre. Quinine induces anæmia (or hyperæmia, if it causes nausea and gastric irritation). In many localized affections, the initial lesions of the optic nerve and the periodical amblyopia are due to cerebral congestion.

The variations in the quantity of blood in the brain coincide, according to the recent researches of Mendel (Virch. Arch., 50 Bd., 1 H.,) with local modifications of temperature. Chloroform and morphine lower the temperature of the cranial contents more rapidly than that of the rectum (cerebral anæmia from the action of the vaso-motor nerves, probably with simultaneous retardation of the nutritive changes). Alcohol, by a contrary effect upon the centres, raises the cerebral temperature above that of the rectum. Casper has observed marked congestion of the brain and menin ges in acute alcoholism. The meningeal thickenings in chronic alcoholism are due to the oft-repeated congestions.

The influences which affect the mass of the blood in the cranial cavity also exert considerable effect upon the condition of the lymphatic circulation. According to Ludwig (Lehrb. der Physiologie, II.), if cerebral congestion is produced artificially, in an animal, by section of the cervical sympathetic, the rapidity of the flow of lymph in the lymphatic trunks of the neck is increased. Gæthgens (Ueber Circulation in der Schædelhœhle, Diss. Inaug., Dorpat, 1872) injected defibrinated blood, under a strong pressure, into the carotid of the horse, collected the lymph and measured it. It became evident that a considerable increase of pres

sure in the brain promptly forced out the lymph contained in the cranial cavity.

Golgi demonstrated anatomically (Riv. Clin., IX., 1870) that the lymphatic vessels are compressed and the current of lymph accelerated in cerebral hyperæmia. The afflux of blood to the brain and the consequent dilatation of the vessels are compensated by narrowing of the perivascular sheath and outflow of the lymph. This is possible (as Golgi has shown by injection of Prussian blue), on account of the connection between the perivascular spaces and the lymphatics of the pia mater, and the communications of the latter with the subarachnoid spaces.

Pathological Anatomy.

Hyperæmia of the brain and meninges, especially of the pia mater, presents great variations of intensity. In *general acute hyperæmia*, which ordinarily follows mechanical disorders of the circulation, there is considerable vascular congestion in the superficial layers of the skull (scalp, diploë). When the dura mater has been detached, it is found to be of a bluish tint. The vessels and dilated sinuses contain partly fluid blood, which flows drop by drop from the incision, and soft clots in part, especially in the posterior regions. The vessels of the pia mater are equally gorged with blood, especially over the most compressed cerebral convolutions. The plexuses are also distended with blood. The brain is enlarged and its surface often flattened, and the medullary substance, on cut section, is found dotted with hæmorrhagic points, and may even, in intense hyperæmia of the new-born, present a reddish or deep red tint. The gray matter is of a darker color from the filling of the vessels, and small hæmorrhages may be present in it.

Partial congestions are rarer and more difficult to recognize on autopsy. They are found under the form of isolated and persistent spots. Thus, the cortical substance is often hyperæmic while the medullary tissue is normal or even anæmic. In localized disorders of the cerebral circulation, the congestion is confined to certain portions of the brain. Circumscribed congestions and capillary hæmorrhages in certain central ganglia may be found in apoplexies which are attended with incomplete loss of motion and sensation.

In general, the brain contains more blood in young people and infants than at a more advanced age. The posterior portions of the brain contain more blood than the anterior parts, on account of the position of the cadaver. The cerebellar meninges are more vascular than the cerebral; the pons varolii, corpus callosum, and fornix usually contain but little, and the cortical substance is better supplied than the medullary tissue.

Chronic hyperæmia is often due to frequent and prolonged congestion of the brain and meninges. It is recognized by opacity and thickening of the meninges, and by the dilatation of the capillaries and small vessels, which, according to Ecker's measurements, are sometimes more than double their normal diameter. As Schroeder Van der Kolk has shown, chronic hyperæmia of the cortical substance is very frequent in insanity, in which it assumes a brownish and pigmented appearance. According to recent researches, nuclear proliferations in the walls of the vessels and in the fibrillary plexus of the cortical substance, and pathological changes in the ganglion cells, or in the cortical connective tissue, occur in psychical disorders of a rapid course.

Etiology.

Among the *hyperæmias peculiar to the cranial cavity* must be mentioned the conditions of congestion in chronic drunkards and opium eaters, and those which are due to paralysis of the vaso-motor centres from violent emotions and intellectual efforts. Extreme heat and cold, and insolation act upon the brain in the same manner. The tendency to cerebral congestion (often with vertigo), which sometimes accompanies lesions of the digestive organs, is also explicable by a vaso-motor irritation and increase of intra-cerebral pressure. This view is confirmed by the recent experiments of S. Mayer and Pribram (Sitz. der Wien. Acad., 1872), in which electrical or mechanical irritation of the walls of the stomach produced a reflex increase of the vascular pressure and considerable diminution in the frequency of the pulse.

The causes of cerebral hyperæmia are more frequently found outside of the cranial cavity, and it is chiefly due to lesions of the circulatory and respiratory organs. The most active of these causes are organic diseases of the heart (valvular lesions) and disorders of the pulmonary circulation (emphysema, chronic bronchitis, cirrhosis of the lung) which produce stasis in the right auricle and superior vena cava, and consequently obstruct the return of blood from the brain. The afflux of blood to the brain may also be increased by marked cardiac hypertrophy. Increase of pressure in the vascular system is always accompanied, according to Jolly's experiments, by increase of the intra-cerebral pressure. This also occurs from stases of the inferior vena cava, in affections of the liver or other abdominal viscera, in dilatation of the hemorrhoidal veins, and in cessation of the menses. Obstacles to the return flow of blood also produce passive hyperæmia of the brain, as in compression of the jugular veins (tumors, enlarged glands, strangulation). The cerebral pressure increases when pressure is exercised simultaneously upon the carotids and jugulars.

Changes in the arteries also furnish frequent causes of cerebral hyperæmia. When the afferent vessels are atheromatous (as in old age), or when their walls present, as in certain general diseases, an unusual fragility or slight fatty degeneration (the hypoplasia described by Virchow in chlorosis), the pressure in the aortic system will be increased when the amount of blood increases, and will be followed by augmentation of the cerebral pressure from want of resistance in the vessels. We may then find a peculiar irritability of the nervous centres which causes them to react upon the slightest stimulant or upon very slight increase of vascular pressure. The laity, and even physicians, mistake this tendency to congestion, which exists even in feeble individuals, for true plethora.

Symptoms.

As a general rule, cerebral hyperæmia begins with symptoms of excitement, sometimes followed by depression. To the first category belong the headache with sensations of constriction, heat or heaviness, vivid redness of the face and conjunctiva (with rise of temperature in the external auditory canal), throbbing of the carotids, increased energy of cardiac action, fulness and force of the pulse. To these symptoms may be added, flashes of light, tinnitus aurium, vertigo, and uncertain gait.

In more severe cases, nausea, vomiting, and hyperæsthesia of sight and

hearing are present, with confusion of ideas, or even hallucinations, excitement, vertigo, and depression. The patient demands quietude, but does not sleep readily, and his slumber is disturbed and broken. After awaking, the symptoms have disappeared for the most part, but sometimes several relapses may occur.

The headache is due to afflux of blood to certain parts of the brain and to the pressure here exerted upon the dura mater and pia mater, which are endowed with a lively sensibility. Vertigo is a frequent symptom, both in cerebral hyperæmia and anæmia. Hyperæmia causes the vertigo found in a large number of cerebral affections, in the disturbances following alcoholic excesses or mental labors, in cardiac diseases, in abdominal congestions, in occupations in which the head must be kept low, etc. Loss of the feeling of equilibrium, which appears to have its origin in the uninterrupted centripetal reaction of sensorial impressions and of the muscular sense, then occurs.

Among the symptoms of chronic cerebral congestion is sometimes observed a peculiar dread in certain places (agoraphobia). I have seen two cases following mental overwork. This condition, described by Griesinger, Westphal, etc., under the name of "platzangst" (fear of places), seizes the patient while traversing streets or open places, narrow lanes, or even in the midst of a crowd. Tremors and flashes of heat are superadded to the feeling of dread. This pathological condition may exist in various morbid conditions of the brain (anæmia and hyperæmia) and cord, and also in hysteria. According to Cordes, it may occur in prolonged gastric disturbances, and may then be provoked by external influences.

In chronic forms of cerebral hyperæmia, the increase and persistence of the pressure give rise to secondary symptoms of depression. Melancholia, anxiety, weak memory, deficient judgment and will-force, and sometimes delirium and insomnia have been observed. The sensibility of the limbs is dulled, and one of them may be slightly paretic. The movements of the tongue are embarrassed, and speech is confused. The condition of depression which follows repeated irritations, or the afflux of blood to the head (coup de sang of the French) with temporary dimness of vision, borders very closely on apoplexy and is ordinarily due to serous transudations or to capillary hæmorrhages.

According to the experiments of Kussmaul and Tenner, the re-establishment of the current of blood after compression of the arteries in the neck, never gives rise to convulsions, despite the intense cerebral hyperæmia which occurs. But Landois's investigations have shown that venous congestion of the brain (and spinal cord) may produce epileptiform convulsions.

Frequent and prolonged congestion in infants may cause, according to Rokitansky, hypertrophy of the brain or chronic hydrocephalus. From the concurrent investigations of Herrich and Popp, it appears that simple cerebral hyperæmia is not, as has been hitherto believed, a cause of sudden death; pulmonary congestion usually coexists; perhaps, also, the death results from sudden paralysis of the vaso-motor centres or of the origin of the pneumogastric nerves. We frequently find, in adults, opacity and thickening of the meninges with vascular dilatation as a consequence of prolonged hyperæmia.

Diagnosis and Prognosis.

A positive diagnosis should not be made until the source of the afflux of blood to the brain has been discovered. We must, above all, ascertain

whether the congestion be not due to disease of the circulatory or respiratory apparatus, and we should carefully examine the heart, lungs, vessels, and abdominal viscera, especially the digestive organs. We must also take into consideration the habits of life, and temperament, and determine whether the hyperæmia does not proceed from an hereditary abnormal excitability of the nervous centres, or whether chlorosis, anæmia, or hysteria do not play a part in the want of resistance in the walls of the vessels. Cerebral hyperæmia forms part of the obscure, initial symptoms of tumors, encephalitis, tubercular meningitis, and other intercranial affections, in which the nature of the disease is only revealed by its further progress or by recourse to the ophthalmoscope. Cerebral congestion occurs as a complication of hysteria, spinal irritation, many convulsive affections of central origin, and exhaustion from seminal losses or onanism, etc.

The onset of acute cerebral hyperæmia is usually marked by the symptoms of irritation already mentioned, viz.: violent headache, vertigo, redness and heat of the face, palpitation of the heart, throbbing of the carotids and radial arteries, hallucinations, and mental excitement. Convulsions and paralyses are not symptoms of cerebral congestion, but belong to more serious disorders. The depression symptoms of motion and sensation, produced by cerebral hyperæmia, usually disappear unless the congestion is the precursor of apoplexy.

The *prognosis* depends upon the nature and duration of the case.

The congestions due to paralysis of the vaso-motor centres, to alteration in the quality of the blood, to abnormal cerebral excitability, or to diseases of the digestive canal, will recover. But on the other hand, the hyperæmias which are produced by organic lesions of the vascular system or lungs, and those accompanying localized lesions or profound alterations in the central nervous system, are beyond the resources of medical art.

Arterial congestions usually run a more rapid course and have a more favorable termination than venous stases. Hyperæmia is more serious in children than in adults and, among the latter, stout, plethoric subjects are more threatened than tall, lean individuals. The violence of the congestion possesses less prognostic importance than the appearance of symptoms of depression.

Treatment.

If the onset is sudden, we must act with great rapidity, but when safety is again restored, we should endeavor to ascertain and combat the cause of the congestion.

In active hyperæmia of intra-cerebral origin, we should employ, in slight cases, ablutions with cool water (used morning and evening), wet compresses to the calves and enemata of cold water (in constipation add sulphate of soda or magnesia, or castor-oil). In severe cases we must advise complete quiet and isolation; apply ice bags to the vertex or occiput, and leeches to the mastoid processes. At a later period, we would recommend a purgative mineral water, the prolonged use of milk or skimmed milk, and cold baths.

In passive hyperæmia, we should avoid all constriction from the clothes, and produce intestinal derivation (enemata of vinegar or salines). Then induce energetic revulsion to the skin, moderate the activity of the heart by digitalis, combined with a little quinine or nitrate of potash, and diminish the pulmonary secretions by the use of ipecac, lobelia, etc. If

hæmorrhoids have ceased to bleed, apply leeches to the anus, and when the menses have suddenly stopped, they should be restored by the application of the electric brush to the thighs, and by douches to the loins and perineum. In portal congestion, advantage is derived from the use of alkaline and saline mineral waters (first allowing the carbonic acid to escape) from a diet of milk, skimmed milk or grapes, and from cool sitz-baths and abdominal douches.

In hyperæmia due to exaggerated excitability of the vaso-motor centres, and to changes in the blood, we must rely upon a sojourn among the mountains, and upon the use of weak, ferruginous waters. The methodical employment of hydropathic measures is especially serviceable against increased reflex excitability

But we must refrain, in very impressionable patients, from the employment of exciting agents (cold baths, douches, etc.), and must rest content with slightly cooled sitz-baths, and gentle affusions to the head and neck. Hydropathic treatment is not indicated in congestion due to organic diseases of the heart, advanced atheroma of the arteries, and in severe passive hyperæmia.

The slight antiphlogistic medication above referred to, will suffice when the irritative phenomena are moderate. The appearance of symptoms of depression or of stasis may demand copious venesection. This should only be performed if the patient is robust, the heart's action energetic, and the pulse full and hard. In old people with poor nutrition, and when the action of the heart is feeble, and the pulse soft and irregular, venesection is contra-indicated, and we must rely upon derivatives and revulsives.

In insolation, good results have been seen from the use of cold affusions, preceded by local abstraction of blood. In chronic cerebral congestions, with tendency to relapse, the therapeutic indications are furnished by the primary disease. In incurable organic affections, sedatives should be employed. In chronic alcoholism and in the hyperæmias caused by moral causes, mental fatigue, and disturbances of digestion, the hygienic conditions and manner of life must be regulated. We should advise rest, a trip to the country, the cautious employment of hydropathic measures, and sea-baths. Insomnia is very frequent, and does not always yield to narcotics. When uncomplicated by any organic disease, I have derived great benefit from the use of half-baths in the evening (lowering the temperature from 20° to 18° or 16° C.) with affusions to the back and head, and followed by a light supper. The feelings of terror which occur, sometimes disappear after the employment of hydrotherapeutic measures, as is proven by the recent observations of Cordes.

CHAPTER III.

CEREBRAL APOPLEXY.

Pathological Anatomy and Physiology.

CAPILLARY hæmorrhages appear as small points, nearly as large as a millet seed, and situated more or less closely to one another. They are usually found in the gray substance of the convolutions or central portions. The surrounding parts are softened, reddish, tinged with blood, the nerve fibres are separated, and the capillaries are softened, friable and infiltrated with granulo-fatty matter. Virchow has shown that capillary hæmorrhages cause either hæmorrhagic infarctions in consequence of capillary embolism, or the commencement of red softening or finally capillary dilatations. According to Rokitansky, capillary hæmorrhages may be situated so closely, as to constitute a large foyer.

Hæmorrhagic foci of this nature, or large extravasations due to rupture of the arteries, form rounded or elongated cavities in the direction of the fibres. Their form and dimensions present great variations, since the lesion may be limited to a part of the brain, or occupy an entire hemisphere, may penetrate into the ventricle or break through the cortical substance and pia mater into the subarachnoid space. Large hæmorrhages may pass from the lateral into the third ventricle through the foramen of Monro, or by destruction of the intermediate portions, or may pass into the fourth ventricle through the canal of Sylvius.

The hæmorrhagic foyer rarely contains less than 3–4 grms. of blood, and more often the quantity varies from 20–60 grms. (In large hæmorrhages, it may amount to from 200–350 grms.) Recent hæmorrhages contain the remains of small vessels which present considerable changes in their walls, in addition to blood and débris of cerebral substance.

Position of the extravasation. The apoplexy is situated superficially when due to lesions in the convolutions or adjacent structures, as in thrombosis of the sinuses and meningeal veins, or when it has been caused by proximity to localized processes. It is situated deeply and centrally when it attacks the chief centres of the cerebral functions.

Recent histological and experimental investigations throw more and more doubt upon the motor properties of the optic thalamus, while the influence of the lenticular nucleus upon voluntary motion is demonstrated by a large number of autopsies in cases of apoplexy.

In 103 autopsies upon cases of apoplexy, made at the Vienna General Hospital, the hæmorrhage or apoplectic cyst was situated

In the corpus striatum alone	32	times
" " nucleus lentiformis alone	20	"
" both these ganglia combined	8	"
" " corp. striat. and optic thalamus	7	"

In the lenticular nucleus and other parts (centrum semiovale, occipital lobe, island of Reil, pons and cerebellum)	6	times
" " optic thalamus alone	20	"
" " optic thalam. and corp. striat. of both sides (recent hæmorrhages and old cicatrices)	2	"
" " optic thalam. and lent. nucleus of both sides	3	"
" " centrum semiovale	3	"
" " parietal lobe	2	"
Total	103	"

It is evident from this table that the corpus striatum and lenticular nucleus were the seat of the hæmorrhage in more than two-thirds of the cases. It is often localized in certain portions of the corpus striatum or lenticular nucleus, which are nourished by small vessels passing off from several arteries at different heights (arteries of the corpus callosum, of the Sylvian fissure, or branches of the posterior communicating artery). (Heubner and Duret.)

According to microscopical researches, the corpus striatum and lenticular nucleus serve as the central origin for the fibres which pass through the foot of the cerebral peduncle and are, consequently, the prolongation of the pyramids. Since, on the other hand, the two cerebral ganglia contain the peripheral portion of the corona radiata, which has its origin in the cortical substance of the brain, they constitute also the paths for the transmission of motor stimuli from the cerebral cortex to the anterior roots. According to Huschke's measurements, the lenticular nucleus, in adults, is the largest and most important of the central ganglia. According to Meynert, the cerebral lobes and the foot of the cerebral peduncle, with its ganglia, are the most developed organs in the human brain, and the lenticular nucleus is so much larger, that the corpus striatum appears to be dependent upon a part of the brain which has been arrested in its development, viz.: the olfactory lobe. On the other hand, the ganglia of the tegmentum of the cerebral peduncle (the optic thalamus and corpora quadrigemina, which serve as the origin of the posterior spinal roots), are much less developed in the human brain.

These anatomical facts agree with the more recent researches upon the functions of the different parts of the brain. According to the investigations of Nothnagel (Virch. Arch., LVII. Bd., 2 H., 1873), who injected small quantities of chromic acid into different parts of the cortex, the lenticular nucleus in rabbits is chiefly an organ for the transmission of motor impulses. When the injection is made into the anterior or middle portions, deflection of the limbs and vertebral column is produced. Lesions of certain portions of the corpus striatum produced analogous motor disturbances. Ferrier's experiments (Lon. Med. Record, 18, 1873) upon the monkey and other animals, demonstrated that the corpus striatum acts upon the muscles of the opposite side of the body. When the irritation was great, the action of the flexors exceeded that of the extensors. The optic thalamus, according to Ferrier, plays no part in the production of movements.

We will now investigate the course and sequences of a cerebral hæmorrhage. Recovery usually takes place by the formation of false membranes and by the secretion of a serous fluid which dissolves the blood clots and the débris of the cerebral tissue. This assumes a yellowish or

brownish color from admixture with derivatives of hematine. The cavities thus formed are traversed by pigmented vascular bands of connective tissue and contain a yellowish fluid. These cavities are formed within 2–3 months after the occurrence of the hemorrhage, and are known as apoplectic cysts. In favorable cases, the connective tissue retracts, the walls approach one another and coalesce, the capillaries become obliterated and a stellated apoplectic cicatrix results.

Great importance must be attached to the secondary lesions of nutrition which are produced as a consequence of hæmorrhage in the paths of transmission of motor and sensory impulses.

With regard to the motor tracts, Tuerck first demonstrated (Sitz. der Wiener Acad. d. Wiss., VI. Bd., 1851) that a foyer of hæmorrhage or encephalitis situated in the optic thalamus, corpus striatum and lenticular nucleus, will produce an atrophic degeneration in the lateral columns of the pyramid, along the longitudinal fibres of the cerebral peduncle to the corresponding part of the pons, and thus to the pyramid and to the posterior half of the opposite lateral column. It also partially affects the anterior column of the same side, but only in the internal portion, bordering on the anterior sulcus. According to Bouchard (Archiv. gén. de méd., 1866) these secondary degenerations do not follow lesions of the corpus striatum, the optic thalamus, and third segment of the lenticular nucleus, or superficial lesions of the cortical substance. On the other hand, they always exist in lesions "en foyer" of the anterior two-thirds of the internal capsule, of the first and second segments of the lenticular nucleus, and in profound lesions of the cortical substance corresponding to the psycho-motor centres of the brain (Charcot).

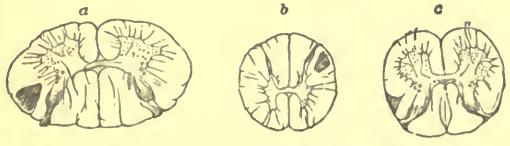

FIG. 1.—Secondary degeneration of the cord, following hæmorrhage into the corpus striatum and internal capsule of the right hemisphere. *a*, cervical region; *b*, dorsal region; *c*, lumbar region. The descending sclerosis (shaded dark) occupies, in the cervical enlargement, the deep portion of the lateral column and becomes superficial in the lumbar region.

To Tuerck, also, we owe our first conceptions of the anatomical changes in the sensory tracts (Sitzber. d. Kais. Acad. d. Wiss. XXXVI. Bd., 1859). In four cases of persistent anæsthesia in the paralyzed limbs of hemiplegic patients, the autopsy and microscopical examination proved the existence of foyers of hæmorrhage and softening (cellular infiltration), at the external periphery of the optic thalami, and measuring from eight lines to one inch, antero-posteriorly, and two inches in the medullary substance. The portions affected were the superior and external parts of the optic thalamus, the third segment of the lenticular nucleus, the posterior portion of the internal capsule, the portion of the corona radiata which passes this point, and the adjacent part of the medullary substance of the superior lobe. The secondary development of granular corpuscles, in some parts of the columns of the cord, had no influence upon the production of anæsthesia. According to the researches of Meissner upon the nerve fibres of the tactile corpuscles in central anæsthesia, the atrophy may also affect the peripheral nervous system.

As complementary to the facts discovered by Tuerck regarding lesions of the sensory tracts, I have set forth (Wochenbl. ges d. Aerzte, n. 15, 1870) the results of seven autopsies, to which I must now add an eighth case since observed, in which left hemiplegia and hemianæsthesia were

caused by two apoplectic cysts in the right lenticular nucleus with a spot of softening as large as an almond in the external part of the optic thalamus (masses of granular corpuscles extending to the posterior lobes).

In the cases in question, the persistent apoplectic anæsthesias were produced by irreparable lesions of the paths of transmission. Spots of softening, of the size of a bean or almond, formed by the débris of nerve fibres and accumulations of nuclei, existed in the white substance between the cerebral ganglia, especially between the optic thalamus and lenticular nucleus, and in the tissue which unites these to the cerebral lobes. The anatomical lesions, in the slight and incomplete anæsthesias, were œdema of the same portions (especially the posterior part of the lenticular nucleus) combined with intense hyperæmia or capillary hæmorrhages. Microscopical examinations of sections of the brain prove that the parts mentioned form the expansion of the posterior columns which, according to Luys and Meynert, decussate in the medulla oblongata, and pass along the pons varolii and external part of the foot of the cerebral peduncle towards the white substance, between the optic thalamus and lenticular nucleus (internal capsule).

The sensory rôle of these nerve-fibres is still further confirmed by the disturbances of sensation observed from lesions in the intermediate portions of their course. Andral and Friedreich have seen sensory disturbances on the side opposite to the lesion in tumors adjacent to the optic thalamus and, more recently, Charcot has made similar observations in hæmorrhage or softening in the posterior portion of the optic thalamus and neighboring parts of the hemisphere (with hemiplegic tremor). Sensation is affected on the opposite side of the body when new growths develop in the peduncles. A diminution of sensation often occurs on the opposite side in tumors of the pons varolii and medulla oblongata.

From the evidence adduced, it appears that the sensory fibres decussate in the medulla oblongata, passing through its posterior part, and through the external fasciculus of the foot of the cerebral peduncle, and terminate in the tissues situated between the cerebral ganglia, in the postero-external portion of the optic thalamus, and in its connections with the occipital, and perhaps with the temporal lobe. Pathological facts serve to show the central seat of sensibility in the brain better than physiological experiments. Thus, the production of hemianæsthesia observed by Veyssière (Arch. de Physiol., 1874, p. 288) in dogs, after lesions of the internal capsule, is less satisfactory than the results obtained in human pathology. According to the latest researches of Carville and Duret, lesions of the anterior part of the internal capsule (beneath the ventricular surface of the lenticular nucleus) produce complete hemiplegia. Lesions of the posterior portion of the internal capsule (between the optic thalamus and lenticular nucleus) produce hemianæsthesia on the opposite side.

Etiology.

Lesions of the vascular system play the most important part in the pathogeny of cerebral hæmorrhage, since the extravasation is caused by rupture of cerebral arterioles.

This rupture arises from degeneration and weakness of the vascular walls, or from an increase of arterial pressure, or from both combined; or the rupture may be due to the debilitating effect of certain diseases upon the vascular tissue, or, finally, to degeneration of the cerebral tissue which has, at a later period, involved the blood-vessels.

The most frequent cause, however, is the degeneration of the vascular walls. Until recently, the disposition to cerebral hemorrhage was attributed to atheroma of the large cerebral arteries and to the friability of the vascular walls accompanying old age, and which is explained by fatty or calcareous degeneration, especially of the internal coat.

More than twenty years ago, Paget (Lond. Med. Gaz., Feb., 1850) called attention to the fatty degeneration of the small cerebral arteries in cases of apoplexy. Almost at the same time, Koelliker (Zeits. f. wiss. Zool., t. I.) and Pestalozzi (Ueber aneurysmata spuria d. Kleinen Hirnart. bei Apoplexie, 1849) found, upon arteries whose diameter varied from 0·05 to 0·009, hæmorrhagic extravasations which, after rupture of the internal and middle coats, had become lodged between these and the tunica adventitia, and had distended the latter into a flask-shape. Shortly after, Wedl obtained analogous results and also observed obstruction of the vessels from cellular proliferation (Grundzüge d. path. Hist. Vienna, 1852). Brummerstaedt and Moosherr (Ueber d. path. Verhalten d. klein. Hirngefässe, Wurzburg, 1854) described in detail the fatty degeneration observed in twenty-one cases, in the small cerebral arteries, and which usually affected the middle coat, more rarely the internal tunic.

The arterial dilatations have more recently received their true interpretation in the pathogeny of cerebral hæmorrhages, at the hands of Charcot and Bouchard (Arch. de Physiol., 1868, p. 110). The miliary aneurisms found by these observers in sixty cases, upon the small cerebral arteries, appeared like small nodosities, of the size of a millet-seed to that of a pin's head, sometimes isolated, sometimes scattered throughout the entire brain in large numbers. They may be present without coexisting sclerosis of the arteries at the base. In miliary aneurisms, following inflammation of the lymphatic sheaths of the arterioles and atrophy of the muscular elements, the internal coats first give way and the distended tunica adventitia then gives rise to a dissecting hæmorrhage. This condition may last a long time, resulting in the formation of a small mass of pigment, or the tunica adventitia may also rupture, giving rise to a true cerebral hæmorrhage. This lesion increases in frequency after the age of fifty; it is more infrequent before this period, but exceptionally appears below the age of twenty.

The latest investigations of Zenker (45th Congress of German Naturalists and Physicians) have also demonstrated that a microscopical examination of these aneurisms and the arteries upon which they are situated, reveals the same tendency to sclerosis (thickening, sometimes fatty degeneration) in the internal coat that has been recognized for a long time in the large arteries. The miliary aneurisms are thus the results of arterio-sclerosis carried to its highest development and affecting the final ramifications of the vessels. There is, therefore, a perfect analogy between apoplexy and extra-cerebral hæmorrhages at the base, which usually result from rupture of an aneurism situated in the arteries at the base of the brain.

The vascular dilatation and atheroma attain their highest development in the corpus striatum and lenticular nucleus, then in the optic thalamus and other portions of the brain which are most frequently the seat of hæmorrhage.

The vascular lesion may exist for a long time without any evil results, but it becomes dangerous when an increase of vascular pressure is superadded to the cerebral arterio-sclerosis.

This may occur from lesions of the brain itself or from peripheral diseases.

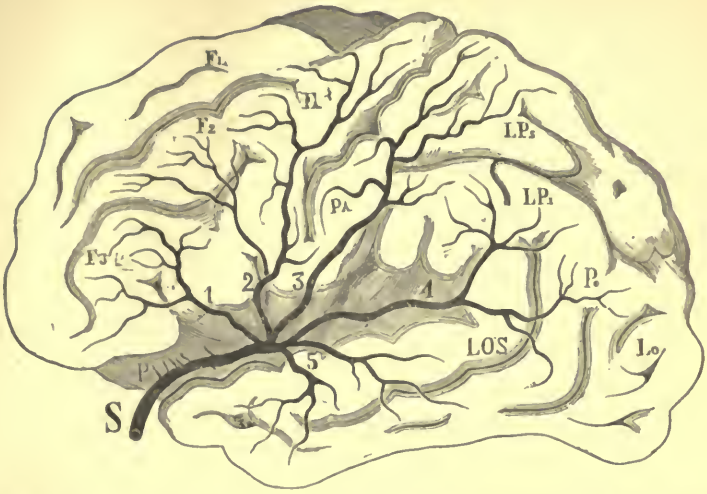

FIG. 2.—External distribution of the middle cerebral artery: S, trunk of the vessels; P, perforating branches to the central ganglia; 1, artery of Broca's convolution; 2, ascending frontal artery; 3, ascending parietal artery; 4 and 5, parieto-sphenoidal and sphenoidal arteries; F_1, F_2, F_3, 1st, 2d, and 3d frontal convolutions; Fa, ascending frontal convolution; Pa, ascending parietal convolution; LPI, inferior parietal lobule; Lo, occipital lobule; Pc, angular gyrus.

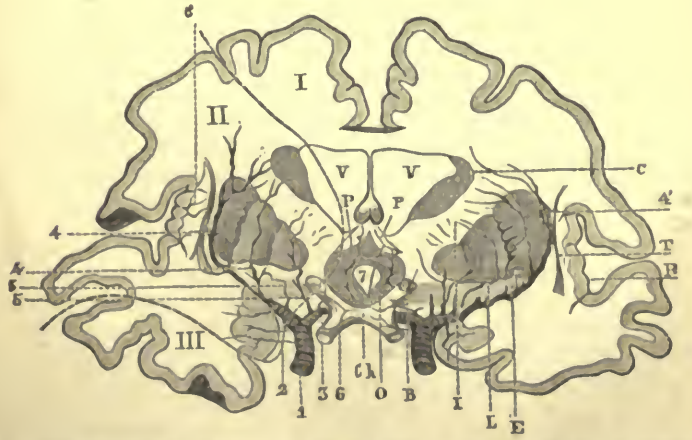

FIG. 3.—Internal distribution of the middle cerebral artery: I. vascular territory of anterior cerebral artery, II., of middle cerebral artery, III., of posterior cerebral artery; 1, internal carotid, 2, middle cerebral artery, 3, anterior cerebral artery, 4, external arteries of corp. striat., 5, internal arteries of corpus striat.; Ch, chiasm of optic nerves; B, section of optic tract; L, lenticular nucleus; I, internal capsule; C, corpus striatum; E, external capsule; R, island of Reil; V, lateral ventricles.

To the first class belong the arterial congestions due to a persistent elevation of pressure in the aortic system and to psychical or toxic irritation; the arrest of circulation due to vascular spasm in epilepsy and eclampsia; the passive congestions following thrombosis of the sinuses or of the veins of the pia mater; the dilatation and rupture of capillaries from pigment emboli (Virchow); the accumulation of white blood-globules in pyæmia (Rokitansky), and in leukæmia (Ollivier and Ranvier). In the same class we must also place the hæmorrhages which sometimes occur in the fœtus and which may have been recovered from at the time of birth (Rokitansky).

In hæmorrhages within the domain of certain cerebral arteries, we should carefully examine the condition of the nutrient vessels, according to the topographical relations indicated by Heubner (Centralb. f. med. Wiss., No. 72, 1872). This applies especially to the circle of Willis and to the large trunks of the base, which nourish the motor ganglia and corresponding part of the middle lobe. As Heubner pointed out, these vessels do not anastomose with each other, and are distributed to perfectly distinct regions (terminal arteries of Cohnheim). The vessels of the cortical substance which anastomose with each other, in the pia mater, are rarely the seat of hæmorrhages.

Among the peripheral causes of cerebral hæmorrhage, we may mention: constriction of the venous orifices, valvular lesions, hypertrophy of the left ventricle (the compensatory action of which does not continue long, and which gives rise, secondarily, according to Traube, to arterio-sclerosis), thrombosis and rupture of the cerebral arteries. Hypertrophy of the heart favors the production of cerebral hæmorrhage when the circulation is obstructed peripherally (pulmonary emphysema, atrophic kidneys, diffuse arterio-sclerosis, compression of the jugular veins or superior vena cava, etc.).

It was, for a long time, supposed that an alteration in the blood occurred in cerebral apoplexy. More modern researches place the real cause in changes of the vascular walls or cerebral substance. Thus, we often find fatty degeneration of the small cerebral vessels in cachectic infants. Chlorosis also favors the early production of apoplexy. Virchow has lately described in this disease a condition known as hypoplasia of the vascular system (Intelligenzbl. 29, 1872), the characteristics of which consist in diminution of the calibre of the arteries, attenuation of their walls, and the presence of wavy or reticulated projections from their inner coat. They show a marked tendency to fatty degeneration, not only of their inner coat, as in atheroma, but also in the external layers. Changes in the middle coat of the vessels and in the heart are more rare in chlorosis, but they occur with frequency in the puerperal condition. Meynert has seen capillary aneurisms in the pons varolii and peduncle (Wochenbl. d. Ges. d. Aerzte, July, 1864) in scorbutus. In syphilitic cerebral diseases, Virchow, Passavant, and Heubner have found exudations around the arteries at the base and in the walls of the small cerebral vessels. Chronic alcoholism is also a frequent cause of fatty degeneration of cerebral capillaries.

It is very doubtful whether *changes in the cerebral substance* alone are sufficient to produce apoplexy. The more or less circumscribed lesions of the cerebral parenchyma, with extravasation, which are observed in tumors, softening, etc., are evidently due to arrest of the collateral circulation, to secondary lesions of the afferent vessels, or to granulo-fatty infiltration of the walls of the capillaries. We have no proof that hæmorrhages occur in primary cerebral atrophy solely from "abhorrence of a vacuum,"

and from increase in the current of blood in consequence of the decrease in the cerebral mass. If we take into consideration the advanced age of these patients, it will be more rational to regard circulatory disturbances and changes in the vascular system as the causes of the hæmorrhage. *Temperament* and *constitution* have also been considered as predisposing causes of apoplexy, but careful observation disproves this opinion. Bad diet, mental and physical over-exertion, constipation, vomiting, excesses, forced expiratory movements, etc., have been regarded as accidental causes. But only a secondary importance must be attached to all these factors, and they never give rise to a cerebral hæmorrhage when the vascular apparatus of the brain is intact. In many cases, heredity is a predisposing cause of apoplexy.

Age exerts a marked influence on the occurrence of the disease in question. According to Burrows's statistics, apoplexy occurs in the proportion of 5.3 per 1,000 between the ages of 20 and 30; of 12 per 1,000 between 30–40 years; of 22.2 per 1,000 between 40–50 years; of 31.3 per 1,000 between 50–60 years; of 54 per 1,000 between 60–70 years; and of 60 per 1,000 between 70–80 years. According to Sormani's statistics of 5,078 cases (Riv. Clin., 2 series, Dec., 1872), cerebral hæmorrhage is rare between the ages of 4 and 22 years; between 22 and 50 years, the mortality increases with the age and rises still higher after the fiftieth year. The mortality from apoplexy is very large also in children, especially in infancy.

Sormani and Mandillon's statistics show that apoplexy is more frequent in cold seasons, a statement which agrees in part, but not entirely, with the observations made in Vienna. Sormani states that there are two periods of maximum occurrence in the 24 hours, the one from 3–5 o'clock in the afternoon, the other from 2–4 o'clock in the morning; this is undoubtedly due to the effect of the day's work, of the ingestion of the meals, and of the use of spirituous liquors. The male sex is more subject to apoplexy than the female, owing to the more laborious work performed by men, and to the greater frequency in them of diseases of the heart and vessels.

Symptomatology.

An attack of apoplexy is usually preceded by prodromal symptoms of cerebral irritation. These consist of headache or a feeling of constriction in the head, dizziness, flushing or pallor of the face, ringing in the ears, nausea, weakness of memory, extreme irritability, tremor or disseminated neuralgic pains, a feeling of heaviness or stiffness in the limbs, difficulty of speech, irregularity of the heart's action and retardation (in rare cases, acceleration) of the pulse.

After the premonitory stage, the apoplectic attack usually occurs with a certain suddenness, although the symptoms may also develop slowly. We may classify the forms of this disease as slight, moderate, and severe. The slight cases are those in which consciousness is retained or disturbed only for a short time, or in which slight alterations of sensibility, and muscular paresis limited to the face, tongue, or one limb ensue, but soon disappear spontaneously.

In the moderate forms, after a more or less complete loss of consciousness, hemiplegia of motion and sensation and difficulty of speech persist. Consciousness is often restored only at the end of several hours, but even during coma we may recognize the paralyzed side, despite the general

muscular resolution, by the fact that when the limb is raised, it will drop inertly; the face is also paralyzed on one side. Reflex excitability is usually abolished, respiration is very labored and irregular, the face is tumefied and cyanosed from venous congestion, the pulse is small and frequent, but deglutition is still possible. The return of reflex movements ordinarily precedes the restoration of consciousness. The mental faculties recover little by little, the patient being still stunned when he begins to look around him, memory is confused, speech difficult, the tongue, when protruded, deviates to one side, and the paralyzed limbs do not perform any active movements. At a later period the hemiplegia improves (it rarely disappears entirely), and the patient may remain in this state for several years, or may sustain other attacks which aggravate his paralysis and general condition.

In the severe forms of apoplexy (with the exception of the immediately fatal fulminant cases), the extent of the cerebral affection, the participation of the lateral ventricles, and the increase of pressure, may cause death within a few hours or days. Profound coma, slow pulse and respiration, resolution of all the limbs, relaxation of the sphincters, absence of reflex movements, and immobility of the pupils, are the grave symptoms which indicate that life is seriously endangered.

If the coma remains profound into the second half of the day following the attack, if the temperature is at first considerably lowered (to 35.5° C.,) and then rises to 42° C. (Bourneville), if all the above-mentioned symptoms of depression remain unchanged, with complete inability to swallow, we must apprehend the near approach of death. This ensues with the symptoms of paralysis of the respiratory and circulatory centres. The gradual return of the sensibility of the pupils, and of reflex contractility indicates a diminution of the intra-cerebral pressure, a fact which is still further confirmed by the restoration of consciousness.

It goes without saying that the intensity and extent of all these symptoms is the surest criterion of the gravity of the hæmorrhage. Even when the symptoms are progressing favorably, new dangers may arise from *inflammatory reaction* around the hæmorrhage. This reaction is sometimes so slight that it does not interfere appreciably with recovery. In the majority of cases, inflammatory symptoms make their appearance some days after the attack. The patient becomes anxious, is affected with pain or heaviness in the head, and delirium, and with contracture of the paralyzed limbs, affecting especially the flexors. The congestion of the face, elevation of temperature, slowing of the pulse and respirations, and the mental disturbances prove the existence of an inflammatory process in the brain. This condition disappears, in favorable cases, at the end of several days, or latest, at the end of a week. In severe cases, when the inflammation extends for some distance around the seat of the hæmorrhage, and collateral œdema of the brain tissue develops, death usually occurs rapidly in the midst of coma, with slowing of the pulse and respiration, and elevation of the temperature (41–42.8° C., according to Bourneville).

When the acute stage of cerebral hæmorrhage (comprising the attack itself and the period of inflammatory reaction) is passed, we find ourselves face to face with the chronic sequences of cerebral apoplexy. In the front rank must be placed the disorders of motion, which are at once the most troublesome and the most evident. They consist of paralyses or pareses affecting the muscles of one eye, one-half of the face and tongue, and the limbs on one side of the body.

Of the *ocular muscles*, the right internus is oftener affected than the right externus. According to Prevost, conjugate deviation of the eyes and rotation of the head to the side of the hæmorrhage or softening occur especially in sudden attacks, and are more apt to be present, the nearer the lesion is to the corpus striatum and expansions of the cerebral peduncles. The pareses of the ocular muscles usually last for a short time. In the *face*, the paralysis involves, by preference, the elevators of the wing of the nose and of the labial commissure, and sometimes the buccinator and the respiratory filaments of the facial nerve, whose centre is in the cerebral ganglia. Judging from the results of experiments on animals, irritation of the corpus striatum and optic thalamus should produce contraction of the muscles on the opposite side of the face (Eckhard, Experiment. Phys. des Nervensystems, 1867, p. 157). Lesion of the peduncle at the inferior surface of the lenticular nucleus may, according to Huguenin, produce isolated paralysis of the orbicularis palpebrarum. In the rare cases in which the paralysis involves the entire domain of the facial nerve, it may also implicate the branches which advance into the cortical layer of the anterior lobe, and whose centre has been found, according to Hitzig and Fritsch, at the boundary of the inferior and middle third of the anterior central convolution. Sometimes, also, the temporal lobe is affected (Chvostek). Duplay has observed the exceedingly rare occurrence of completely isolated paralyses of individual muscles of the face in circumscribed lesions, once in the corpus striatum, and again in the optic thalamus (Union Méd., 1857, No. 100–102).

In *paralysis of the tongue*, the organ is protruded with difficulty and deviates to the paralyzed side. This symptom, which also occurs after section of one of the hypoglossal nerves, is caused, according to Schiff, by the predominance of the sound genioglossus, the fibres of which draw the tip of the tongue to the opposite side. When the tongue is carried back, it deviates to the sound side, owing to the contraction of the styloglossus muscle.

The *paralysis of the limbs* usually affects the side of the body opposite to the seat of hæmorrhage. It is generally more marked in the upper than in the lower limb, and especially in the extensors of the fingers and hand. When the paralysis affects the extensors of the thighs, the patient cannot use the paralyzed leg in walking, except by bringing the muscles of the trunk and pelvis into play and the limb then describes slight oscillations from behind forward. The lower limb generally recovers more rapidly and to a greater extent than the upper, but the contrary is sometimes observed. When the paralysis of the extensors continues a long time and is complicated with muscular atrophy, it is followed by contracture of the flexors of the arm and fingers.

When *the hemiplegia is situated on the same side as the cerebral hæmorrhage*, we never find a lesion in the corpus striatum or lenticular nucleus of one side alone. It also exists in the corresponding cerebral lobe, and concomitant softening or œdema of the cerebral substance is equally evident in the opposite hemisphere.

The *electrical reactions* of the paralyzed muscles present no appreciable differences from those of the unaffected side. It is only in old and complete paralyses of adults that we can observe a reduction of faradic excitability in the muscles of the contractured limbs, due to atrophy and degeneration of the muscles. The galvanic excitability of the nerves is increased at the onset of the attack.

The *disturbances of sensation* form a second class of changes follow-

ing in the train of apoplexy. It was formerly held that disturbances of sensation were a relatively rare complication, but at the present time they are not regarded as exceptional occurrences. From a careful examination of about fifty cases of hemiplegia from cerebral hæmorrhage, I find that nearly all are accompanied, after a variable period, by disturbances of sensation which are manifested either by diminution or by abolition of electro-cutaneous or electro-muscular sensibility. The sensibility is usually restored at an early period, or at the close of the first week. In certain profound lesions of which we have spoken, the anæsthesia involves the skin, the muscles, and even the smaller joints. Passive movements and electrical contractions are not perceived by the patient although voluntary movements can still be performed. Sometimes the brachial plexus and certain nerve trunks also take part in the anæsthesia, which extends over the paralyzed half of the body and face as far as the median line. The parotid and masseteric regions, innervated by the third cervical nerve, and the region to which the auriculo-temporal and occipital nerves are distributed, usually escape.

In most cases the paralysis of sensation disappears more rapidly than that of motion. The return of sensibility may be complete, but sometimes the functions of the sensory nerves remain permanently changed (sensation of cloth upon the skin, or formications). There are few cases in which motion is entirely restored, sensation meanwhile remaining permanently abolished.

The recovery from profound anæsthesias of apoplectic origin follows a centrifugal course. When the anæsthesia disappears it immediately gives place to hyperæsthesia, and the latter may show itself in the superior parts while the inferior portions are still anæsthetic. The deeper layers recover their sensibility sooner than the superficial ones, and the nerves before the muscles and skin. Electrical sensibility usually returns before the sensibility to mechanical irritants and temperature, and also before reflex sensibility.

The different varieties of sensation may be more or less affected in apoplexy. Spring and Landois and Mosler have seen two cases in which analgesia and loss of sensibility to temperature existed, although tactile sensation and power of localization were normal. In some cases, insensibility to changes of temperature is alone present. These observations support the hypothesis of the existence of distinct centres for the different varieties of sensation. Reflex excitability is usually increased.

The "tendon reflex" described by Erb and Westphal (Arch. f. Psych. u. Nervenk., Bd. I.), which is especially noticeable when the ligamentum patellæ is gently struck, is easily obtained upon the paralyzed side. It is followed by rapid contraction of the triceps femoris, with extension of the previously flexed thigh. I have even seen it followed by strong concussion of the upper part of the body, although percussion of the ligamentum patellæ on the sound side merely produced feeble muscular contractions.

Sympathetic movements appear in the paralyzed limbs as a consequence of mental excitement, in coughing, yawning, sneezing, micturition, defecation, and during vigorous movements of the healthy side. This phenomenon is especially well-marked, according to Westphal, in hemiplegia with contracture dating from childhood.

The *organs of sense* are more or less affected in apoplexy, but in most cases they promptly recover their function. Persistent alterations of sight and hearing are due to increase of intracranial pressure and conse-

quent stases. Schmidt (Arch. f. Anat. u. Physiol., 1869, p. 152) has experimentally demonstrated the existence of a communication between the cavity of the arachnoid and the lamina cribrosa, and E. Weber (Monatsb. f. Ohrenheilk., 8, 1869) has discovered an analogous relation between the cavity of the arachnoid and the labyrinth of the ear. The amauroses sometimes observed before or with apoplectic paralyses, may be due to embolism of the central artery of the retina, or to hæmorrhage in the sheath of the optic nerve, in the corpora geniculata, or in the corp. quadrigemina.

Disorders of intelligence either precede or follow cerebral hæmorrhage. Experience has shown that psychical disturbances after hæmorrhage depend less upon the situation than upon the amount of the extravasation. Everything depends (according to Heubner) upon the pressure sustained by the large meshes formed in the pia mater by the cerebral arteries, and by the vessels of the cortex which pass off from the former at right angles. In slight circulatory disturbances of the cortical substance, the intelligence remains intact or is only altered to a slight extent. In large extravasations and in hæmorrhages into the cortical substance, the loss of consciousness is complete, the coma is profound and persistent, and if the attack does not prove fatal, more or less serious disorders of the psychical faculties become permanent and are manifested by feebleness of intelligence and memory, by apathy, childish ideas, and by inclination to weep, (more rarely by an irresistible inclination to laugh in the midst of serious conversation). Insanity, properly speaking, is exceptional.

In most cases we find, in the beginning, an elevation of temperature varying, according to Folet and others, from 0.3°–0.9° C., but never reaching 1° in the axilla. Charcot has found an elevation of 3°, 4°, and even 9° in the hands. At a more advanced stage of the paralysis, the temperature becomes normal, and later still, when progressive paralytic atrophy occurs, it is subnormal. In many cases, Charcot has seen the blood of the paralyzed side redder than that of the sound side.

Eulenburg (Berl. klin. W'schr., 1868) has shown, by the sphygmograph, that the arterial tension is lessened in the paralyzed limbs. The initial elevation of temperature and its subsequent fall are explained by paralysis of the vaso-motor nerves in the cerebral peduncles, by the persistent slowness of the circulation, and by the passive hyperæmia.

Certain disturbances—viz.: bed-sores and articular swellings, which, according to Charcot (Leçons sur les maladies du système nerveux, pp. 68–106) are of a trophic nature—remain to be discussed.

Acute bed-sores appear (in hæmorrhages and other cerebral affections) upon the paralyzed side from the second to the fourth day of the disease, under the form of erythematous patches (towards the centre of the sacral region). A central bulla quickly forms and opens, leaving the derma bare and strewn with deep violet specks. Excoriation rapidly occurs, and at the end of a few days, a dry, brownish crust, 6–7 centimètres in diameter, forms in the centre.

Death usually occurs before the eschar commences to detach itself at the borders. The eschar is confined to the paralyzed side, or may also extend to the sound side, but less rapidly and to a slighter extent. It is still impossible to determine whether these trophic lesions depend upon hyperæmia from paralysis or whether, according to Charcot, they are the result of the irritation of certain portions of the brain presiding over the nutrition of the parts involved.

Arthropathies in hemiplegia from cerebral hæmorrhage have been described by Scott Alison, Brown-Séquard, and especially by Charcot. They

occur most frequently in the shoulder, and more rarely in the knee, elbow, or wrist. The joint is swollen (not œdematous), hot and moist, painful during motion, and sometimes spontaneously.

Upon autopsy, we find the synovial membrane swollen, villous, and injected, proliferation of cells and connective tissue, and development of capillary vessels, and sometimes an accumulation of serum; in two cases, the median nerve was thickened and injected. Brown-Séquard and Charcot consider these hemiplegic arthropathies as neuro-paralytic in character.

Special Symptoms depending upon the Seat of the Hæmorrhage.

Hæmorrhage in the Cortical Substance.— Psychical troubles play the chief part in these hæmorrhages. Extravasations limited to the cerebral convolutions are manifested in the beginning by loss of consciousness, convulsions, muscular spasms of the face, and disturbances of the special senses. Extravasations involving the cortex of the sphenoidal and occipital lobes have no effect upon voluntary motion. The recent researches of Charcot and Pitres (Rev. Mensuelle, Jan., 1877) have demonstrated that destruction of the inferior parietal lobe, angular gyrus, island of Reil, cuneiform and quadrate convolutions, the orbital gyrus, and the anterior portion of the first, second, and third frontal convolutions, may occur from hæmorrhage or softening, without the supervention of motor disturbances. Lesions of these parts do not give rise to secondary degeneration of the spinal cord.

When an abundant extravasation occurs into the pia mater involving the anterior parts to a considerable depth, paralysis preceded by convulsions will occur. Death takes place in coma, either within twenty-four hours or at the end of several days. Respiration is more frequently slowed than the pulse.

If the patient recovers, derangement of the mental faculties may remain and finally lead to imbecility or idiocy.

Hæmorrhage into the anterior lobe. Lesions affecting the anterior lobe to a considerable depth are characterized by aphasia and hemiplegia with incomplete facial paralysis.

Heubner has shown that the middle cerebral artery, the upper part of which furnishes blood to the motor ganglia, not only supplies the island of Reil, but divides at the same time into four or five branches, the first of which nourishes the second and third frontal convolutions. From these anatomical facts, we can readily comprehend that hæmorrhages into the territory of the middle cerebral artery produce disorders of the motor centres and of the organ of language, the latter being situated in the island of Reil and the parts uniting it to the anterior lobe.

Hæmorrhage into the middle and posterior lobes.—In hæmorrhages into the middle lobe, with integrity of the ganglia, I have twice observed headache, dizziness, slight nausea and amblyopia, without any affection of motion, sensation, or of the mental faculties. In Hughlings-Jackson's analagous cases, the amaurosis was accompanied by congestion of the retinal veins and injection of the papilla, which presented white spots in its centre. Hæmorrhages into the posterior lobes are marked by a more persistent disturbance of intelligence, and by the absence of hemiplegia and of sensory disorders. If aphasia occurs in lesions of the middle and posterior lobes, the seat of language, referred to above, has become involved by some complication.

Hæmorrhage into the motor ganglia.—Hæmorrhages in this situation cause disorders of intelligence and of the special senses, of short duration; hemiplegia (more complete in lesion of the lenticular nucleus than in that of the corpus striatum), incomplete facial paralysis, temporary pareses of ocular muscles, and diminution of sensibility in the paralyzed limbs. More severe and permanent disorders of sensation, added to the other symptoms just mentioned, indicate a lesion involving the expansion of the external fibres of the foot of the cerebral peduncle at the level of the white substance situated behind the lenticular nucleus.

Hæmorrhage into the cerebral peduncle.—Intelligence and special senses are unaffected. Hemiplegia occurs with considerable diminution of cutaneous sensibility, together with alternating facial paralysis of an incomplete and temporary character, and a more or less profound paralysis of the oculo-motor nerve (ptosis, mydriasis, diverging strabismus). In a case observed by Andral (Clin. med., V., p. 326) of a hæmorrhagic cyst in the middle of the cerebral peduncle, paralysis of the oculo-motor nerve did not develop on account of the remoteness of the cyst from the origin of the third pair of nerves.

Hæmorrhage into the Pons Varolii.—When a large part of the pons is suddenly destroyed by a hæmorrhage, death usually occurs quickly. There is sudden loss of consciousness and speech, and paralysis of motion and sensation in one half of the body (sometimes after slight spasm). In lesions limited to the median portion of the pons, incomplete paraplegia, alternating facial paralysis, narrowing and insensibility of the pupils, and disorders of the special senses (taste, smell, or hearing) are produced. In some cases, all the characteristic signs are absent.

Hæmorrhage into the cerebellum.—The determination of the symptoms of hæmorrhage limited to the cerebellum is difficult, since the lesion or its sequences are frequently propagated to the pons, cerebellar peduncles, and medulla oblongata. Considerable hæmorrhages into the lateral lobes are manifested by obstinate headache, vertigo, vomiting, amblyopia, amaurosis, dilatation of the pupils, hemiplegia of the opposite side of the body and difficulty of speech. When the hæmorrhage involves the neighboring structures, it may give rise to general weakness and uncertainty of movement, disorders of co-ordination, general paralysis of the limbs, convulsions, contractures, rotatory movements, strabismus, and difficulty of deglutition and respiration.

Apoplectic paraplegia usually results from the combination of two distinct hemiplegias, and is distinguished from spinal paraplegia by the presence of motor paralyses of the cranial nerves. Double paralysis of cerebral origin results from symmetrical hæmorrhages (one old, the other recent) into the motor centres, from circumscribed lesions in the median portion of the pons or from aneurisms at the base. It may also be due to hæmorrhages into the lateral ventricles or towards the base. But, in these latter varieties, symptoms of irritation (spasms, contractures), lasting a short time, are followed by general paralysis, and the patient dies in profound coma.

Meningeal hæmorrhage.—Virchow has shown that the so-called intra-arachnoid apoplexies are usually the result of inflammation of the internal surface of the dura mater (internal pachymeningitis), and that the new-formed false membranes give rise to secondary hæmorrhages between their different layers from rupture of the rich capillary network which traverses them. But, in exceptional cases, primary hæmorrhages may also occur into or between the cerebral meninges.

The extravasated blood is situated in the cavity of the arachnoid and in the tissue of the pia mater, but may also invade different parts of the cerebrum or cerebellum, or may extend into the ventricles, to the base of the skull, and even into the cavity of the spinal arachnoid.

The blood is usually coagulated and small in quantity, but if rupture of a large vessel or aneurism at the base of the skull has occurred, the extravasation may equal a pound in weight. Œdema, flattening of the convolutions and softening may develop as secondary lesions.

As *etiological factors* we may mention injuries to the skull, rupture of a sinus of the dura mater, rupture of large degenerated vessels, and of aneurisms, and apoplexy of the new-born. Aneurisms of the cerebral vessels occupy the chief rank among the causes of this condition.

Meningeal apoplexies of the new-born after difficult labors result from mechanical obstruction to respiration and circulation, and after easy labors, from wounds of the vessels caused by the sliding of the bones of the skull over one another (F. Weber). The Vienna statistics show that these hæmorrhages are very frequent when the mother succumbs to diseases of a pyæmic nature. The infants are often still-born, or so deeply asphyxiated that they die soon after delivery. In most cases, somnolence, general muscular resolution, often also spasms and trismus, are produced. In nine cases reported by Elsaesser (Wuerttemb. med. Corr.-Bl., Dec., 1844), death only occurred at the end of from one to three weeks, after the sudden appearance of vomiting, convulsions, dyspnœa, and somnolence. The infants had evidently succumbed to secondary hæmorrhages, to cerebral inflammation, or to softening. Cases of recovery are of rare occurrence.

Meningeal hæmorrhages in the adult present such a diversity of symptoms that an exact interpretation is only possible in a small number of cases. They often present, in the beginning, symptoms of irritation of the meninges, such as headache, somnolence, delirium, mental excitement, spasms, or even epileptiform convulsions of an intermittent character. This condition is followed by symptoms of depression, such as coma and paralysis of the limbs (without the appearance of hemiplegia). Death occurs quickly, and is rarely delayed more than a few days. In the cases due to rupture of aneurisms at the base, the symptoms observed are pain in the occiput and neck, disorders of audition, deglutition, and respiration, alternate hemiplegia or paraplegic paresis, and rigidity of the arteries. When the rupture occurs in an aneurism of the middle cerebral artery, the principal phenomena are the slow, insidious march of the cerebral symptoms, epileptiform convulsions, and youthful age (of 17 cases, 6 were under thirty years). In rupture of an aneurism of the posterior communicating artery, the hæmorrhage is preceded, according to Gougouenheim (Aneurismal Tumors of the Cerebral Vessels, 1866), by symptoms of compression of the third, sixth, or even the fifth pairs of nerves (when the aneurism also involves the ophthalmic branch). Cerebral hæmorrhages of aneurismal origin are characterized, therefore, by tumor symptoms of long duration, followed by the sudden development of apoplexy. Spontaneous recovery is extremely exceptional.

Diagnosis and Prognosis.

When a patient, whose clinical history cannot be obtained, is found suffering for a short time from loss of consciousness and general resolution of the limbs, it is difficult to form an immediate diagnosis. In such

cases the advanced age of the patient, manifest rigidity of the arteries, the existence of cardiac or pulmonary disease, and the flaccid condition of one of the limbs, afford some grounds for the diagnosis of apoplexy. Cerebral hyperæmia, with its symptoms of irritation and of slight depression, usually subsides very quickly. Meningitis is distinguished from hæmorrhage by the initial chill, appearance of convulsions, cutaneous hyperæsthesia, and increased reflex irritability added to coma, and by the incomplete paralyses which slowly appear from increase in the intracerebral pressure.

In cerebral tumors, the headache is intermittent, and gradually increases; neuralgia, spasms, and neuroretinitis often occur. These symptoms ordinarily precede the apoplectiform attacks, which occur rarely, and persist after the latter have disappeared. The characteristic signs of rupture of cerebral aneurisms are chronic development of multiple paralyses of cranial nerves and convulsive symptoms, followed by sudden and, usually, fatal attacks of apoplexy.

The apoplectic hemiplegia, with which anæsthesia of the deep tissues is sometimes combined, presents great resemblance to those forms of hysterical hemiplegia with loss of consciousness which occur after emotional excitement.

In apoplectic hemiplegia, the median line forms the boundary of the anæsthesia and analgesia; in hysterical hemiplegia, we often find anomalies of sensation on the other side of the body. In apoplectic hemiplegia, also, paralysis of the respiratory fibres of the facial nerve and of the muscles of the tongue, and aphasia, often occur while these phenomena are not met with in hysterical hemiplegia. In the former, the affected thigh is balanced from behind forward in walking, contracture of the extremities only occurs after paralyses of long standing, and the electro-muscular contractility is preserved. In hysterical hemiplegia the patient drags the thigh, in walking, like an inert mass, contracture develops more rapidly, and the electro-muscular contractility diminishes after it has lasted for a certain length of time. Disorders of menstruation, or irritation of the uterus or ovaries coexist, in hysterical hemiplegia, and characteristic attacks have usually occurred previously, attended with hiccough, tympanites, retention of urine, abnormal tenderness of the spinal column, etc.

In spinal hemiplegia, from a lesion of one of the lateral halves of the cord, one side of the body is paralyzed, but its sensation is preserved; the electro-muscular contractility is diminished, and reflex excitability increased; upon the opposite side, sensation is lost but motion and electro-muscular contractility are intact. With regard to *prognosis*, we must consider not only the site of the hemorrhage but also its amount. The increase of pressure fatal to life corresponds, according to Leyden, to the vascular tension; it is 180 milligrammes of mercury or even upwards. The increase of the cerebral compression may cause paralysis of the respiratory centre, although the heart may still continue to act for a short time.

If the intracerebral pressure diminishes and the circulation becomes regular, improvement will become manifest by the return of consciousness and speech, by more quiet movements of the heart and respiration, by the ability to swallow, by the return of motion and sensation, as well as by the moderate elevation of the temperature. On the other hand, the persistence of coma for 36–48 hours, complete loss of reflex excitability, irregular, noisy respiration, small, intermittent pulse, general resolution of the limbs, relaxation of the sphincters, and dilatation of previously con-

tracted pupils, are signs of bad omen. Among the early signs of a fatal termination, according to Bourneville, are a lowering of temperature (36° and 35·4° C.) for several hours after the attack, followed by elevation to 41° and 42·8° C., after a short stationary period, and, finally, according to Charcot, the appearance of bed-sores during the first few days.

After the immediate consequences of the hæmorrhage have been safely passed, the inflammatory reaction may again place the patient in danger. The patients then complain of a feeling of constriction in the head, dizziness, soon followed by delirium, disturbances of the special senses, numbness and spasms or contracture in the paralyzed limbs. Death usually occurs in the third or fourth week. The prognosis is also rendered more grave in old and weak patients suffering from cardiac affections or, pulmonary emphysema, or from marasmus which has led to uncontrollable diarrhœa, hypostatic pneumonia, cystitis, bed-sores, etc. During convalescence, also, cerebral atrophy may occur and slowly give rise to imbecility. Finally, there is danger of a relapse.

The prognosis of paralyses which persist after the attack is not very favorable. In the rare cases of slight hæmorrhage, situated at a distance from the motor ganglia or in which their fibres have been very slightly involved, hemiplegia and hemianæsthesia may completely disappear or the limbs may only exhibit a slight degree of paresis. In weak, cachectic subjects, after repeated hæmorrhages, and in old hemiplegia with progressive muscular atrophy of the extremities, with multiple contracture and lowering of temperature, there can be no question of any considerable and permanent improvement.

Treatment.

When the attack is light, it will be merely necessary to apply frequently changed cold compresses to the head, and to keep the head elevated. The bed and room should not be too warm, and no nourishment should be given except cool, acidulated drinks. If the attack occurs after a hearty meal, and if vomiting does not take place spontaneously, it may be hastened by the introduction of a finger into the pharynx. When obstinate constipation exists, purgative enemata should be administered mixed with vinegar, sulphate of soda, or infusion of senna.

If the symptoms of increased vascular tension (which goes hand in hand with increase of cerebral compression) appear after the attack; if there is turgescence or intense redness of the face, or considerable injection of the conjunctiva, if the carotids and radials pulsate with force, and if the temperature is raised, venesection should be immediately performed in strong, well-nourished individuals. Venesection should also be performed if signs appear which indicate a threatened second hæmorrhage. In weak, cachectic patients, with cold skin and feeble pulse, the employment of leeches and cold applications should be substituted for venesection. Cutaneous revulsives are superfluous in light cases and useless in severe ones. The stimulant measures, which we have previously indicated, should be employed to combat the symptoms of depression.

The violence of the irritative symptoms and of the convulsions in the stage of inflammatory reaction, may indicate venesection. But usually, leeches applied repeatedly to the mastoid processes and the application of cold compresses, or of an ice-bag to the head, will prove sufficient. In general, moderate intestinal derivation has a better effect than cutaneous revulsives. Cold ablutions and small doses of opium are of benefit in violent excitement and in insomnia.

The conditions of chronic weakness following apoplexy necessitate a quiet life, strict regimen, and light but good diet. Weak and aged patients may take a little wine, and must have the functions of the bowels, bladder, and respiratory organs well regulated. In summer, we may recommend a residence in the country, moderately cold half-baths, frictions with a moist cloth, and moderate exercise. In slight lesions of the brain, when resorption and cicatrization have progressed favorably, the incomplete paralyses are, at least, susceptible of improvement. We should advise the use of baths (24–26° C.) only after the irritative symptoms have disappeared for a long time (about six months), and if signs of congestion have not reappeared. In patients suffering from organic disease of the heart or rigid arteries, and who are very susceptible to the exciting action of heat, we should prohibit warm baths, which might expose them to the danger of a fresh hæmorrhage.

Hydropathic treatment exercises a favorable influence upon the general condition in a large number of apoplectics, but we must avoid the extremes of temperature. Individuals predisposed to congestions are easily excited by moist and cold frictions. We should then employ half-baths of short duration, and not too cold, and should gradually lower their temperature. In less impressionable subjects, benefit will be derived from moist and not too cold frictions, and from half-baths.

The electrical treatment of the paralyses may be begun, in the slight forms, at the end of two months. In more severe cases, electricity should not be employed until the disappearance of all the symptoms of irritation —perhaps in from four to six months after the apoplexy. The continuous current, which possesses an irritant effect upon the nervous centres and organs of special sense, should only be applied with great caution to the head and neighboring parts.

The faradic current is preferably employed in paralyses of the extensors with predominance of the flexors. In long standing disorders of sensation we may use the electric brush. Faradization may give good results in slight muscular atrophy and in contracture of the flexors. In the treatment with galvanism, descending currents should be employed from the dorsal spine to the nerves of the paralyzed or contractured muscles. The current should produce moderate muscular responses, and its intensity may be then gradually increased. Three or four séances, lasting from five to eight minutes, are to be given per week.

In severe forms of paralysis we may obtain improvement by alternately employing galvanization of the centres with faradization of the extremities; and, at other times, by combining hydrotherapeutic measures with electricity. Despite all treatment, however, the proportion of recoveries is very small.

CHAPTER IV.

SEROUS EXUDATIONS INTO THE BRAIN.

1. Cerebral Œdema.

Serous infiltration of the cerebral parenchyma appears, in various diseases of the brain, as a circumscribed process of slight intensity. A cut section of the white substance then shows an unusual moisture and brightness, with an almost imperceptible change of consistence. When the œdema is more marked and general, the medullary substance is infiltrated with fluid of a doughy consistence and a dull, shining, white color. In very intense acute infiltration of the subarachnoid spaces and ventricles, and in lesions "en foyer," the cerebral substance is reduced to a pulp and nearly liquefied, bathed in fluid, and at times of a white color, at times colored yellow in the neighborhood of a hæmorrhage or encephalitis, from absorption of the coloring matters of the blood.

Chronic cerebral œdema may, little by little, cause a true maceration of the brain, as has been observed in certain cases of senility and idiocy, with all the signs of moral and physical decrepitude.

Acute cerebral œdema may cause sudden death by rapid increase of the compression and volume of the brain.

It is a matter of experience that in diseases of the heart and kidneys, in bronchitis and chronic tuberculosis, suddenly fatal cerebral compression may develop, and nothing be discovered on autopsy beyond considerable serous infiltration into the brain, and, sometimes, hyperæmia of the meninges. The patients suddenly lose consciousness, fall to the ground, the muscles are relaxed, sphincters paralyzed, reflex irritability considerably weakened, the contracted pupils reacting slightly or not at all. Delirium usually appears, respiration and deglutition become more and more irregular and difficult, and death occurs in coma at the end of a few hours or days.

The diagnosis of this condition is always very uncertain. Even pathological anatomists (Rokitansky, Lehrb. d. path. Anat., II Bd., p. 452–454) admit the existence of serous apoplexy in the cadaver with a great deal of reserve.

2. Hydrocephalic Processes.

In measuring the quantity of fluid contained in the brain we must not lose sight of the fact that it may vary under certain physiological conditions such as age and sex. Weisbach (Med. Jahrb., XVI., Bd., 1868), has shown that the proportion of water gradually diminishes from birth until the 20th year, and then again increases. It is more abundant in the male sex. In adults the gray matter is richer in water than the white substance; in the new-born the reverse holds good.

Acute diseases generally cause increase of the quantity of water in the body, and especially in the brain. The cerebral fluid decreases in chronic diseases, the diminution occurring in the cerebrum in men and in the cerebellum in women. It attains its maximum in meningitis and in chronic hydrocephalus.

External hydrocephalus and œdema of the pia mater possess merely an anatomical interest. Internal hydrocephalus may be divided into three varieties, viz.: acute, chronic, and congenital.

a. *Acute Hydrocephalus.*

The effusion of serum into moderately distended ventricles varies from 15–70 grammes. In larger infiltrations the ependyma is softened and macerated, the adjacent cerebral tissue, especially the septum and fornix, deliquesce, and the parenchyma is often congested and strewn with small hæmorrhages. If the fluid is still more abundant, the brain is enlarged and softened, the medullary substance pale and anæmic, and the convolutions are flattened.

Acute hydrocephalus a is very frequent disease of infancy. With the large proportion of water contained in the brain of nursing infants, the frequent hyperæmias which may arise in tuberculous, scrofulous, and rachitic affections may readily lead to sudden or progressive (subacute) serous transudations.

Tubercular meningitis, cerebral tumors, and pulmonary phthisis may also be complicated by acute hydrocephalus. Acute pulmonary diseases, according to Weisbach, affect by preference the amount of water contained in the medullary substance and cerebellum.

In the less severe forms absorption of the fluid is possible, but some fluid ordinarily remains in the enlarged ventricles and may give rise to chronic hydrocephalus. The diagnosis of acute primary serous effusions within the cranial cavity is difficult and uncertain. The symptoms of serous apoplexy, which appear in these cases, have already been mentioned.

If the irritative phenomena predominate, the treatment should consist of cold applications and bleeding, when not contraindicated by anæmia. Bleeding should not be resorted to if we suspect considerable venous stasis. We may also employ derivatives, cutaneous revulsives, and small doses of iodide of potassium or iodide of iron. If coma supervenes, stimulant measures are useless.

b. *Chronic Hydrocephalus.*

In this variety the serous transudation is more abundant (200–400 grs.), and the symmetrical or unsymmetrical dilatation of the ventricles is more considerable. Thickening of the ependyma, condensation of the adjacent cerebral tissue, flattening of the optic thalamus and corpus striatum, and breaking down of the upper wall of the lateral ventricle, are noticeable.

Chronic hydrocephalus in infants usually follows the acute form. In middle life it is caused by mechanical hyperæmia, the pressure exercised by tumors (upon the sinus rectus, Barrier), the occlusion of one or both lateral sinuses, tubercular products in the cerebral hemispheres, compression of the vena Galeni and of the middle ventricle and its neighborhood,

and chronic diseases with dropsy. In old age, hydrocephalus follows the cerebral atrophy occurring after hemorrhage or encephalitis. Insanity, especially dementia, is often complicated with chronic hydrocephalus.

As ætiological conditions in infants we may mention rachitis, and sometimes congenital syphilis, with abnormal conformation of the cranial bones, or diminished resistance to the internal pressure. At a more advanced age it may arise from the chronic cerebral hyperæmias which are produced by alcoholism and prolonged mental excitement, and in the course of meningitis. It may also be due to disturbances of the circulation from cardiac or pulmonary diseases, from Bright's disease, and from chronic tuberculosis as well as from changes in the blood, such as leukæmia and hydræmia.

The symptomatology presents great variations. In the initial period there are uncertain symptoms of cerebral irritation which gradually assume the characters of depression. The special senses and intellectual faculties gradually become blunted, the head becomes heavy and drooping, the expression of the face, which was at first more animated, becomes apathetic; pareses occur in the domain of the facial nerve with ptosis, inequality of the pupils, and difficulty of speech; the gait is uncertain, heavy, and tottering.

A large number of these patients arrive at a continually increasing stage of stupor, with all the symptoms of idiocy with paralysis; sometimes epileptiform attacks, which had never been previously observed, also occur. Bouchut has found atrophy of the optic nerves (with flattening of these nerves and of the chiasm) in the chronic hydrocephalus of infants (Gaz. des Hôpit., 1872). Finally, the patients become weaker and weaker, and consciousness entirely disappears; motion, sensation, and reflex excitability are lost. Or acute transudation may occur, and sudden loss of consciousness develops with violent pains in the head, mental weakness, inequality of the pupils, strabismus, slow, irregular pulse, and complete or incomplete hemiplegia (from compression of the motor ganglia on the side of the affected ventricle). There is no hope of a permanent arrest of this affection. The disease is incurable and may last a few months, rarely several years. Death occurs from cerebral compression or general paralysis, or from complication with meningitis, pneumonia, cystitis, decubitus, or from a fresh fatal exudation. The treatment will be indicated in the following section.

c. *Congenital Hydrocephalus.*

Congenital external hydrocephalus, meningocele, anencephalie, hemicephalie, and hydrencephalocele possess a greater interest for the pathological anatomist than for the neuropathologist.

Chronic hydrocephalus, in the variety usually known as congenital, either exists from birth or develops with the greatest intensity soon after delivery. The skull is then increased in size (rarely diminished, as in premature union of the sutures in cretins), the bones of the cranial vault are enlarged and thinned, the sutures are wide, the fontanelles gaping, and their membranes are strewn with bony points. The skull is distended, and the superciliary region, the squamæ of the temporal bone, and the occipital bones are pushed downward to a considerable degree.

The meninges are markedly softened and thinned; the cerebral hemispheres are pressed against one another by the distention of the ven-

tricles, and they may be reduced to the thickness of a sheet of paper; the convolutions are barely indicated, and the white and gray substance cannot be distinguished from one another. The optic thalamus, corpus striatum and lenticular nucleus are pressed downward; the peduncles are separated; the optic chiasm is flattened; the pons varolii and tubercula quadrigemina are elongated; the third ventricle is enlarged (like the lateral ventricles); the fornix and commissures, the corpus callosum, and the septum are thinned, distended, or torn. The superior portion of the cerebellum is flattened, together with the nerves at the base of the brain. The ventricular fluid contains albumen and chloride of sodium, and may amount to 2–4 kilog. and upwards.

The *symptomatology* varies with the changes in the cerebral structures. The slight forms, in which the cranial deformity is barely perceptible, may exist for a long time without giving rise to any symptoms. In the severer forms, with considerable distention and deformity of the skull, hydrocephalus is especially recognized by the striking contrast between the increased volume of the skull and the small dimensions of the face. The depressed sunken eyes, which are closed with difficulty, the disproportion of the body, and the slight development of the limbs, give the patient a characteristic appearance.

Examination of the skull shows that the fontanelles are gaping and the sutures ununited. Bright, Watson, and others have seen the skull translucent.

It is rare that the cerebral functions are only slightly affected, and the mental condition generally corresponds to the physical deterioration. The children comprehend poorly and with difficulty. They may evince a certain aptness, but no real mental activity; and even after they have grown up, speech remains difficult, and intelligence and memory are poor. They possess no energy, and their intellect retains the qualities of childhood. In more severe cases they soon become completely imbecile, but the power of motion may be retained to a greater or less extent; they utter inarticulate cries, laugh unmeaningly, etc.

In severe cases the functions of the special senses are entirely abolished. The children appear to develop normally, but upon careful examination they are found to be deprived of sight and hearing, and the pupils are dilated and react slowly to light. In less severe forms, myopia, asthenopia and strabismus occur; hearing, taste, and tactile sensibility are intact or only slightly affected, though many hydrocephalic patients eat disgusting articles with avidity. Headache usually occurs in paroxysms, and is especially intense in ossification and thickening of the skull, and during mental or physical excitement. We may attribute it to increase of cerebral compression.

Disturbances of motion are also often observed. The majority only learn to walk after the lapse of several years, and the gait is tottering and unsteady. They experience great difficulty in keeping the head erect. In certain severe forms they are neither able to walk, stand, nor use the upper limbs. Very often there is only hemiparesis or pareses of certain limbs, or the patient is bow-legged on account of the frequent complication with hydrorachis. Electro-muscular contractility is not appreciably changed. The motor disturbances of irritative origin are as follows: strabismus, contractures, spasms of the face or limbs, tremor, convulsions, and epileptiform attacks.

Digestion is good, even in cases of gluttony; vomiting only occurs after violent shocks to the head or after great excitement. The patients

are usually constipated. The general nutrition is very poor; the patients are pale and bloated, the abdomen tympanitic, the skin dry, and the muscles atrophied.

The pathogeny of congenital hydrocephalus is still obscure in many particulars. During intra-uterine existence the congestions of the ependyma—which, according to Rokitansky, are often connected with congestion of the choroid plexuses, pia mater, and cerebral tissues in the neighborhood of the ventricles — may give rise to increased effusion of serum into the ventricles, and to changes in the ependyma and adjoining brain-tissue. The existence of an intra-uterine predisposition is proven by the fact that one mother may give birth to several hydrocephalic children, and that drunkenness in the father acts as a cause of congenital hydrocephalus. During extra-uterine existence, and even after ossification of the sutures, inflammatory irritation of the walls of the ventricles may give rise to serous transudation, an hypothesis which harmonizes with the frequent existence of vascular meshes, thickenings, and false membranes upon the ependyma. Congenital hydrocephalus runs a chronic course. The volume of the skull may increase gradually from birth, or it may remain stationary for a certain length of time. Partial absorption of the fluid may occur, especially after ossification of the deformed skull has taken place. The duration of the disease is extremely variable. The large majority of the patients perish at birth or during the first years of life, although, in a very few instances, life is prolonged to puberty and, still more rarely, to an advanced age.

Death may happen in the first years of life from cerebral compression from cachexia, from ependymitis or meningitis, or from intercurrent febrile diseases (bronchitis, pneumonia, tuberculosis, intestinal affections). Rokitansky saw a case of hydrocephalus terminate in perforation of the brain and dura mater, and in the passage of the ventricular fluid underneath the pericranium and epicranial aponeurosis. According to him the disease also frequently terminates from hæmorrhages into the arachnoid sac and ventricles. In most cases death occurs in coma, preceded by convulsions.

The *diagnosis* is usually not difficult. Simple cranial rachitis never causes such marked mental and physical deterioration as congenital hydrocephalus. In adults, we must inquire into the various cerebral disorders which are capable of producing hydrocephalus. Cerebral hypertrophy, with increase in the volume of the skull, will be almost inevitably mistaken for hydrocephalus, especially in adults and after ossification of the cranium. The absence of rachitis, the slight disturbance of the intellectual faculties and of nutrition, the early appearance of convulsions and of epileptiform attacks, point rather towards cerebral hypertrophy than towards hydrocephalus.

The *prognosis* is not favorable. Recovery from complete absorption of slight effusions or from the escape of the fluid through a perforation in the meninges, is an extremely rare event. Even when partial absorption occurs, there is danger of relapse or of fresh congestions. In the chronic form, the disease is the sad precursor of inevitable physical and moral deterioration.

The *treatment* must, in general, be directed towards combating the most annoying symptoms. Intestinal derivatives, diuretics, absorbents, and active cutaneous revulsives, are more injurious than useful. Methodica. compression of the head by means of strips of adhesive plaster, as recommended by Engelmann and others, has not yielded any good results

Puncture, advised by Conquest and others, is performed with a fine trocar at the border of the anterior fontanelle in the coronal suture. Langenbeck prefers to pass behind the upper lid through the superior wall of the orbit, and thus to enter the anterior horn of the lateral ventricle. But, as a rule, the inflammatory complications attending these operations, render their palliative utility very doubtful. If the patient recovers from the meningitis following the puncture, the hydrocephalic fluid becomes rich in albumen (Dickinson).

CHAPTER V.

CEREBRAL ANÆMIA.

Pathological Anatomy and Experimental Investigations.

In cerebral anæmia, the meningeal vessels are usually empty and markedly collapsed. The vascular canals of the dura mater and the large venous trunks appear to be better filled. In some cases, hyperæmia of the meninges coexists with anæmia of the brain. The pia mater is often thin and transparent and its tissue, together with the ventricles, contains a considerable quantity of serum. The medullary substance is of a dull white color and presents few or no puncta vasculosa upon a cut section. The gray substance is unusually white and poorly defined, and the cerebral parenchyma is generally dry and firm, rarely moist and soft. This condition is found, as a rule, in those cases in which the cerebral anæmia is one of the manifestations of general anæmia. Partial cerebral anæmia is met with in localized lesions, in tumors, large hæmorrhages or encephalitis, and in obliteration of certain vascular territories of the brain in consequence of thrombosis or embolism (ischæmia). In all these cases, the local compression or circumscribed circulatory disturbances give rise to partial cerebral anæmia which is often compensated by hyperæmia in other parts of the brain.

The most recent experiments of Kussmaul and Tenner have clearly shown that, when the carotid and vertebral arteries on both sides are compressed, intense cerebral anæmia (with epileptiform convulsions) will occur from irritation of the centre of vascular innervation. Toxic doses of belladonna, morphine, chloroform, ergotine, nicotine, etc., cause pallor of the fundus of the eye, and lowering of the intra-cranial temperature, as well as cerebral anæmia, from irritation of the vaso-motor centre (which, according to Jolly, accompanies increase of intra-cerebral pressure.) Finally, faradization of the cervical sympathetic (Kussmaul), irritation of peripheral nerves (Loven and Nothnagel), and electrical or mechanical irritation of the walls of the stomach (S. Mayer and Pribram) may also cause reflex narrowing of the arteries and cerebral anæmia.

Etiology.

Cerebral anæmia is observed in infants from a few months to two or three years of age (Marshall Hall's hydrocephaloid), and is caused by poor and insufficient nutriment (bottle-fed infants), and by profuse hæmorrhages (poorly performed circumcision, ill-timed antiphlogistic measures). In these cases the cerebral anæmia is merely a part of the general loss of blood, and its gravity is due to the peculiar weakness and irritability of the infantile brain, and to the slight power of resistance of the youthful economy. At a more advanced age, various morbid condi-

tions may give rise to cerebral anæmia. Copious hæmorrhages (of the lungs, uterus, intestines, stomach, etc.) occupy the chief rank among the causes of this condition. It may also be due to the condition of exhaustion following typhoid fever, dysentery, and puerperal diseases, etc.; to abundant secretions, as in repeated or prolonged lactation, and to anomalies or insufficiency in the production of blood, as in leukæmia, chlorosis, tuberculosis, malarial cachexia, etc.

Powerful irritation of the centre of vascular innervation also produces contraction of the cerebral arteries, and the irritation may be propagated to the neighboring centres in the medulla oblongata. These vaso-motor disturbances usually last but a short time. As the action of the vaso-motor nerves quickly subsides, the excitation and anæmia are soon replaced by depression and hyperæmia, but, on account of the periodical return of the vascular spasm, the anæmia may acquire the upper hand during a certain period. The preceding remarks apply especially to hysteria. Strong and sudden emotions, prolonged and depressing moral causes, also present the same alternation of symptoms (pallor, vertigo, insensibility of the senses, weakness of the heart's action, followed by the opposite signs of hyperæmia).

Vaso-motor disturbances and temporary cerebral anæmia, with syncopal attacks, may be produced by reflex means, as in cerebral concussion and shock. Symptoms of irritation of the vaso-motor centres are sometimes observed in chronic catarrhal affections of the stomach and intestines, and in constipation, especially in impressionable individuals (vertigo stomacal, vertigo a stomacho læso).

As another variety of reflex cerebral anæmia, I may mention the sudden pallor, vertigo, nausea, and slow pulse, followed by symptoms of hyperæmia, which sometimes occur in very nervous females upon the application of strong electrical currents to the cervical vertebræ, and in cases of spinal irritation after the painful action of the galvanic current upon the skin of the back or other regions.

Symptomatology.

The symptoms, in infants, are very similar to those of acute hydrocephalus (hydrocephaloid). Marshall Hall distinguishes the stages of irritability and depression. In the first stage the head is hot, the face red, the temperature of the body considerably increased, and the pulse and respirations are slowed; there is great excitement; exaggerated sensibility of sight and hearing; sleep is short, and frequently interrupted by groans, cries, and starts; the abdomen is tympanitic; the stools are of a mucous character and irregular. In older children delirium is sometimes observed in this period.

In the stage of depression the symptoms of cerebral anæmia proper make their appearance. The features are relaxed; the face remarkably pale and cold; the eyelids remain half closed, even when the lashes or ball of the eye are touched; the pupils do not react; the organs of special sense are insensible; the respiration is noisy, and becomes slower and slower.

In *older patients* the symptoms differ a great deal, according as the cerebral anæmia is acute or chronic, and according as smaller or larger vascular territories of the brain are subjected to spasm or paralysis. In rapidly developed cerebral anæmia, the features are relaxed; the face pale and cold. We also observe vertigo, nausea, dimness of vision, loss of

consciousness, relaxation of the limbs, subsultus, and feebleness of the respiratory movements and cardiac action.

In *anæmia from lack of nutrition* (which usually develops slowly), we may observe, in slight cases, frequent headaches (in the form of migraine), vertigo, roaring in the ears, flashes of light, impairment of ideas, dread of mental work, exaggerated sensibility to external impressions, muscular weakness and tremor after movements. The skin and mucous membranes are pale, the appetite poor, and sleep disturbed. In the severer forms, attacks of syncope and convulsions may occur; the pupils are dilated and do not react; and the respiration and movements of the heart are slowed. Increase of drowsiness is one of the most unfavorable symptoms in cerebral anæmia. In some cases the symptoms mentioned alternate with those of hyperæmia (temporary redness of the face, flashes of heat, increased vigor of the heart's action).

In conclusion, we may mention two distinct forms of cerebral anæmia, viz., cerebral concussion and shock.

Cerebral concussion of traumatic origin is accompanied by loss of consciousness, and insensibility of the organs of special sense; the pupils are dilated and react slowly; the voluntary muscles are paralyzed; the respirations are slow and deep; the pulse is irregular with intermissions; finally, vomiting occurs. Consciousness may be restored after several hours, rarely only after the lapse of a few days. The functions of the organs of special sense, motion and sensation, return to their normal condition, some slight symptoms (headache, redness of the face, slight acceleration of the pulse, and weakness) persisting for a little while.

Nothnagel's experiments seem to show that mechanical irritation produces reflex spasm of the cerebral arteries, and increase of the intra-cranial pressure from irritation of the vaso-motor centre. But, according to Fischer (Volkmann's Sammlung klinischer Vorträge), the symptoms of concussion are due to a reflex paralysis of the cerebral vessels in the same way that mechanical irritation of the abdominal viscera (in Goltz's experiments) produces slowing and even arrest of the heart's action.

Diabetes mellitus often occurs in cerebral concussion; in some cases, diabetes insipidus, and still more rarely albuminuria, have also been observed. These symptoms are probably due to irritation of the medulla oblongata between the origins of the pneumogastric and acoustic nerves (Schiff's centre of vascular innervation of the kidneys).

By the term *shock*, we refer to the paralyzing influence of any sudden and severe nervous lesion upon the action of the heart. Fischer distinguishes the *erethistic* form with symptoms of furious delirium (prostration with excitement, according to Travers), and a *torpid* form, characterized by the syncopal condition peculiar to cerebral anæmia. The erethistic may pass into the torpid form, and *vice versâ*.

Weak, impressionable individuals are very liable to shock. Copious hæmorrhages, contusions of the thorax, abdomen, testicles, and fingers, reduction of hernia, cystotomy, etc., especially predispose to its production.

Goltz's experiments lead us to believe that shock results from reflex paralysis of the vascular nerves, especially of the splanchnics (according to Fischer), following traumatic concussion. The stasis in the abdominal veins, and the afflux of a large proportion of the entire mass of blood into the dilated vessels of the intestinal tract, in consequence of the reflex paralysis of the intestinal vaso-motor nerves, cause anæmia of the brain, skin, and muscles, and thus leads to all the grave symptoms of shock which have been mentioned above.

Diagnosis and Prognosis.

In young children the condition may be mistaken for cerebral congestion and acute hydrocephalus—an extremely serious mistake as regards treatment. When the symptoms above mentioned are present, especial attention should be paid to previous debilitating conditions (poor, insufficient diet, persistent diarrhœa). If the cheeks and limbs are pale and cold, if the eyelids are half-closed, and do not respond to stimuli, if the pupils are dilated, and scarcely react, if there is no strabismus or true febrile symptoms, if the head is heavy and painful, if there is somnolence, hoarseness of voice, and a dry, hacking cough from time to time, the ensemble of all these symptoms, and the previous history of the patient, should prevent the physician from committing an error.

Previous hæmorrhages, debilitating exudations or secretions, abnormal or insufficient production of blood, depressing psychical influences, and general poverty of the blood, facilitate the diagnosis of cerebral anæmia in the adult. As regards the prognosis in the cerebral anæmia of infants, the diminution in the frequency of the pulse, the greater regularity of the respiration, increased sensibility of the pupils to light, returning warmth of the skin, greater interest in surrounding objects, more complete closure of the eyelids and a more animated expression may be considered indicative of a favorable termination. But if, on the other hand, the somnolence passes into coma, if the coolness of the skin increases, if the eyes become sunken, the pulse rapid, soft and wiry, if reflex excitability is abolished, and the respiration becomes irregular, intermittent, and finally noisy, we have every reason to fear a fatal termination.

In adults the prognosis is favorable if we are able, in a measure, to remove the causes of the cerebral anæmia. In general, the anæmia from defective nutrition is slower in appearing, but more persistent in its effects. On the other hand, anæmia from nervous irritation develops quickly and disappears in a similar manner. Cerebral concussion may prove rapidly fatal, or may even, according to Pirogoff, last several weeks. In many cases, feebleness and mental weakness may persist for some time.

Treatment.

During the stage of excitement, warm baths are very useful. Small doses of hydrate of chloral are serviceable in anæmic convulsions; in nursing infants, 0.20–0.40 may be given daily in a little milk. If there are signs of decline, warm applications should be made to the abdomen, and the patient given plenty of fresh air; diarrhœa, when present, may be combated by the use of laudanum. Then employ active stimulants (a little red wine, a few drops of brandy or aromatic spirits of ammonia in a draught of milk (M. Hall) every two or three hours,) and continue these measures until a positive improvement is evident. In infants at the breast, the milk of a young and well-nourished nurse constitutes one of our chief resources. In extreme cerebral anæmia, due to copious hæmorrhages, we may frequently obtain rapid improvement by means of transfusion. But, more frequently, it is necessary to restore the wavering activity of the brain by the aid of stimulants, such as strong wines, hot tea, coffee, and rum, acetic ether upon a lump of sugar, ammoniacal preparations, and musk. In prolonged syncope and in shock, good effects are

often seen from faradization of the phrenics when respiration is suspended, but the heart still continues to act. In cerebral concussion we must avoid active measures, and moderate stimulants will usually suffice. In chronic forms, in which the cerebral anæmia is only an effect of the general poverty of the blood, our remedies should be directed against the latter. Animal diet, ferruginous waters, country air and, later, the use of mild hydropathic measures and of sea-baths, furnish good results. Very impressionable or debilitated individuals should be treated with dialyzed iron, spoonful doses of some ferruginous water, or a small quantity of citrate of iron during meals.

CHAPTER VI.

CEREBRAL INFLAMMATION (ENCEPHALITIS).

Pathological Anatomy.

INFLAMMATION of the brain is never general; it always exists under the form of circumscribed "foyers," which sometimes occupy the superficial layers, and sometimes the deeper structures of the cerebrum (cerebritis) or cerebellum (cerebellitis). The foyers, which vary in number and dimensions, are situated in the white substance, or, more frequently, in the gray matter of the cortex and cerebral ganglia. If the foci are large, the cerebral substance is infiltrated with fluid, softened, swollen, and strewn with red spots and capillary hæmorrhages, the convolutions are flattened, and the surrounding cerebral parenchyma and meninges are dry and anæmic.

In congenital encephalitis, which sometimes originates during intrauterine life, but can be recognized only after the lapse of several weeks or months after birth, the lesion consists, according to Virchow, in a fatty change of the neuroglia cells. The cells are enlarged and filled with fatty granulations, especially in the white substance of the cerebral hemispheres and in the columns of the cord.

In many cases the parenchyma is not sensibly altered. The spots which have suffered the greatest change and in which there is a considerable accumulation of fatty granules, present, to the naked eye, a dull grayish-white, opaque appearance. The white substance is grayish-red from capillary congestion; the gray substance, on the contrary, is whiter than usual. The consistence is not modified unless there is a coexisting change in the nervous substance (especially softening).

The vascular sheaths and the walls of the capillaries often present fatty degeneration. The vessels are then usually unequally dilated, and sometimes obstructed in their smaller subdivisions. According to Hayem, fatty degeneration of the neuroglia-cells is only indicative of inflammation when we find, at the same time, extreme congestion and granular corpuscles present. Virchow maintains, in opposition to Hayem, that the proliferation of the vessels is an important anatomo-pathological sign when present, but that its absence in the cadaver does not disprove the inflammatory character of the process during life. The increase in size, the nuclear division, and even cell-proliferation which precedes the fatty degeneration of the neuroglia-cells, the coexisting inflammatory changes in the parenchyma of other organs, as the kidneys, and the frequent production of cerebral softening from inflammatory diseases, such as variola, scarlatina, and syphilis—all these facts lead us to regard the process under discussion as inflammatory in its nature.

Independently of the inflammatory character of the whitish-gray or yellowish foci, there is also, according to Virchow, a peculiar kind of softening. In these foci of softening we observe a characteristic variety of

fusiform corpuscles, which are only visible on careful examination, in the midst of the myeline within the undestroyed nerve-tubes. They constitute the so-called varicose hypertrophy of the axis cylinder.

According to Hayem, irritation of the connective tissue elements is also a cause of cerebral inflammation. An exudation then occurs into the irritated elements of the connective tissue, from which it is propagated to the nuclei, to the amorphous reticulum of the neuroglia, and to the capillaries. In a case of spontaneous encephalitis, Hayem found—in addition to inflammatory foci in the corpus striatum and optic thalamus—a large number of cellular or nuclear elements in the interstitial connective tissue and transition forms between these and fatty granules, with fatty infiltration of the walls of the vessels. Exudations, with a large proportion of fluid, may present the appearances of œdema. Hayem distinguishes three varieties of interstitial encephalitis, viz., a *suppurative form*, in which we find pus-globules, and which is usually acute, rarely chronic; a *hyperplastic form*, which gives rise to indifferent elements and runs a subacute course; and, finally, a *sclerotic form*, in which the neuroglia becomes transformed into connective tissue, and which is almost always chronic. Each of these varieties may be limited to one spot or disseminated, circumscribed, or diffuse, and may exist as primary or secondary affections.

The inflammatory processes just described are also found in several forms of insanity and in the encephalitis of old age. Division and absorption of the exudation, retrogressive changes and sclerosis, or even suppuration follow, more or less rapidly, upon the above-mentioned inflammatory processes. The phenomena of absorption consist, according to Virchow, in the filling of the pre-existing cells of the neuroglia and of the new-formed cells with fatty granules (Gluge's inflammatory globules). This process is not peculiar to inflammation, but has also been observed by Tuerck in encephalitis, with atrophic degeneration of the central nervous system. When the inflammation goes on to suppuration, the pus usually forms in the medullary substance, especially in the cerebral hemispheres, and, according to Lebert, oftener on the left than on the right side. The surrounding cerebral tissue is softened and œdematous, and the affected portion of the brain is swollen and anæmic. When the pus penetrates to the surface of the brain, the cortical substance also becomes involved. The cerebellum, which is rich in gray matter, is very liable to abscess formation. The cavity of the abscess is rounded, usually filled with a thick, greenish, inodorous (but sometimes fœtid) pus, and with the débris of the parenchyma. The contents vary from a few grammes to 200 grammes or more, and the volume of the abscess from the size of a pea to that of an apple. In rare cases an entire hemisphere has been found transformed into a large collection of pus. The purulent foyer is usually single, but sometimes several foci are present.

A diffuse abscess of the brain lies in immediate contact with the surrounding, softened cerebral tissue. In most cases it extends far and wide, and forces its way outward, setting up an inflammation of the sinuses and meninges. In rare cases it may appear at the exterior of the skull, or force its way into the cavity of the tympanum or into the nasal fossæ. More frequently, however, it opens in the direction of least resistance (towards the ventricles) and the fatal issue soon occurs.

Cerebral abscesses become encysted in the majority of cases. The cyst-wall is formed of a very vascular membrane, which is, at first, thin and soft; from the second to the third months it becomes thicker and more resisting, and is composed of two or three layers, the external layers

being fibro-cellular in structure, the internal ones presenting a mucoid appearance. In very rare cases, recovery may occur from absorption and partial calcification of the purulent mass, with retraction and disappearance of the cyst-wall. Very often a relapse of the inflammation terminates in death. Sometimes the membrane, being greatly distended and compressing the adjacent parts, ruptures and the pus escapes.

Etiology.

The cerebral parenchyma may become inflamed from external morbid influences, or from irritation within the cranial cavity. In the first category we frequently find: traumatic lesions and concussion of the skull (otitis has been observed in a quarter of the cases of cerebral abscess), suppurating erysipelas of the face and scalp, high temperature (insolation), etc. Cerebritis is sometimes primary, sometimes secondary. In the latter variety, the meninges are almost always involved in the diseased process; the vascular system plays an important role, either directly or indirectly. Among the internal causes of cerebral inflammation, we must here include fatty and calcareous degeneration of the vessels, epithelial changes in the arteries, obliteration by emboli, the transportation of septic and infectious particles (pyæmia, typhoid fever, puerperal diseases, glanders), and the toxic action of certain metals (mercury and lead). The cerebral inflammations due to hæmorrhages or tumors are also caused by disturbances of circulation and nutrition. According to Virchow, we must also classify the inflammatory processes, occurring in children who are born of syphilitic or variolous parents, among the internal causes leading to interstitial encephalitis and to fatty degeneration of the cells of the neuroglia. We may finally mention the interstitial connective-tissue changes found in insanity.

Encephalitis occurs at all ages. It may be found even in childhood; in youth and mature age, otitis and the infectious, contagious, and toxic conditions above mentioned are the most frequent sources of encephalitis and its sequences. In old age, it is more frequently due to diseases of the vascular system. Cerebral inflammation and abscess are more common in males, on account of the greater frequency in them of the etiological factors.

Symptomatology.

Cerebral inflammation presents various aspects as regards its symptoms and course. A certain classification has been adopted, corresponding to the most important forms of encephalitis.

These are the *meningeal form*, characterized by symptoms of febrile excitement followed by depression; the *comatose form*, characterized by the prompt appearance and persistence of drowsiness, immobility, and dilatation of the pupils, convulsions, early abolition of muscular activity, and a fatal termination; the *paralytic form*, which is manifested in circumscribed foyers, by the extension of paralysis to the limbs, or to one-half of the body, by disturbances of gait, speech and intelligence, without severe symptoms of irritation; the *apoplectic form*, which is usually fatal after a fulminant course, attended with loss of consciousness and sudden paralysis; finally, the *epileptic form*, which begins, especially in children and females, with convulsions or with eclamptic attacks, and terminates, after

temporary return of consciousness, in general paralysis. All these forms really represent the persistence and aggravation of different stages of encephalitis with predominance of certain symptoms of irritation and depression.

The study of the *special pathognomonic signs* embraces severe and profound functional disturbances of the nervous system.

Cephalalgia is a very frequent initial symptom, although not a constant one. It is sometimes especially violent in those forms which follow traumatic lesions or otitis, but it does not furnish any precise indications as to the seat of the foyer.

The *psychical disturbances* vary a great deal, according to the course and form of the disease. In the acute forms we especially observe an early change in the intelligence, some delirium and somnolence, and, at a later period, profound coma usually supervenes. Chronic encephalitis very rarely leaves the intellectual faculties intact. In most cases there is a progressive loss of intelligence, varying from mere loss of memory to complete imbecility. The disturbances of speech are sometimes due to paralysis of the tongue, and sometimes they are of an aphasic nature.

The disorders of motion are also extremely variable. In the beginning, certain symptoms of partial irritation usually predominate, such as clonic or tonic convulsions in the muscles of one-half the body, of one extremity or of one side of the face, convulsive strabismus, and much more rarely general convulsions. The frequency and intensity of the symptoms testify to the severity of the affection. The paralyses which occur are often intermingled with convulsive disorders. They generally appear early, progressing usually from the extremities to the trunk, may be either complete or incomplete, and usually assume the hemiplegic type. With a little care, we may distinguish contracture or paralysis of the facial muscles. In lesions of the facial nerve in the Fallopian aqueduct, as a consequence of otitis, these symptoms have a peripheral origin.

Sensibility is often increased from the beginning. Neuralgic pains in the limbs and cutaneous hyperæsthesia in some situations, are frequently observed. Sensations of cold, pruritus, formication, and heaviness of the limbs are very frequent. Later, we may observe more or less complete anæsthesia, usually combined with motor paralysis.

These various conditions are accompanied by a more or less intense fever. The acceleration of the pulse and respiration, and the elevation of temperature, are due to the intensity of the inflammatory process; but sometimes, when the cerebral compression increases, these functions are lowered beneath the normal. The nausea and violent vomiting which appear at the onset, disappear during the course of the disease. The tongue is coated and the appetite lost, as in other febrile affections. Deglutition may be impeded from the beginning by spasm of the pharyngeal muscles, and, later, it may be completely abolished by their paralysis. Very frequently obstinate constipation exists; involuntary evacuations are more rarely observed; retention and incontinence of urine may also occur. Relaxation of the sphincters, bed-sores, and asphyxia are the final signs of the general decay.

In *spontaneous encephalitis*, the signs of cerebral congestion (headache, vertigo, somnolence, delirium, slight subsultus, nausea, etc.) may continue for a long time before a noticeable embarrassment of speech, strabismus, spasms in the muscles of the face and limbs, convulsions, loss of consciousness, and persistent hemiplegic disorders show the existence of serious cerebral disease.

In *traumatic encephalitis*, the symptoms more often bear the stamp of meningitis. In many cases, symptoms indicative of a slight brain lesion give place, after a long period, to a sudden and fatal onset of cerebral disturbances.

Encephalitis following otitis is preceded by the symptoms of the local disease. Pain in the ear, purulent otorrhœa, inflammation of the external auditory canal, lesions of the tympanum, and loss of hearing, precede the symptoms of cerebral inflammation, which usually runs an insidious course and is often attended by disease of the meninges and vascular canals. Violent headache, vomiting, delirium, fever with chills, subsultus, spasms or paralyses of the muscles of the face and extremities, and hemiplegia, may be present for a long time; then follow coma and death. Abscess of the brain has been noted by Rud. Meyer (Zur Pathologie des Hirnabscesses, diss. inaug. Zurich, 1867) twenty-eight times in 86 cases of caries of the temporal bone. Toynbee has shown that lesion of the osseous structure of the external auditory canal gives rise to disease of the lateral sinus and cerebellum, lesions of the cavity of the tympanum to disease of the cerebrum, and those of the labyrinth to disease of the medulla oblongata; exceptions to this rule may, however, occur.

Cerebral abscess, following chronic inflammation of the mucous membranes of the auditory apparatus or nasal fossæ, and that following lesions of the skull, violent cerebral concussion or foci of suppuration in different organs, begins with intense headache, corresponding to the site of the affection, with high fever, severe and frequent convulsions and, usually, with loss of consciousness. Then follow drowsiness and the motor symptoms of a circumscribed lesion. Spontaneous cure of cerebral abscess has never been observed and it can only occur after a surgical operation, giving exit to the pus.

The following are the most recent observations with regard to the site of the encephalitis and the corresponding symptoms: *In encephalitis, or abscess of the frontal lobes* (following a trauma or ozæna), frontal headache is a frequent symptom. There are disturbances of consciousness and of the mental faculties, convulsions, pareses of the opposite side of the body, and sometimes aphasia. *In encephalitis of the walls of the Sylvian fissure*, or of their paths of communication with the frontal and parietal regions, aphasia is the most striking symptom. *Encephalitis of the motor ganglia* is manifested by disorders of intelligence and contractures, followed by paralysis of one half the face and body. *In encephalitis of the tubercula quadrigemina*, amaurosis occurs at an early period (ophthalmoscopic examination, according to Galezowski, is at first negative, but finally shows atrophy of the optic nerves). *Encephalitis of the cerebral peduncle* causes paralysis of the oculo-motor nerve on the same side, disorders of motion and sensation in the limbs on the opposite side of the body, and disturbances of vision from coexisting lesions in the roots of the optic nerves. *Encephalitis of the pons varolii* is very rarely of an inflammatory nature. The softening usually results from atheroma and obliteration of the basilar artery, and produces hemiplegia of motion and sensation, either of an apoplectic type or with a slow course, with dilatation of the pupils, and difficulty of speech or respiration. *Encephalitis of the cerebellum* is manifested by violent occipital headache, vomiting, uncertainty of movements, general convulsions, and stiffness of the muscles of the neck. These last symptoms, with absence of signs of a lesion "en foyer," indicate the presence of an abscess in the cerebellum.

Complete recovery is one of the rarest terminations of encephalitis.

The majority of cases of improvement only lead to a greater or less degree of restoration of the cerebral functions. In a fatal case, reported by J. Russel, and one of recovery reported by Scholz, the encephalitis terminated by the discharge of the pus through a perforation in the cranial vault.

The usual termination is in death. This is caused by extension of the cerebral lesions, by the compression and anæmia due to œdematous swelling, or by complication with meningitis, hæmorrhage, effusion of serum, opening of the abscess into the ventricles or cavity of the arachnoid, hypostasis, gangrenous bed-sores, etc. Death usually occurs from the first to the fourth week of the disease.

Diagnosis and Prognosis.

It is especially difficult to distinguish encephalitis from meningitis. High fever and violent, irritative symptoms at the onset, irritation of the sensorial functions, equal implication of both sides of the body (except the paralyses in the last stage of the disease) are indicative of meningitis. In encephalitis, on the other hand, we observe less fever, the early appearance of partial convulsions, usually limited to one side of the body, and contractures and paralyses. It is also very difficult to differentiate cerebral hæmorrhage from the apoplectic form of encephalitis. We may mention as diagnostic signs of some importance, that the symptoms of hæmorrhage attain their maximum severity at the beginning and then gradually improve, while the reverse holds true in encephalitis (Rostan). In the latter, also, the loss of consciousness is less complete, the unilateral convulsions and contractures are much more frequent and severe, and the paralyses are less marked and persistent. Furthermore, in cerebral inflammation, the pulse is usually very irregular and intermittent during the first few days, and cutaneous hyperæsthesia may be present—symptoms which are absent in apoplexy.

In common with tumors, cerebral abscess presents the signs of compression and of inflammation around the abscess, and the diagnosis may depend upon the etiology and progress of the disease. When, after injuries to the skull and violent cerebral concussion, disturbances of intelligence and motion develop within a few days and terminate, after a remission of greater or less duration, in convulsions and localized paralyses, we are justified in diagnosing an abscess of the brain. If any of the etiological factors of cerebral abscess are present, and symptoms of a local lesion in the brain (terminating in death at the end of three or four weeks) develop, we are also warranted in making a diagnosis of abscess.

The prognosis is extremely unfavorable. Even in the rare cases in which the patients recover, they are always threatened by the danger of a relapse. Several recoveries have occurred from traumatic encephalitis and, without doubt, because the brain had been previously healthy in these cases. Meningo-encephalitis following otitis usually terminates rapidly in death, and, even when the increase of the otorrhœa diminishes the violence of the cerebral symptoms, a cerebral lesion remains which may afterward give rise to a fatal exacerbation.

Treatment.

We may employ active antiphlogistic measures (cold applications, leeching, intestinal derivation) to combat the initial symptoms in trau-

matic encephalitis and in the meningeal or apoplectic forms. When symptoms of encephalitis develop after otitis, we should prescribe mild laxatives in addition to cataplasms and lukewarm, non-irritating injections into the ear, with small doses of the narcotics, if the patient suffers from severe pain and insomnia. The latter drugs are contraindicated when signs of cerebral compression are present. Leeches should not be employed when the patients are debilitated, tuberculous or scrofulous. In diathetic conditions, good results have sometimes been obtained from the use of iodide of iron. When symptoms of depression are imminent, we may employ cold affusions. Surgical interference may perhaps be resorted to, if the brain has been previously healthy and if the patient is young and robust. But, on account of the great difficulty of locating the exact site of the abscess, trephining may only be performed when certain severe symptoms are present, such as aphasia, or when there is irritation from compression of the skull. Renz obtained a recovery in a case of traumatic cerebral abscess (Tuebingen, 1867), by means of aspiration of the pus through a fine trocar.

CHAPTER VII.

CEREBRAL EMBOLISM AND THROMBOSIS.

Pathological Anatomy and Experimental Investigations.

In cerebral embolism, which is generally caused by a clot that has been detached from the heart or large vessels, anæmia is immediately produced within the territory of the branches which start from the obliterated trunk. This anæmia may be partly masked by the coexisting venous stasis. The increase of the current of blood in the other vessels soon gives rise, in favorable cases, to a sufficient collateral circulation; or the persistence of the ischæmia and the increase of the collateral blood-pressure cause compensatory hyperæmia of the neighboring parts, with serous transudations, more or less appreciable extravasations and capillary hæmorrhages.

Necrobiosis of the cerebral tissue usually develops in the centre of the ischæmic region. The parenchyma is softened, and after a few days, contains large masses of granular corpuscles and cells. The nerve fibres and cells, which have undergone retrogressive changes, present a red, yellow, or white color, according to the proportions of the coloring-matter of the blood and of the fatty corpuscles present. The embolismic focus may also give rise to peripheral inflammatory changes which, like those of primary encephalitis, may terminate in softening, cyst formation, abscess, or sclerosis. When these secondary inflammatory conditions have attained a certain intensity and duration, the obliterated vessels may also become involved in the inflammation or, on the other hand, they may again become permeable.

In the very frequently occurring embolism of the middle cerebral artery, which principally supplies the central ganglia and the surrounding medullary substance, the foci of softening not only involve the motor centres, but also the medullary substance situated behind the lenticular ganglion, and which serves to transmit sensory impressions. If the secondary disturbances of circulation are not of a serious nature, they will be followed, in this region, by less severe disturbances and by a more ready recovery.

In thrombosis of the carotids or cerebral arteries, due to inflammatory or specific changes in the walls of the vessels, to cachectic or diathetic diseases or to local compression, a thrombus, which completely obstructs the lumen of the vessel and the approach to its branches, may cause the development of foci of softening.

Experimental investigations by numerous observers have shown that cerebral embolism in animals gives rise to a rapidly fatal apoplectiform attack, characterized by convulsions and tetanic spasms, followed by relaxation, by difficulty and slowness of respiration, spasm of the respiratory muscles, and insensibility of the conjunctiva, with preservation of corneal sensibility.

Etiology.

Cerebral embolism is most frequently due to diseases of the endocardium and heart-valves. Inflammatory or atheromatous processes of the endocardium may cause a deposit of fibrin, portions of which, as well as fragments of the softened valves or calcareous concretions, may become detached and be carried into the cerebral arteries. With the exception of the kidneys and spleen, the brain is the most frequent site of embolism.

In addition to endocarditis, the other causes of cerebral embolism are: fatty granules from depots of suppuration, obstruction of very small cerebral arteries by the accumulation of white blood-globules in pyæmia (Rokitansky) and in leukæmia (Ranvier, Thudichum), or by pigment in intermittent fever. Finally, embolism may be caused by the introduction of foreign bodies into the vascular system, such as particles of cancerous, tuberculous, puriform or gangrenous matter, or such as are derived from perforating myocarditis, the vesicles of echinococci, or syphilitic tumors breaking into the cavities of the heart.

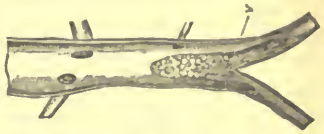

FIG. 4.—The left middle cerebral artery, containing an embolus in the main trunk, upon the distal extremity of which two smaller thrombi have formed in the terminal branches.

Arterial thrombosis, which predisposes to the formation of emboli, may be caused by senile atrophy of the arteries. Disturbances of nutrition in the walls of the vessels, fatty degeneration of the cerebral arteries in pyæmia, puerperal diseases, scurvy and chronic alcoholism, and syphilitic exudations into the walls of the carotid, of the arteries at the base, and of the cerebral arterioles, may also give rise to the formation of thrombi and the detachment of fatty particles or corpuscles, constituting emboli. Venous thrombosis is sometimes observed in inflammation and suppuration of the internal wall of the veins in consequence of phlebitis and pyæmia, or when inflammation and exudation are propagated from the tunica adventitia to the tunica interna or, finally, in some diathetic conditions or traumatisms.

Certain vessels are particularly predisposed, by their anatomical situation, to receive obstructing clots. Among the large vessels of the head, the left carotid is very much exposed to embolism, by reason of its long, straight course and the oblique direction to the left and backward which it assumes on leaving the arch of the aorta. Among the cerebral arteries, the middle cerebral is most liable to embolism (45.7 to 100), then the internal carotid, then the basilar and vertebral arteries, and, very rarely, the artery of the corpus callosum.

It appears, from recent observations, that the anatomical relations of the affected artery to the neighboring vessels exert a considerable influence upon the accidents of embolism. According to Cohnheim, if an arterial anastomosis exists it will carry blood into the capillaries of the obliterated artery. But if the embolism affects a terminal artery, deprived of all anastomosis, the blood will remain stagnant in the course of the artery, and in its capillaries and veins, until such time as the blood, starting from the veins in which the circulation has remained unobstructed, returns into those which have been shut off, and passes backward into the capillaries, and then into that portion of the artery situated beyond the embolus.

After prolonged action of the stasis upon the vascular walls, the latter

will permit the solid constituents of the blood to pass through them. Thus, embolism of a terminal artery will not only lead to necrosis, but also to a rapid and abundant extravasation of blood-globules outside of the vessels, and to hæmorrhagic infarctions.

Heubner has ascertained the territories over whose nutrition each of the cerebral arteries presides. He has found that the Circle of Willis, and the principal vessels of the brain, send terminal branches (without anastomosis) to perfectly distinct regions of the central ganglia and adjacent portions of the mesocephalon. The cortex, however, is supplied by vessels which anastomose very freely in the pia mater, from the meshes of which vessels pass off at right angles towards the cortex and subjacent medullary tissue. This explains the infrequency of embolism and softening in the cortical substance. As accidental causes of the disintegration of thrombi and of sudden obliteration of the vessels, we may mention mental excitement, and especially the physical strain caused in many of these patients by change of posture, by defecation, and by coughing, etc. The male sex manifest a greater predisposition than the female, owing to the greater frequency of endocarditis, of diseases of the heart and vessels in the former, as well as to the greater hardships endured by them. Cerebral embolism is rarely observed before the age of twenty years. It is most frequent from twenty to forty years, and from this period until the age of sixty it continually diminishes in frequency.

Symptomatology.

The symptoms usually begin suddenly, the premonitory symptoms (headache, vertigo, dyspnœa) belonging to the primary disease. The patient suddenly falls down, often in a convulsion. The face is pale and cold, consciousness is partially or completely lost, but returns after a little while or at the end of a few hours. After the return of consciousness, the disorders of motion, sensation, special senses and speech, appear more clearly. There is usually incomplete hemiplegia on the side opposite to the embolism, with paresis of the respiratory muscles of the face, and greater or less disturbances of sensation. The movements of the tongue remain intact.

Among the disorders of the special senses, hemiopia and unilateral amaurosis with alternate hemiplegia are characteristic signs. They are due to extravasations into the optic nerve, and to embolism of the central artery of the retina on the corresponding side. The ophthalmoscope shows pallor of the papilla and absence of pulsation in the retinal arteries. The latter are narrow, the veins are larger at the periphery, and the macula is surrounded by small hæmorrhages. Embolic amaurosis may precede the cerebral embolism for several days.

Aphasia is another characteristic symptom of cerebral embolism. This is a peculiar affection of the faculty of speech, usually occurring with preservation of intelligence and of the movements of the tongue, and which has given rise to much discussion during the last few years.

Broca formulated the doctrine that aphasia is due to a lesion of the left frontal lobe and of the posterior part of the third frontal convolution.

Within recent times, considerable change has occurred in the clinical and anatomical study of aphasia. From a clinical point of view, we have a better appreciation of the various forms of aphasia, its connection with hemiplegia of motion and sensation, and its appearance in consequence of

embolism, softening, or other lesions of the brain, in typhoid fever, the acute exanthemata, erysipelas, Bright's disease, diabetes, hysteria, catalepsy, epilepsy, etc. Not infrequently the affection appears temporarily in the course of cerebral diseases and passes unnoticed. At other times aphasia is an intermittent symptom, but it is more often persistent.

According to the varying intensity of the aphasia, the patients lose the faculty of calling surrounding objects by name, of telling their own name or those of their acquaintances. They change words, invert syllables, or answer all questions by certain stereotyped phrases or fragments of words, while some of them mechanically repeat verses or prayers learned at a previous period. In other cases, the aphasics invert syllables in reading as well as in writing from dictation, although they are able to transcribe properly after having well considered the words which are placed before them. Sometimes the adaptation of the words or characters to ideas is lost, and sometimes the adaptation of ideas to the words. In severe forms the disturbances of the understanding, as well as the discord between ideas and objective sensations, testify to disorder of the intel lectual faculties.

Lesions of the frontal convolutions in aphasia have been demonstrated for some years by a large number of autopsies. The anatomical value of these data is not diminished by the fact that language may be preserved after well-defined traumatic or pathological lesions of the anterior lobes, or by Cruveilhier's case of an idiot, aged 12 years, and speaking distinctly, with congenital atrophy of the anterior lobes. In the first case, complete destruction of all the active parts of the anterior lobes has never been determined by the microscope. In Cruveilhier's case, the posterior and transverse halves of the third left frontal convolution and a considerable portion of the right anterior lobe were preserved.

In addition to the lesions of the frontal, and adjacent portions of the parietal lobe, a large number of cases have been observed in which the island of Reil and its neighborhood (either alone or together with the anterior lobe) were the seat of a hæmorrhage or encephalitis with aphasia. Of fifty cases of aphasia collected by Lohmeyer (Langenb. Arch., t. XIII., 1872), the third frontal convolution was involved thirty-four times, and in the sixteen remaining cases, the island of Reil and the middle or posterior lobes were alone affected. The majority were due to embolism of the left middle cerebral artery.

A critical examination of published cases proves that the lesions found in the brain agree perfectly with the topographical notions recently acquired concerning the distribution of the cerebral vessels. As Heubner has shown, the middle cerebral, in the first few centimètres of its course, nourishes, in great part, the central ganglia and adjacent medullary substance. The same vessel supplies the island of Reil, which it traverses, and then divides into several branches, the first two supplying the second and third frontal convolutions, and the third and fourth branches supplying the central convolutions, the parietal gyri which are directed towards the convexity of the brain, and the adjacent temporal convolutions. When all the branches of the artery are plugged up by emboli, hemiplegia of motion and sensation will result with marked aphasia. In slighter forms the centres of motion and sensation remain intact.

The united results of anatomical and clinical investigations prove that in man the function of language has its central seat in the region comprising the island of Reil, and its connections with the frontal lobes and with the central and parietal convolutions. In various forms of aphasia

the lesion may be situated in the deeper or more superficial portions of the region above mentioned.

Popham and Ogle have differentiated amnesic aphasia, in which there is loss of memory of words as symbols of ideas, from ataxic aphasia, in which loss of co-ordination occurs in the muscles presiding over the articulation of speech. But this division is too artificial for all cases. It is more reasonable to suppose that a function so complex as language arises from the combined action of important parts of the brain. The abolition or weakening of their functional manifestations will operate upon the formation of language in the brain, in the same way that interruption of motory or sensory stimulation in more or less wounded portions of the gray substance will act in the cord. The predominance of left-sided lesions in aphasia is due, according to Leyden, to the more ready flow of blood in the left carotid and, according to Gratiolet, to the earlier embryonic formation of the left hemisphere (denied by Carl Vogt), and of the muscles on the right side of the body.

Aphasia from lesion of the right hemisphere has been observed, according to Ogle, in left-handed individuals. According to Broadbent and Ogle, the cerebral convolutions, in the brains of left-handed individuals, have been found more developed on the right side, while, as a general rule, the convolutions of the left side are larger. The facts hitherto observed indicate that the left hemisphere plays the most important, but not exclusive, rôle as the central organ in the formation of language.

Among the *complications* of cerebral embolism we may mention bilateral embolism and the coexistence of cerebral embolism with that of other arteries. Bilateral embolism is rarely produced at the first onset, but almost always in different attacks. These cases are characterized by the following symptoms: epileptiform attack, temporary loss of speech, paralysis of one half, rarely of both halves of the body, hurried and convulsive respiration, unilateral anæsthesia of the conjunctiva with normal sensibility of the cornea. Erlenmeyer's case of double cerebral embolism presented all the appearances of general progressive paralysis. A complication with embolism in other arteries manifests itself by the symptoms peculiar to the latter. Crural embolism is followed by absence of pulsation and sudden paralysis in the lower limb (especially the extensors of the thigh, with loss of electro-muscular contractility) and by the gangrene which usually follows arrest in the current of blood. Embolism of the splenic artery is followed by sudden and painful swelling of the spleen; embolism of the renal artery, by hematuria and albuminuria; embolism of the mesenteric artery, by sudden intestinal hæmorrhage and abdominal pain. Embolism of the capillaries of the skin, under the form of isolated or grouped spots, which partially disappear under pressure, may also occur in the course of cerebral embolism.

The *termination* of cerebral embolism depends upon the intensity and seat of the lesions. When the extravasation is promptly absorbed, and the arterial plug disappears by fragmentation, before the brain has suffered any serious disturbance, when only the large arteries have been obstructed, and the circle of Willis has quickly restored the collateral circulation, and when the anæmia of the affected portions has not extended too far, the disorders dependent upon the cerebral lesion and the disturbances of speech may retrograde. In embolism of the internal carotid (manifested by loss of consciousness, hemiplegia of the opposite side, often by severe convulsive attacks, by symptoms of anæmia, and of collapse, with slow pulse and respiration), the signs of passive hyperæmia of the brain do not appear, if

the re-establishment of the collateral circulation is not delayed beyond 24–48 hours.

The progress of the disease is less favorable when the embolus is situated beyond the circle of Willis, since the collateral circulation is not established so readily and foci of softening almost always occur. In the most favorable cases, a cyst or cicatrix forms, attended by persistent paralysis of motion and sensation, and by disorders of speech and of the mental faculties.

Diagnosis and Prognosis.

The diagnosis of cerebral embolism sometimes presents great difficulties, especially in its differentiation from hæmorrhage. Only a small number of the symptoms have a diagnostic value. The premonitory appearance of hemiopia or a sudden subsequent embolic amaurosis, with alternate hemiplegia; the persistence of aphasia after an apoplectiform or convulsive seizure, and the combination of aphasia with hemiplegia of motion and sensation (usually on the right side); the paralysis of both halves of the body after several attacks of apoplexy, and the appearance of aphasia with right hemiplegia,—all these symptoms belong to cerebral embolism and not to ordinary hæmorrhage. The existence of cerebral embolism is extremely probable, when the apoplectiform attacks, in the absence of prodromata and of the symptoms peculiar to cerebral hyperæmia, appear in young or adult subjects who have suffered from previous endocarditis.

Embolic processes in other organs contribute but little towards clearing up the diagnosis. Embolism of the splenic or renal arteries may exist without characteristic physical signs. The obstruction of the arteries of the extremities does not always facilitate the diagnosis, since the apoplectiform symptoms may appear much later.

Pigmentary embolism of the cerebral capillaries (apoplectic intermittent fever) is recognized by the appearance of coma and convulsions, with enlargement of the spleen, and a history of previous attacks of pernicious intermittent fever.

The *prognosis* of cerebral embolism is grave. In embolism of the large arteries the collateral circulation may, indeed, be quickly re-established, the embolus be absorbed, the vessel become permeable, and even the paralysis may recover. But the primary disease still exists and exposes the patient to the danger of new attacks. Often, also, disorders of motion and of the mental faculties persist in consequence of the changes in the cerebral parenchyma.

Treatment.

In slight cases the patients require complete rest, cold compresses to the head and cooling drinks. If the symptoms of irritation or venous stasis develop, we may apply ice to the head and employ digitalis and intestinal derivatives internally. In young and vigorous subjects we may even resort to leeching. When collapse threatens, stimulants should be employed for a short time. During the progress of recovery we may hasten the cure of the aphasia by teaching the patient short words in common use, and by employing writing exercises. The faradic current may be applied to the paralyzed limbs. In aphasia due to traumatic lesions of the skull, this symptom sometimes yields to trephining.

CHAPTER VIII.

CEREBRAL ATROPHY.

Pathological Anatomy.

Cerebral atrophy varies according to the age of the patients, the development of the disease, and the morbid conditions which have given rise to it. *Infantile cerebral atrophy* may date from intra-uterine life, or constitute an atrophy of development (cerebral agenesia of Cazauvielh), dating back to the period at which the development of the brain and remainder of the economy occurred. The characteristic lesions are obliquity of the skull (one side of which is shrunken, deformed and thickened from premature closure of the sutures) and atrophy of the corresponding cerebral hemisphere, with diminution in the size of the convolutions. The atrophy usually extends to the central ganglia and, to a greater or less extent, to the peduncles, pyramids and antero-lateral columns of the cord. The atrophied portions are usually hardened, rarely softened; the cortical and medullary substances have lost their color and are studded with small collections of serum.

In *senile cerebral atrophy*, diminution of the volume of the brain occurs physiologically, in consequence of a partial disappearance of the interstitial connective tissue, with a more or less marked diminution of the proper elements of the parenchyma, abundant pigmentation of the cortical cells, fatty and pigmentary degeneration of the walls of the vessels, and the appearance of amyloid corpuscles.

The cerebral atrophy which occurs at different periods of life, in the course of certain primary affections of a destructive nature, is sometimes partial and limited to one side, and sometimes general. Partial cerebral atrophy affects a portion of the medullary substance of the brain, with cicatricial retraction of the parenchyma, extending further towards the periphery, or the atrophy may follow the anatomical course of the fibres through the peduncles, pons varolii, and pyramids to the opposite side of the spinal cord; or a crossed lesion is found in the superior portions, such as atrophy of one cerebral and of the opposite cerebellar hemisphere.

In general atrophy, the medullary substance of the hemispheres is chiefly involved. The frontal convolutions are unequally thinned, the sulci larger and deeper, the medullary tissue is grayish-yellow and hard, the cortex is thin, and of a pale gray color, and the cortical cells are swollen and in a condition of amyloid degeneration. The consistence of the brain is increased, especially near the ventricles, which are dilated with fluid. The ependyma is granular and covered with warty growths of connective tissue. In the more advanced forms, the cerebral tissue, in consequence of the proliferation of the interstitial connective tissue, may acquire a leathery hardness, and the cut sections present a corrugated appearance. The vessels are dilated from hyperæmia and retraction of the surrounding tissue, and the medullary tissue and corpora striata are

strewn with small holes (état criblé of Parchappe and Durand Fardel). The loss of weight in the brain in general paralysis of the insane is greater than in any other cerebral affection. The atrophy particularly involves the frontal lobes; the cerebellum remains intact, although epilepsy is accompanied by great diminution of cerebellar weight.

Etiology.

Among the causes of atrophy of the cerebral substance, we must mention local affections of a destructive nature as of the first importance. A large number of cases of so-called agenesia are due to intra-uterine affections, such as hæmorrhage, encephalitis, and external and internal hydrocephalus. Physical shocks or violent emotions during pregnancy are, in some cases, the primary causes of the fœtal diseases. In children several years old, a fall may cause compression of one side of the skull, followed by convulsions and atrophy of one hemisphere. Cerebral atrophy in weak-minded old people may also be due to the destructive lesions which usually follow apoplexy, encephalitis, &c.

The cerebral atrophies, which occur at various periods of life, are very often due to inflammations of the pia mater (by involving the vessels which supply the cortex), to hæmorrhage, encephalitis, disturbances of nutrition following thrombosis and embolism, chronic hydrocephalus, and large tumors. The *cerebral atrophy*, which sometimes occurs *under the influence of causes modifying the general nutrition* (alcohol, opium, lead, and syphilis), is due to lesions of the small cerebral arteries of which we have frequently spoken, and to the consequent disorders of nutrition. *Lesions of the peripheral nerves* often give rise to a consecutive atrophy of the corresponding central parts. Diseases of the retina and optic nerve may be followed by atrophy of the tubercula quadrigemina and, in diseases of the spinal cord, degeneration of nerve fibres may result even in the brain.

Symptomatology.

In *congenital atrophy*, or in that which is produced during the first years of life, the premature arrest of development of one half of the brain is generally accompanied by considerable disturbance of the intellectual development. In marked cerebral atrophy, weakness of intellect, abolition of the functions of the organs of special sense, deaf-mutism, and imperfect sensibility in the paralyzed limbs, may be present. When the disease begins in childhood, the psychical changes are usually less marked. The varieties of crossed and unilateral atrophy, described by Virchow, are characterized by paralysis and atrophy of the opposite half of the body in addition to the obliquity and asymmetry of the skull. The limbs are more or less paralyzed (almost never completely), and deformed by contractures. In two cases which I observed the electro-muscular contractility was not affected. In many cases strabismus and ptosis occur. Speech is usually only slightly changed. Frequently, the patients are neither able to walk nor to eat alone, and remain infirm and crippled during their whole lives. The atrophy often begins with epileptiform convulsions or choreic trembling, which soon disappear or may recur from time to time until death. Atrophy of one half of the pelvis, and of the bones, muscles, nerves, and spinal cord, upon the same side as

the atrophy of the cerebellum, and on the side opposite to the cerebral hemisphere involved, is often observed in these cases.

In *senile atrophy*, progressive deterioration of the mental faculties occurs with occasional stages of excitement. The old people lose memory and intelligence, become absent-minded, and relapse into childishness with apathy and somnolence. The senses become blunted, power of motion is gradually lost, the uncertainty of the movements and the tremor continually increase, and the patients finally take to bed, and succumb to paralysis of the centres in the medulla (irregularity of pulse and respiration, paralysis of deglutition), or to hypostasis, bed-sores, bronchitis, or acute pulmonary œdema.

In *partial atrophy in the first years of life*, the symptoms differ according to the site and origin of the primary disease. In young people, the motor paralyses often disappear completely while the mental disorders gradually increase. *General cerebral atrophy*, after meningitis or disturbances of the general nutrition, is characterized by the progressive extension of the paralysis to both sides of the body, and by the gradual loss of the mental faculties until a stage of imbecility is reached.

Diagnosis and Prognosis.

The intra-uterine varieties are recognized immediately after birth by the existence of paralyses and spasmodic seizures. In the forms which arise during early childhood, the deformity of the skull, the crossed hemiplegia with atrophy and contracture of the limbs, the convulsive and epileptiform seizures, and the mental disturbances, render the diagnosis easy. In the partial cerebral atrophies which occur at different periods of life, the previous existence of local affections of a destructive nature, of chronic changes from toxic influences, or of partial or hemiplegic disturbances of motion and sensation, and coexisting mental weakness, are sufficient to enable us to make a diagnosis.

The *prognosis* of cerebral atrophy depends upon the period at which it develops, and upon its nature and causes. The atrophic degeneration of the brain which begins during intra-uterine life or shortly after birth, has a much more serious effect upon the physical and especially upon the mental development than atrophy occurring in a brain whose organization is more advanced and capable of resistance. In the latter case, improvement of the mental faculties has often been observed on account of the compensatory functional activity of the corresponding portions of the intact cerebral hemisphere.

The prognosis of senile atrophy, as regards recovery or even improvement, is absolutely unfavorable. When partial cerebral atrophy occurs in young subjects, the local lesion may disappear in many cases, especially in those of traumatic origin, and the patients, thanks to their youth and previous good health, may recover the power of motion and the nutritive functions, but the mental faculties are usually not restored in a corresponding manner.

If an increase of mental weakness occurs under the observation of the physician, it is justifiable to conclude that secondary atrophy is developing.

Treatment.

The treatment of cerebral atrophy merely consists of palliative measures. In infantile cerebral atrophy, when the symptoms of irritation have

subsided, the application of electricity to the paralyzed and contractured limbs may produce some improvement.

Orthopædic and gymnastic measures and careful and persevering instruction for the purpose of developing the intellectual faculties, are also useful. In senile atrophy improvement is out of the question. When the atrophy occurs at a less advanced age, in consequence of toxæmia or diathetic disturbances of nutrition, as in chronic alcoholism, lead poisoning and cerebral syphilis, improvement may be obtained by the appropriate treatment of these conditions. In partial atrophy, following destructive local lesions, we may prescribe general tonic and sedative remedies and may also employ mild antiphlogistic medication when signs of irritation occur.

CHAPTER IX.

CEREBRAL HYPERTROPHY.

Pathological Anatomy.

When the brain is hypertrophied we will find, after removal of the skull and incision of the meninges, that the organ projects outside of the skull. On horizontal section of the hemispheres we observe an unusual development of medullary tissue. The centrum semi-ovale and Ammon's horn are unusually prominent, and the medullary substance is of a white color, almost like that of bone, and of much greater consistence than normally.

The hypertrophy affects either an entire hemisphere or only certain portions of the brain, as the optic thalamus, pons varolii, and medulla oblongata. In young children the skull is increased in size as in hydrocephalus. When the sutures are united, cerebral hypertrophy may lead to absorption of the internal table of the convexity and base of the skull, or to the formation of lacunæ in the walls of the frontal and sphenoidal sinuses, and in rare cases the sutures become separated.

New formation of the gray substance of the brain (heterotopia of the gray substance, Virchow) sometimes occurs. Usually we find diffuse interstitial hyperplasia, morbid production of cells and intercellular substance, and proliferation of the neuroglia, as the cause of the increase in the cerebral volume and weight.

Etiology.

This is especially a disease of childhood, but may sometimes be congenital. It usually appears towards the sixteenth month. Sometimes several members of a family suffer from this affection. When it develops in the later periods of childhood, swelling of the lymphatic glands and thymus and signs of rachitis in the bones are found to coexist.

At a more advanced age the causes of cerebral hypertrophy are alcoholism, lead poisoning, epilepsy, and insanity. It also occurs secondarily in cerebral tumors.

Symptomatology.

When the disproportion between the capacity of the skull and the volume of the brain becomes slowly established, several years may elapse before the pathognomonic signs of cerebral hypertrophy make their appearance. When the neuroglia proliferates rapidly and the development of the brain is retarded with predominant lesion of certain centres, the intra-cerebral pressure will be increased and manifest itself by symptoms of irritation, followed by symptoms of depression. Weakness, tremor, and

uncertainty of gait are produced. The children carry the head erect with difficulty, and often stumble and fall. Convulsions limited to the muscles of the eye or upper extremities occur, and very frequently terminate in paralyses with strabismus.

To the convulsive phenomena belong the periodical laryngeal spasms described by Muenchmeyer, under the name of thymic asthma. General convulsions are much more frequent and characteristic, but the attacks are usually short and may present the symptoms of eclampsia or of tetanic stiffness. These convulsions are not due to the cerebral hypertrophy but rather to the periodical increase of cerebral anæmia.

There are few disorders of sensation in this affection. The patients are usually very timid and frightened at the least noise.

The diminution of sensibility and of the functions of the special senses, which is observed in many cases, is undoubtedly the consequence of previous attacks. Disorders of intelligence occur generally in the more advanced stages of cerebral hypertrophy. During the first stages of the disease, there is even precocious intelligence with temporary symptoms of excitement. At a later period the mental faculties may become weaker and weaker, and the patient finally becomes imbecile.

Other frequent symptoms of cerebral hypertrophy consist of rachitic lesions of the bones, in the lower limbs as well as in the skull; difficulty of keeping the head erect, with a continual tendency to fall forward; and sucking the tongue, which usually protrudes from the mouth, and, in some cases, is very much increased in size. The increase of the intracerebral pressure gives rise to vomiting, disturbances of the intelligence, retardation, followed by acceleration of the pulse and respiration, contraction or dilatation of the pupils, and coma. Death usually occurs with symptoms of cerebral compression, or in an attack of convulsions, more rarely in consequence of complications.

Diagnosis and Prognosis.

In a great many cases the symptoms are too obscure to permit an exact diagnosis. Many authors have recognized, as a differential sign, a characteristic shape of the skull (angular with prominent frontal, parietal, and occipital protuberances). But this sign is only of value when other characteristic symptoms of the disease are present. In cerebral hypertrophy, the increase in the size of the head occurs very slowly, while in chronic hydrocephalus the skull may assume considerable dimensions within a few months. In the former disease the fontanelles are considerably enlarged and pulsate strongly, while in the latter they are very large and prominent but pulsate very slightly or not at all. The cerebral souffle is heard in hypertrophy, but not in chronic hydrocephalus. When the fontanelles have closed at an early period and convulsive attacks supervene (at first partial and of short duration, and afterward assuming an epileptiform character), we are justified in making a diagnosis of cerebral hypertrophy and in excluding chronic hydrocephalus.

The prognosis is so much the more grave, the earlier the appearance of the cerebral symptoms in children, and the more frequent and violent the convulsive seizures and laryngeal spasms. Death may occur suddenly in an epileptiform attack, or in a laryngeal spasm from asphyxia, or the disease may not prove fatal until a more advanced age.

Slight hypertrophy, limited to certain portions of the brain, may terminate favorably by an arrest of the process.

Treatment.

Treatment can only be directed towards regulating the habits of life and preventing the dangers arising from intercurrent complications. The patients should receive hearty and easily digested nourishment (eggs, milk, scraped meat). We may recommend country air, cool baths; the head should be elevated and screened from the heat. During epileptiform convulsions, apply cold compresses to the head, remove the mucus from the mouth, and facilitate respiration by drawing the tongue forwards.

CHAPTER X.

SCLEROSIS OF THE BRAIN AND CORD.

Pathological Anatomy.

Sclerosis affects various portions of the nerve centres. The brain and cord are rarely affected separately, but, usually, both are involved at the same time. In the brain the lesion affects, by preference, the medullary tissue, the cortical substance being involved much more infrequently. Upon a cut section of the brain, the foyers of sclerosis are seen to vary in size from a grain of hemp-seed to a hazel-nut, and form spots of a pale-gray color, transparent, hard, isolated or confluent, sharply defined and rounded, or having serrated borders. Some of these spots have a reddish gray color and a softer consistence. *In the brain*, these foyers are scattered throughout the white substance of the hemispheres, in the walls of the ventricles, corpus callosum, centrum ovale, septum lucidum, Ammon's horn, optic thalamus, corpus striatum, lenticular nucleus, peduncles, pons varolii, and in the medullary substance and rhomboidal body of the cerebellum. The process is sometimes accompanied by unilateral atrophy of different portions of the brain. In very rare cases, heterotopia of the gray substance is found in addition to the sclerosis of the medullary substance. In the *medulla oblongata*, the sclerosis affects the olivary bodies, the pyramids, the different columns, and the floor of the fourth ventricle, with the nuclei of origin of the cranial nerves. In the *spinal cord*, the foyers involve all parts indifferently; they sometimes form small nodosities in certain isolated spots.

Patches of sclerosis have been observed also upon the anterior and posterior roots of the spinal nerves. Among the cranial nerves, the foyers have been seen most frequently upon the optic, olfactory, and trigeminal nerves. They are much more rarely observed upon the motor nerves of the eye, upon the facial, and the roots of the hypoglossal, pneumogastric, and glosso-pharyngeal nerves.

The microscopical examinations of Fromman, Rindfleisch, Vulpian, and especially of Charcot, have given the following results: a nuclear proliferation occurs in the peripheral zone of the sclerosed patches, within the trabeculæ of the reticulum which is, at first, very much thickened, then becomes more and more indistinct and is finally replaced by connective-tissue fibres. We can also detect some small cellular elements, with atrophy and partial disappearance of the nerve fibres and preservation of the axis cylinders, which are sometimes very much hypertrophied.

In the central portions of the patches the reticulum is entirely replaced by new-formed fibres, containing many amyloid corpuscles. The trabeculæ, cellular elements, and nerve fibres have disappeared with the exception of a certain number of atrophied axis cylinders. There is considerable thickening of the walls of the vessels, with proliferation of the nuclei.

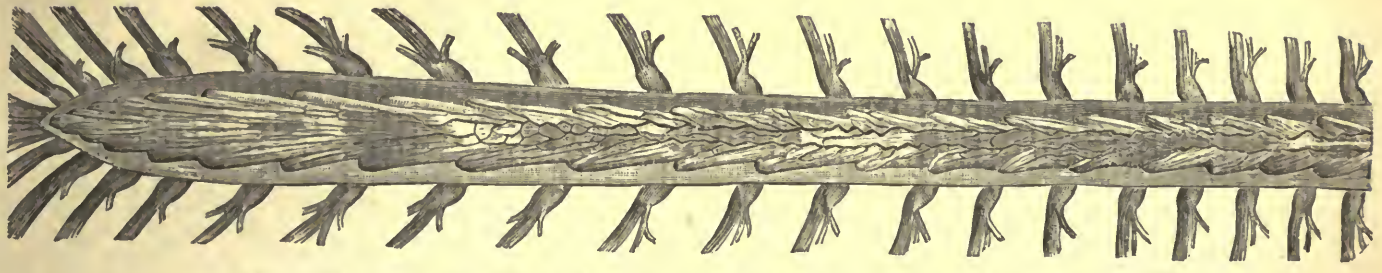

FIG. 5.—The spinal cord seen upon its posterior surface (the dura mater has been slit open and thrown aside). The light patches represent the normal tissue of the cord; the shaded spots represent irregularly disseminated patches of sclerosis.

FIG. 6.—Horizontal sections of the spinal cord, made at various positions from the cervical enlargement to the filum terminale. This cut shows the unequal irregular distribution of the sclerosed (darkly shaded) patches, both in the columns of the white matter, in which they predominate, and in the gray substance.

The spinal cord is strewn with tracts of connective tissue, which contain, in addition to cellular elements, a considerable number of amyloid corpuscles. The nerve cells are in a condition of yellow degeneration (Charcot) and atrophy, in the anterior horns (Schuele), posterior horns (personal observation), and in the nuclei of the nerves of the medulla (Joffroy).

Etiology.

This affection occurs most often between the ages of twenty and thirty years, and is much more infrequent at a more advanced age. Sex appears to have no special influence upon the development of the disease. Extreme cold, prolonged mental distress, and intellectual over-exertion have been mentioned as causes; Guérard has seen the disease develop during pregnancy.

The vascular system plays an important part in the genesis of the lesions of sclerosis, as has been previously pointed out by Rindfleisch (Virch. Arch., Bd. XVI., 1863). The researches and observations made since that time have given us a more accurate knowledge of this subject. The dilatation and thickening of the vessels observed in the patches of sclerosis, and even in the adjacent healthy portions of the parenchyma; the predilection of the sclerosis for such portions of the brain, as the medullary substance and ganglia, to which the vessels of the base furnish terminal arteries, and the rarity of sclerosis in the cortex, which is provided with anastomotic meshes by means of which the equilibrium in the circulation is readily re-established; the predominance of the proliferation of the neuroglia in the neighborhood of the vessels; and finally, the clinical fact that sclerosis of the nervous centres may be due to conditions of excitation in the vascular system from emotional disturbances, physical and intellectual efforts and cold,—all these circumstances tend to prove that the vascular system primarily plays an active part in the course of sclerosis, and that the nuclear proliferation, hyperplasia of the network of the neuroglia and atrophy of the nerve elements are secondary phenomena.

Congestion of certain regions of a protracted course may also give rise, in virtue of a peculiar predisposition, to the formation of patches of disseminated hyperplasia in the nerve centres, and since the time of Andral and Rokitansky, this hyperplasia has been considered a cause of cerebral hypertrophy.

Symptomatology.

In multiple sclerosis of the nerve centres, the first manifestations of the lesion may appear either on the part of the brain or on that of the spinal cord. In the cephalic forms (Charcot) the disease begins with nausea, headache, vertigo, syncopal and apoplectiform attacks followed by diplopia, amblyopia, nystagmus, and disturbances of the intelligence and of speech.

In the spinal forms, the patients, in the beginning, are easily tired and suffer from periodical pains and a sensation of cold; the paresis of the thighs causes a slow and trembling gait; inequality of the pupils often occurs. More frequently the symptoms have a mixed character (cerebrospinal), in which either the cerebral or spinal disturbances may predominate.

In order to give a better idea of the characteristic symptoms of this

disease, I shall give the following notes of a hitherto unpublished case which came under my observation, and which is instructive in several respects.

A peasant woman, aged 23 years, was taken sick about three months previous to her admission into the hospital, in consequence of overwork and exposure to cold, and had then suffered from headache, vertigo, weakness and frequent cramps in the lower limbs. Examination of the patient revealed nothing abnormal in the abdominal or thoracic viscera. When she attempted to walk or stand, she suffered from tremor of the thighs and excessive fatigue after the lapse of a few minutes. When the patient was raised, even after a prolonged rest in bed, she had a peculiar rocking movement of the head and tremor in the limbs, which could even be produced in the horizontal position when the patient was suddenly addressed in a loud tone of voice, or when she was in a condition of excitement. The gaze was fixed, the pupils markedly dilated, and speech slow but intelligible.

In a very short time she began to suffer from attacks (lasting two or three minutes) which were characterized by convulsive closure of the eyelids, contraction of the mouth, tetanic extension of the inferior limbs, and contracture of the upper limbs as far as the wrist and elbow; consciousness was intact. The sensorium became temporarily affected at a later period, on account of the repetition and prolongation of the attacks. In the following months, speech became more and more embarrassed and unintelligible. For a few moments the patient could not speak at all, or only in a weak tone of voice and in monosyllables. I was unable to note any disorder of the memory or intelligence. The prolonged use of bromide of potassium, nitrate of silver and electricity had no effect upon the course of the disease.

At the onset, reflex excitability was increased and sensibility diminished in the inferior half of the body, and towards the end of the first year of the disease the sensibility of these parts had become much more obtuse. The galvanic excitability of the nerve trunks of the lower limbs and the faradic excitability of the extensors of the thigh were considerably diminished. Three months later, a febrile attack (pulse 100–104, temperature 38.5–39.8 C.) occurred, which persisted for several days, at the end of which time motion was completely abolished in the lower limbs.

In the eighteenth month of the disease, vesical spasm and strangury occurred and a week later, paralysis of the sphincters of the bladder and rectum. The lower limbs, which were paraplegic, atrophied and slightly œdematous, had lost their sensibility and electro-muscular contractility. The impairment of sensibility extended anteriorly to the sixth rib and posteriorly to the second lumbar vertebræ. Towards the close of the nineteenth month of the disease, the deglutition of liquids was alone possible. Death occurred at this time, after increase of the bed-sores and of the somnolence.

Autopsy.—Brain anæmic, hyperæmia limited to the cortex of the anterior lobes, atrophy of the posterior lobes with thinning of their convolutions. The white substance is strewn with patches varying in size from a hemp-seed to an almond, hard, resisting under the knife, of a pale gray color, and transparent. Similar patches were found in the corpus callosum, centrum ovale, optic thalamus, corpus striatum, anterior tubercula quadrigemina, pineal gland, interior of the atrophied cerebellum, anterior surface of the pons varolii, and in the medulla oblongata. The spinal cord is atrophied and extremely hard; nodosities are found upon the surface of dorsal region as well as upon transverse section; numerous patches, similar in appearance to the first, are visible at the inferior portion of the lumbar region and in the lateral and posterior columns. The gray substance, especially in the lumbar region, is of a pale red color and projects perceptibly above the plane of the transverse section.

On microscopical examination of the sclerosed patches in the brain and cord, I observed a firm net-work of wavy connective-tissue fibres, with cells scattered here and there. The sheaths of the vessels were thickened and infiltrated with proliferated nuclei. The cord in the anterior, and even more in the posterior portion, is crowded with connective tissue and amyloid corpuscles, surrounding small islets of healthy tissue. The large nerve cells of the anterior horns present few changes; the cells of the posterior horn, on the other hand, especially in the lumbar region, are very much diminished in number, atrophied, opaque, without prolongations, and infiltrated in places with a yellowish pigment.

Like the majority of cerebral lesions, "sclérose en plaques" also begins with symptoms of irritation which scarcely attract attention, such as headache, vertigo, nervous irritability, migraine, neuralgic pains in the

limbs, partial convulsions, etc. Weakness of motion and paralysis of one of the upper or lower limbs are soon superadded. In rare cases, these symptoms appear under the form of hemiplegia developing after epileptiform convulsions (Zenker, Léo, Hirsch). More frequently the lower limbs become gradually affected. At a later period their movements are executed with manifest difficulty, and are accompanied by hesitation and tremor. This latter symptom may be produced in voluntary movements, in communicated movements, and in consequence of excitement.

This peculiar paralytic tremor is rarely absent, and appears to be due to the presence of patches of sclerosis in the motor ganglia, pons varolii, and neighboring parts.

The paralysis of motion very often affects the muscles of the face and eyes, and especially those of the tongue, and results in slowness of speech, which is sometimes syllabic. Articulation of sounds may be completely abolished, and in the case which I reported above there was intermittent loss of speech. If the sclerosis affects the medullary nuclei of the nerves and the nerve roots to which they give origin, multiple paralysis of these nuclei (labio-glosso-pharyngeal paralysis) may develop. This has been observed several times by Leube and Schuele (D. Arch. f. klin. Med., 8 Bd., 1870) and explained by Joffroy (Gaz. méd. de Paris, 23 and 24, 1870) by the microscopical demonstration of lesions in the nuclei of origin of the hypoglossal and facial nerves. If the process involves the anterior horns of the spinal cord, muscular atrophy will also supervene.

In the final course of the disease we observe, though with less constancy, symptoms of motor irritation, such as spasms of the muscles of the face, clonic convulsions of the ocular muscles (nystagmus), persistent contractures and a periodical stiffness of the limbs, which sometimes affects one half of the body and sometimes the upper or lower limbs to a more marked extent, with or without disturbances of the intelligence.

Disorders of sensibility are not regarded as frequent symptoms of diffuse sclerosis of the nerve centres, but the absence of sensory disturbances is not, as authors still think, a characteristic sign. We can with difficulty comprehend how sensibility should remain intact in the presence of such frequent changes in the parenchyma of the cord. The sensory disturbances vary according to the extent and intensity of the medullary sclerosis. If the lesions of the posterior portions of the cord and the gray matter are slight, sensibility is only moderately affected. On the other hand, if the posterior columns are seriously involved as well as the adjacent parts (as in the sclerose en plaques and ribbon-shaped sclerosis of Bourneville and Guérard), there is considerable diminution and even complete abolition of sensibility.

The most complete loss of sensibility is met with in those forms in which the sclerosis and atrophy attack, by preference, the posterior columns and cornua, in a transverse direction. In a patient observed by Hirsch (Deutsche Klinik, 33–38, 1870), in whom there was insensibility of the lower limbs, the lesions were found to be present, to the greatest extent, in the posterior segments of the white substance of the cord. I may also here remark that, in the case which I have above reported, the anæsthesia extended along the distribution pointed out by Voigt upon the peripheral nerves. Several years ago I called attention to the existence of this phenomenon in other diseases of the spinal cord.

Irritative sensory symptoms are usually present in the beginning, sometimes also in the latter part of the course of diffuse sclerosis of the nerve centres. These symptoms consist of peripheral neuralgic pains, formication,

and increased reflex excitability, the latter being replaced by exaggerated contractions under the influence of mechanical or electrical stimulation. In my patient, faradization of the extremities gave rise, at first, to violent muscular contractions. When the intensity of the current was increased, tremor of the limbs was produced, which also extended in great part to the trunk on the opposite side.

The electrical reactions are not sensibly changed when the spinal cord is not much involved, as in Baerwinkel's case (Arch. d. Heilk. 6. H., 1869), in which the spinal cord only contained small patches in the form of little islands. When the inflammatory process involves the parenchyma to a more serious extent, and is disseminated throughout different portions of

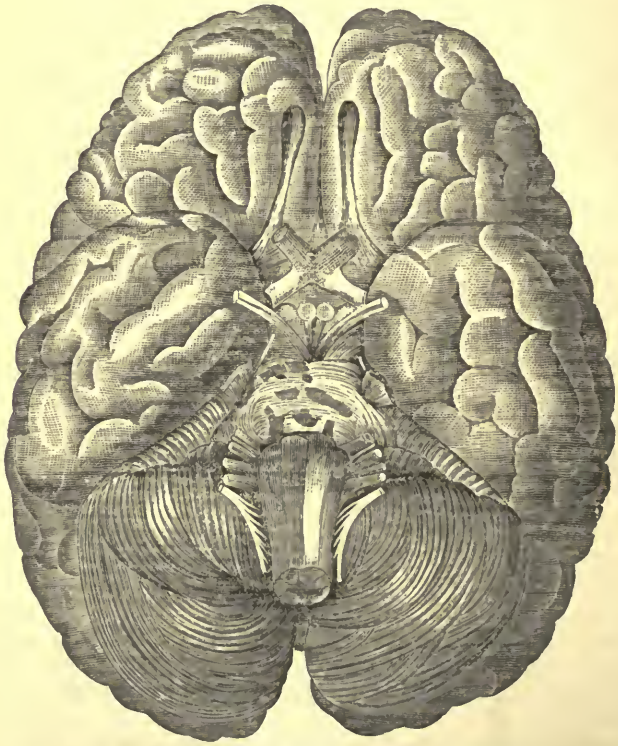

Fig. 7.—Inferior surface of the brain. Patches of sclerosis (darkly shaded) are visible upon the left cerebral peduncle, various parts of the pons varolii, medulla oblongata, and spinal cord.

the spinal cord, the electro-muscular contractility diminishes (as my case serves to show), together with the galvanic excitability of the nerves, and both may grow weaker and weaker toward the end of the disease.

The organs of special sense also take part in the disturbances of the nervous centres. Sight sometimes diminishes on account of the early atrophy of the optic nerves. Gray degeneration of the optic nerve, extending as far as the chiasm, may give rise to amblyopia and amaurosis of one or both eyes. Some patients, also, suffer from deafness in one ear, complete abolition of the sense of taste upon one-half of the tongue (Hirsch), perversion of taste and smell (Liouville and Melicher). These disturbances of

the organs of special sense are explained by the anatomical lesions found in the roots of the corresponding nerves.

The *psychical functions*, in the majority of cases, present symptoms of exaltation in the beginning, followed later by signs of depression. Most of these patients suffer from weakness of memory and intelligence, a condition of childishness, irritability, and fits of unmeaning laughing and crying. In other cases the patient is in a condition of melancholia and excitement. At a more advanced stage, weakness of intellect and complete apathy may supervene. Speech is generally difficult and slow; pronunciation is often syllabic, and the voice becomes feeble and monotonous.

In proportion as the disease progresses, all the symptoms become aggravated. It often progresses by fits and starts, and the patient sinks more and more into a condition of depression. The movements become much weaker and inco-ordinated, and sensibility and the special senses become more and more blunted. The psychical disorders and embarrassment of speech increase; mastication and deglutition become difficult, and the sphincters cease to act. The general nutrition also suffers to a great extent.

In the last stages of the disease, the paralysis, which is usually most intense in the lower limbs, gives place to contracture with loss of reflex excitability. Later, other paralyses occur in both the voluntary and involuntary muscles. Fever appears, either continuous or intermittent, and is due, in most cases, to intercurrent acute diseases. In other patients, on the other hand, we can discover no organic cause for the rise of temperature. The difficulty of speech, dyspnœa, increase of aphonia, and dysphagia, slowness and weakness of the pulse and elevation of temperature, testify to the progressive invasion of the bulbar nuclei and vaso-motor centres by the sclerotic process. The patient dies in collapse, usually attended with loss of consciousness.

Diagnosis and Prognosis.

In the beginning of the disease, so long as there are merely isolated and temporary symptoms of cerebral or spinal irritation, the diagnosis of the sclerosis is impossible. It can only be made after long observation and after the appearance of more characteristic morbid signs. The differential diagnosis must be especially made, on account of the similarity of symptoms, between sclerosis and paralysis agitans, cerebral softening, cerebral tumors, and ataxia.

Paralysis agitans is characterized by rhythmical tremor, which is propagated on one-half of the body from the upper to the lower limb, and is only increased by excitement or muscular effort. In walking, the patient inclines to the paralyzed side, and leans forward, and the muscular rigidity causes a peculiar deformity of the fingers and toes. There are no disorders of articulation, nystagmus, or increase of reflex excitability, or inco-ordination of movements. The existence of these symptoms prevent us from mistaking paralysis agitans for diffuse sclerosis of the nervous centres.

Foci of cerebral softening are accompanied by an early and sudden abolition of the psychical functions, by loss of speech or aphasia, and unilateral paralysis and contractures. They occur generally in old age. In the intervals of repeated attacks of apoplexy, resulting from encephalitis, the patients recover very promptly, and are affected merely by partial weakness of the power of motion in one limb. The apoplectiform attacks are much more infrequent than in sclerosis (Léo, Hirsch) and give rise, in addition to hemiplegia, to anæsthesia or paralysis of one or the other ex

tremities on the opposite side. They also cause tremor, loss of speech, and frequent neuralgias of the lower limbs. Cerebral tumors are distinguished from sclerosis by periodical headaches, attacks of vertigo, irritative phenomena, convulsions, the gradual development of hemiplegia and neuro-retinitis, and by the absence of tremor in the limbs and of especial disorders of speech. In order to distinguish sclerosis from ataxia we must remember that cerebral symptoms are wanting in the latter affection or appear at a late period, while, on the other hand, we frequently observe intermittent paralyses of the ocular muscles with diplopia. Lan-

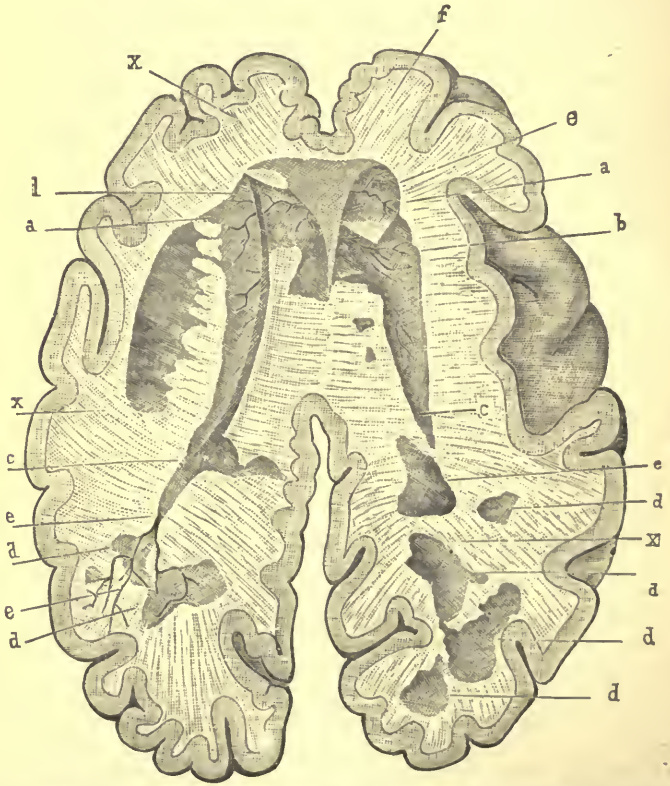

FIG. 8.—Horizontal section of the brain: *a*, patches of sclerosis in the anterior regions; *b* and *c*, patches in the walls of the lateral ventricles; *d*, patches scattered irregularly throughout the posterior lobes; *e*, large vessels in the midst of sclerosed tissue; *x*, normal white substance of the hemispheres.

cinating pains are more frequent along the sciatic nerve or in the arms, with girdling pains, irritation of the genital organs, increased galvanic excitability of the nerves, and, at a later period, ataxic disorders of movement.

As regards *prognosis*, all impartial observers agree in the statement that a fatal termination is the rule in diffuse sclerosis of the nervous centres. Even when the patient presents, at times, the appearances of an arrest of the disease, which may create the hope of recovery, a relapse usually occurs and terminates in death. The fatal termination is most frequently caused by intercurrent affections such as pneumonia, pleurisy,

tuberculosis, or lesions produced by bed-sores. Sclerosis may prove fatal within a period of two or three years. Its average duration is from six to ten years, and only a few cases are prolonged beyond this period.

Treatment.

If we are unable to arrest the course of less severe inflammatory processes, limited to certain regions of the brain or skull, we are even more powerless when the inflammatory proliferations extend into the substance of the brain and cord under the form of multiple foci. The onset of the affection, at which period we might perhaps hope for beneficial results from treatment, eludes our notice. The more advanced forms, with multiple lesions of the nervous centres, may be readily recognizable, but they remain unaffected by treatment.

The chloride of iron, recommended by Vulpian, has met with as little success in the treatment of sclerosis as the phosphide of zinc vaunted by others. Nitrate of silver and strychnia have served, in a great many cases, to moderate the tremor and weakness of the movements, but their action is not permanent. Galvanization (central and peripheral) and hydrotherapeutics have diminished the severity of the symptoms in some favorable cases, but without producing a permanent arrest of the sclerotic changes occurring in the nerve centres. Faradization is even harmful in its effects on account of its exciting action and the consequent increase of the tremor which it produces. We should abstain from its use in all well-marked cases of sclerosis.

CHAPTER XI.

CEREBRAL TUMORS.

Pathological Anatomy.

Among cerebral tumors, we may first mention those which are the result of a hyperplasia of the connective tissue (neuroglia of Virchow) extending throughout the nervous centres, and to which tumors Virchow has applied the term glioma (Krankh. Geschwuelste, Berlin, 1863–67). They are found isolated in the cerebral substance, and vary from half the size of a cherry-pit to that of a fist. They are often adherent to the meninges, and form masses which are similar in appearance to the cortical substance of the brain. They are sometimes white, and at times very vascular, and of a reddish-gray color. Gliomata of the brain, like those of the cord, develop very often in the white substance; they appear most frequently in the cerebral hemispheres.

Under the microscope we may, perhaps, find only a granular substance containing nuclei or cells of various sizes, from a rounded to a more or less oval shape, with finely granular contents, and one or two nuclei. Collections of fusiform and stellate cells, with one or two processes, may also occur in places. The fundamental tissue is formed of fine ramifying fibrillæ. The intercellular substance is sometimes soft and nearly fluid, sometimes more compact and firm.

In *soft* gliomata, the intercellular substance is less abundant and contains more or less fibrillary tissue, which in the myxo-gliomata assumes a finely reticulated appearance. When the stellate cells form a large meshed network with increase of the myxomatous tissue, it leads to the formation of myxomata or of the various mixed forms. The increase of the cellular products, and the narrowing of the meshes of the network, give rise to the development of gliosarcomata.

In *hard* gliomata, the fundamental structure is composed of very fine fibrillæ, arranged parallel or interlacing, or it forms a compact tissue of bundles or lamellæ, containing in places nucleated cells (fibro-gliomata).

The hæmorrhagic tendency of gliomata of the brain and cord is caused, as Virchow has shown, by their great vascularity. The blood coagulates and forms whitish-brown or red nuclei of a very firm consistence, looking like fibrinous concretions in the spleen, or like tubercles or gummata.

In general, gliomata develops slowly with inappreciable symptoms. If they enlarge more rapidly, or if they possess very great vascularity, congestions and even hæmorrhages may be produced. When the tumor attains a considerable size it may give rise to irritation, cerebral compression or dropsy of the ventricles (*vide* die Krankh. Geschwuelste, Virchow, II. Bd., 1 H.). On the other hand, in gliomatous tumors containing yellowish or reddish portions, we find a fibrous, resisting tissue, thickening of the tunica adventitia of the vessels, dense bands of interlaced connective

tissue, atrophy of the cells, a deposit of reddish-brown pigment, and signs of atrophy. In the vicinity of the tumor we may observe, according to E. Wagner, fatty granulations, crystals of cholesterine, nuclei of the neuroglia, and débris of axis cylinders, as the remains of the necrosed nerve tissue, in addition to the redness and softening. In fatty degeneration, with removal of intercellular substance, cavities form in the same way as in cerebral softening. But these cavities are distinguished from true cysts by the fact that their walls are covered with villosities, and are not sharply defined, and by the persistence of permeable vessels.

Sarcoma and its different varieties, recently investigated by Virchow (gliosarcoma, myxosarcoma, etc.), are usually situated in the cerebral hemispheres, anterior lobes, optic thalamus, processus cerebelli ad pontem (according to Virchow and Friedreich), and in the cerebral peduncles. The consistence may be either firm or soft, the interior is more compact, the periphery often rugous, and the surface nodular. They frequently adhere to the meninges, and expose the brain to the dangers of compression, softening, or inflammation, which develop in their neighborhood.

The *cholesteatoma* (pearly tumor of Virchow) arises usually from the arachnoid (Rokitansky), and more often from the pia mater than from the dura mater or interior of the cerebral structures. It must not be confounded with the masses of cholesterine which are often found in the choroid plexuses. The pearly tumors form crystalline masses which, in the isolated state, are as large as a grain of mustard, and by their aggregation attain the size of a goose-egg. They are enclosed in a thin membrane of an indistinct fibrous structure. They present an irregular shape, and their wavy surface possesses a beautiful mother-of-pearl lustre. These tumors, deprived of vessels, exhibit, on section, concentric layers of epidermic cells, partly in a condition of vitreous and partly of fatty degeneration.

They develop slowly, and very often remain latent. Only after a long period do they provoke inflammatory reaction in the neighboring tissues. In a case of pearly tumor of the mastoid process, Virchow saw an abscess develop in the adjacent portion of the brain. In another case, in which caries of the mastoid process and otorrhœa had existed during life, thromboses occurred in the transverse sinus, extending even into the jugular vein.

Tubercles of the brain vary in size from a pea to a goose-egg, and are found most frequently in the cerebral hemispheres and cerebellum, less often in the corpus striatum, optic thalamus, peduncles, pons varolii, and ventricular ependyma (Foerster), and very rarely in the fornix and medulla oblongata. In children, they are most frequently observed in the first of the above-mentioned situations. With the exception of the discrete forms of tubercle in the pia mater and cortex, tubercle usually consists of an agglomeration of several nodosities, which, under the microscope, are found to contain rounded cellular elements, partially atrophic, and in a condition of fatty degeneration, and enclosed in a delicate network, with nuclear proliferation of the walls of the vessels (Wedl).

Cancer of the brain is a very frequent form of intra-cranial tumors. It is usually primary, and generally remains isolated for a long time. Of 48 cases collected by Lebert, 45 were primary, and 13 of these were complicated with carcinosis of other organs.

In the cerebral tissue primary cancer is almost always isolated. When several tumors are present, the interesting fact has been noted that they develop symmetrically in homonymous parts of the brain (Rokitansky). In those forms which affect the brain secondarily, we usually find several tumors of small size. The largest carcinomata are those which pass

through the skull, the varieties of scirrhus which start around the orbits, and those tumors which occupy the centre of a cerebral hemisphere. Those situated in the pons, base of the brain, and medulla oblongata, are the smallest in size ; cancer is very rarely found in the latter situation, in the corpus callosum and in the tubercula quadrigemina. It is much more common in the optic thalamus, corpus striatum, and in the cerebellum.

The primary forms may attain the size of a fist. Their growth is much more rapid, according as their tissue is rich in cells and blood-vessels. The ordinary form of cerebral cancer is the medullary variety, while fibro-carcinoma is more infrequent. Melanotic cancer is generally secondary, and may, according to Rokitansky, cause death by the rapid and multiple development of cancerous products in the brain. Cerebral cancer exercises an injurious influence upon surrounding parts by its pressure effects, causing atrophy of the parenchyma, secondary anæmia or hyperæmic stases, hæmorrhages, inflammation, œdema, softening, and even abscess. Many carcinomata, especially those which are connected with the bones, present ossification of their stroma. The medullary varieties may undergo a tubercular degeneration, an occurrence which has given rise to confusion between cancer and tubercle of the brain. According to Rokitansky, a partial transformation of soft cancer may present a somewhat similar appearance to foci of encephalitis.

Syphilomata of the brain are rare. They usually originate in the thickened and degenerated meninges, and form nodosities varying from the size of a pea to a hazel-nut, yellow, lardaceous, hard, and distinctly separated from the adjacent tissues.

We usually find other manifestations of the syphilitic diathesis upon autopsy, but we shall discuss this subject at more length in the chapter on cerebral syphilis.

Osseous intra-cranial tumors are rarely met with, if we exclude the partial ossification of cancerous products and enchondromata. *Syphilitic exostoses* are more frequent, usually starting from the external surface of the cranial vault, but also situated at times upon the internal surface, and giving rise, by their development, to the symptoms of cerebral compression which appertain to other tumors occupying a similar situation. We have also observed *osteoid growths* (after injuries or spontaneous inflammatory processes of the cranial bones or cerebral substance) under the form of large tumors, denticulated, looking like apophyses, globular, sometimes compact, sometimes porous. In very rare instances we find osteomata of the cerebellum, composed of real osseous tissue with medullary cavities, such as Virchow has described as following circumscribed encephalitis in young subjects. We may also mention, in this category, calcareous degeneration of tubercles and of the envelopes of cysticerci.

Cystic productions, *myxomatous tumors* situated in the sella turcica, and the *psammomata* of Virchow (which are formed by a white mass of crystalline granulations), are of small size, rarely observed, and usually do not give rise during life to any especial symptoms.

General Symptomatology.

The first stages in the development of cerebral tumors, usually remain obscure. Only a small number of tumors of the brain are entirely free from symptomatic manifestations during life. It has been shown that, in these cases, the new-growth occupies a very small space, and develops

slowly, compressing the parenchyma little by little, but without affecting it seriously, and without producing any manifest interruption in the conducting fibres. A tumor which is soft or deficient in blood-vessels is not subject to any considerable change of volume. Finally, the seat of the tumor is especially important, since even large tumors, imbedded in the hemispheres, often provoke no symptoms, while other growths, which are much smaller, may give rise to considerable functional disturbance in the cerebral activity, to irritative lesions in the neighboring parts (even to forms of descending neuritis), or may give rise to nutritive disturbances in remote portions of the brain in consequence of the compression of the vessels which cross the tumor. Large tumors may, even for a long time, escape careful observation. The disease passes unnoticed by the patient (who is often in the best years of life), and even by the physician, until suddenly some serious symptom gives the alarm. The fatal growth continues its ravages within the brain until the danger is revealed by symptoms of evident gravity.

The series of events which Wunderlich describes as general and initial symptoms, usually begins with headache (which the patients refer to the frontal or temporal region, and sometimes to the occipital region, without any constant relation being found between the pain and the site of the tumor). Ladame (Symptom. und Diagn. d. Hirngeschwuelste, 1865) has found headache present in two-thirds of the cases. The pain is, at first, intermittent, and, later, it appears in paroxysms. It then becomes remittent, but sometimes continuous, and is rebellious to treatment. It is caused by local congestions, and by the compression exercised upon certain parts of the brain or upon the acutely sensitive pia mater. In most cases, vertigo also appears in connection with the neuralgic headache. According to the most recent experiments of Goltz, Breuer, etc., this is due to an affection of the semicircular canals which represent the terminal apparatus of the sensation of equilibrium, and to a sensorial perception caused by a deviation of the lymph-current in these canals. The more this organ, situated within the temporal bone, is irritated, as by pressure from a neighboring tumor, the more intense does the vertigo become. This also occurs in consequence of lesion of the fibres of the posterior columns in the cerebellum.

Paroxysmal headache and vertigo may, for several months, be the only symptoms which distress the patient, who in other respects may enjoy good health. As a natural consequence, the situation, in the majority of cases, does not inspire the attending physician with any serious apprehensions. In general, these symptoms are very soon followed by disturbances of sensation and motion, which may be regarded in the beginning as signs of irritation, and later as evidences of depression. We not infrequently observe, side by side with paralysis of one region, symptoms of irritation in adjacent regions. This alternation is due to collateral hyperæmia or œdema, while, in other parts, conduction has been already destroyed.

The symptoms of sensory irritation often appear as precursors of disorders of motion. They consist of painful twinges, formication, and numbness in the extremities (often accompanied by reflex cramps), and, in certain cerebral tumors, trigeminal neuralgia occurs. An increase of sensibility and reflex action is often observed upon one side. This heightened sensibility usually lasts for a short time, and gives place to a more permanent anæsthesia. Painful anæsthesia of the paralyzed limbs is an exceptional occurrence. Ladame observed cutaneous anæsthesia in one-

seventh of all his observations. The frequency of sensory disorders depends upon lesions of the paths of transmission at different points below their central termination. Alternate hemianæsthesia sometimes occurs.

The initial motor disturbances consist of a sensation of stiffness, a relaxed condition of one limb, and spasms in various parts of the body. These spasms vary in intensity, from slight contractions of the facial muscles, to tonic, clonic, or choreiform muscular spasms (with disorders of co-ordination, Duchek), or tremor of a limb, or of one-half of the body. Contracture sometimes occurs in the muscles of the neck, jaws, or extremities. In some cases the paralyzed parts are affected by the convulsions. The convulsive paroxysms often assume the appearance of an epileptiform attack. In a case under my observation (chronic cephalalgia with total amaurosis and left facial paralysis), loss of consciousness occurred from time to time, lasting from ten to fifteen minutes, with convulsive movements in the upper limbs and extension of the lower limbs. In addition, the face was colorless, the patient lay upon the side corresponding to the tumor, and the pulse was slowed to 44 or 40 pulsations per minute. Attacks of this nature may be due to hyperæmic enlargement of the tumor and rapid increase of the compression, causing reflex spasm of the cerebral vessels, and consequently anæmia of the brain.

After a longer or shorter duration of the symptoms of motor irritation which we have enumerated, an abolition of motor power becomes gradually established under the form of paresis or hemiparesis, of partial paralysis, of hemiplegia or paraplegia. The paralysis usually progresses from above downwards, the opposite course being much more infrequent. The most common form of paralysis is hemiplegia (Ladame has found it in one-third of his cases). It appears in the domain of the cerebral as well as in those of the spinal nerves, and usually upon the same side, the cerebral lesion being situated upon the side opposite to the paralysis. The lesion is very rarely situated upon the same side as the hemiplegia. Crossed hemiplegia consists in paralysis of the limbs upon one side and paralysis of the cranial nerves upon the opposite side (alternate hemiplegia of Gubler). These alternate paralyses have an especial importance in the diagnosis of certain cerebral tumors.

Certain complexes of symptoms of cerebral growths, such as paralysis of the limbs and cranial nerves upon the same side, will be investigated in the discussion of tumors of the pons varolii.

As a consequence of the irritation of the hemispheres or of other portions of the brain in tumors, we may observe, according to Benedikt, an "exhaustion reaction" in the paralyzed muscles, *i. e.* a rapid diminution of reaction to faradic excitations of short duration. At other times, the reaction assumes a convulsive character, as is shown by sudden and abnormal increase of electro-muscular contractility. In the same manner we may find, according to Brenner, an increase or diminution of the secondary galvanic excitability. After prolonged compression of the roots of the peripheral nerves, by tumors of the pons or base, a gradual abolition of the nervo-muscular faradic contractility, with increase of the galvanic idio-muscular contractility, occurs, as I have shown, especially upon the paralyzed half of the face, while the electrical contractility of the muscles, in those paralyses which take their origin in the hemispheres or in the ganglia, is left intact or sometimes slightly increased. Among the disorders of special sense we may mention the disturbances of vision as the most important, both on account of their frequency and intensity. Amblyopia is present, according to Calmeil, in about two-fifths of the cases,

and amaurosis, according to Ladame, in one-fifth of the cases of cerebral tumor. We may recognize with the ophthalmoscope, intra-ocular swelling of the optic nerve, optic neuritis and atrophy. Inflammation of the optic nerve may present itself as papillary stasis and descending neuritis. In the first variety we may recognize the real seat of the inflammation by the cloudy appearance, considerable swelling of the papilla, and the unusual enlargement of its contours. In advanced forms we find, according to Leber, upon transverse section of the optic nerve, thickening and œdema of the internal sheath and of the laminated tissue between the external and internal sheaths, with hyperplasia of this tissue. The nerve fibres of the papilla are increased, according to Schweigger, to four or six times their normal diameter. In descending neuritis, the inflammation affects the papilla, which is slightly swollen, less than the adjacent portions of the retina. When the neuritis has continued for a time it is usually followed by consecutive atrophy of the optic nerve; the papilla is then depressed and of a dull white color. According to the microscopical investigations of Schweigger, Saemisch, etc., we find, in addition to proliferation of the connective tissue of the papilla, atrophy of the nerve fibres and ganglion cells of the retina, with fatty degeneration and thickening of the external coats of the vessels. Sometimes simple atrophy of the optic nerve occurs (the papilla of a shining white color) without any evidences of inflammation.

The retinal changes consecutive to these neuritic processes may some times develop without appreciable subjective disturbances of vision; much more infrequently we find diminution of sight (amblyopia) with few ophthalmoscopic evidences. In the majority of cases, when symptoms of tumor exist, we may obtain important diagnostic signs by ophthalmoscopic examination. At a more advanced stage, the amblyopia usually changes to complete blindness of both eyes. In a case which I observed in a physician, progressive narrowing of the field of vision occurred from the periphery towards the papilla, due to a fatty degeneration which gradually advanced from the two sides to the centre of the optic nerve. An analogous process is found in atrophy of the facial nerve, in consequence of compression from tumors or from caries of the mastoid process.

The development of optic neuritis and of secondary atrophy of the optic nerve, is due to different causes. The central fibres of the optic nerve may be the starting-point of the neuritis and descending atrophy, in consequence of tumors compressing the corpora geniculata, tubercula quadrigemina, cerebral peduncles, pons varolii or cerebellum. New growths or inflammatory processes situated at the base of the brain may, in addition to other symptoms, produce optic neuritis. Finally, according to Graefe, lesions of the optic nerve (especially papillary stasis) may be attributed to compression of the cavernous sinus, and to an obstruction to the return of blood occasioned by the inextensible ring of the sclerotic. But Seseman has since proven (Arch. f. Ophth., XII. Bd.) that compression of the cavernous sinus is not followed by any considerable venous stasis in the retina, so long as the anastomoses with the facial vein remain permeable. Another explanation must therefore be sought for the neuritic symptoms in question. We must here take into consideration the researches of H. Schmidt (Arch. f. Anat. u. Phys., 1869), who has demonstrated, by means of injections, the existence of a communication between the cavity of the arachnoid and the lamina cribrosa; the latter becomes œdematous when fluid is forced from the arachnoid by increase of the cerebral pressure, and gives rise to stases and inflammation by strangulation of the terminal intra-ocular portion of the optic nerve.

Auditory disturbances are a frequent symptom of cerebral tumors. Calmeil has found them in one-ninth of his cases. Often there is merely enfeebled hearing or buzzing in the ears; in seventeen cases complete deafness was observed, which, in one case, was only temporary. Since a communication exists, according to E. Weber's experiments (Monatb. f. Ohrenh., 1869), between the arachnoid cavity and the labyrinth through the aquæductus cochleæ, we can readily understand why an excess of cerebral pressure should react upon the auditory apparatus, in the manner already pointed out for the eye, according to Schmidt's experiments. In addition, the auditory disturbances may be due to compression of the trunk of the acoustic nerve, and of the fibres which pass underneath the flocculus, median cerebellar peduncle, and pons varolii. In a case of fibro-sarcoma, recently described by Boettcher of Dorpat (Arch. f. Augen- und Ohrenh., II. Bd., 1872), the tumor was situated on the left side near the pons varolii, and extended to the porus acusticus; in addition to atrophy of the chiasm and retinal lesions, Boettcher found atrophy of the lamina spiralis of the cochlea; in the axis of the cochlea were bands of connective tissue rich in nuclei, but without any traces of nerve fibres; the membrane of Corti was markedly striated, and the internal and external auditory cells were replaced by small round chambers.

Smell is much more rarely involved in cerebral tumors. But the statistics upon this point, found in medical literature, are evidently very deficient, because the patient is often in such a condition of prostration that the loss of smell does not inconvenience him, and also because, in the majority of cases, the physician fails to pay any attention to the condition of this function.

I have found smell notably disturbed in two cases of tumor at the base of the brain; one of them was situated upon the left anterior half of the pons, and the other upon the left cerebellar peduncle. In one of these cases the patient distinguished odors through the left nostril with much more difficulty and much more confusedly than through the right. In the other case there was complete anosmia of the left nostril. Upon hermetically closing the right nostril, the patient could not distinguish, upon the left side, either alcohol, ether, creosote, sulphuretted hydrogen (the mouth being closed), asafœtida, etc. When a bottle of ammonia was placed under the left nostril, the patient stated that he experienced a burning sensation, which was evidently due to the effect produced upon the filaments of the trigeminal nerve on the internal surface of the nares. Upon the right side, smell was not very delicate, but the patient could, nevertheless, recognize sufficiently well the majority of the substances which we have named.

Since the olfactory nerve, according to our present knowledge, is the only cranial nerve which does not leave the cerebral hemispheres, it remains subject to their influence, and especially to that of the anterior lobes (according to Meynert, however, it is under the dominion of the temporal lobes). Serious lesions of these lobes, especially at the base, where the olfactory bulb is situated upon the anterior cribriform lamina, will give rise to more or less profound disturbances of smell.

We have, also, only an incomplete knowledge concerning the sense of taste in cerebral new-formations; careful attention, however, in this direction would furnish important data in the physiology of the gustatory functions. In the two patients above referred to, the sense of taste was also involved.

The first patient (who had incomplete loss of the sense of smell upon the right side) did not perceive the taste of a concentrated saline solution placed in contact with the left half of the tongue. In the second patient (who had complete anosmia of the

left side), explorations made with the necessary precautions upon the tip, sides, and base of the tongue, and upon the roof of the palate (moistening them with solutions of acids, chloroform and sulphate of quinia), showed normal perception of taste in none of these parts. After moistening, for a long time, the region of the cup-shaped papillæ, or when the posterior part of the tongue was applied against the palate, the patient several times stated after the application of quinine or cinnamon, that he perceived a bitter or acrid taste.

This observation is interesting from a physiological point of view, be cause it demonstrates that especially the base of the tongue, the corresponding portion of the roof of the palate, and the isthmus of the fauces, are concerned in the perception of the most delicate gustatory sensations (especially those of bitterness and acridity); this is perhaps due to the more delicate structure, and to the greater number of nerve fibres furnished by the glosso-pharyngeal nerves to the cup-shaped papillæ (Koelliker). In Boettcher's above-mentioned case, the patient also complained of a scalding sensation in the mouth, and of a bitter taste. At the autopsy the glosso-pharyngeal and pneumogastric nerves were found to be compressed by the tumor, and in a condition of fatty degeneration.

More or less severe disturbance of the organic functions is also observed in these patients. Violent cephalalgia may disturb the sleep of the patient at night, and the prolonged insomnia thus developed may have a very injurious effect upon the general condition. Vomiting may occur at the close of violent paroxysms of headache, or even without any increase of the pain (either with or without regularity), and its frequent repetition compromises nutrition. We also observe signs of irritation on the part of the vagus nerves, such as irregularity of the heart and slowness of the pulse, and these are sometimes evident in the course of convulsive attacks; we have already cited a case of this character. The respiratory rhythm is sometimes changed; it may be accelerated in cerebral irritation and slowed in cerebral compression. In Vierordt's and Hegelmaier's experiments upon rabbits, in which the respiratory movements of the upper part of the abdomen were registered upon the drum of the kymograph, moderate artificial compression of the brain was found to diminish the number of respirations to one-half the normal amount, while, on the other hand, increased compression accelerated it. In the first case, the inspirations became more infrequent and the expirations longer. Polyphagia only exists in some patients; in general it is without any favorable influence upon the lowering of the nutrition. In a case which I reported some time ago, the polyphagia was accompanied by polyuria and glycosuria.

The disorders of nutrition do not run a parallel course with the severity of the cerebral symptoms. In general, the symptoms of decline appear more promptly when the tumors are of a cachectic origin. In some cases, however, cancer may have been present for a long time, although the patient has preserved an appearance of good health and patients suffering from sarcomata may even present a certain amount of obesity. Obstinate constipation exists in the majority of cases. In two instances which I recently had under observation, coitus could only be accomplished with difficulty, and was followed by general prostration for a considerable period. Wunderlich has observed impotence in cerebellar tumors, and Friedreich has seen one example of priapism. Psychical disorders are not rare in cerebral tumors. Authors differ very much in their estimates of the frequency of these disturbances. Friedreich gives the proportion as 43 per cent; Calmeil has found them in half, and Lebert and Ladame

in one-third of the cases; Andral and Durand-Fardel have noticed them much more rarely. These symptoms also belong to two categories, viz., those of irritation and those of depression. The first consist of irritability, agitation, absence of mind, hallucinations, melancholia; in severe forms, even attacks of mania may occur. The symptoms of depression consist of somnolence, apathy, idiocy, embarrassment of speech, and imbecility.

The psychical disturbances usually appear at an advanced stage of the cerebral affection, and not unfrequently decided remissions occur. These disorders are explained by the atrophy and discoloration of certain parts of the cerebral cortex from the pressure exercised by the tumor. They may also be attributed, on the other hand, to interruptions occurring between those fibres of the corona radiata which take their origin in the ganglia and the corresponding cellular portions of the cortex.

Speech is more or less seriously involved in cerebral new-growths. Sometimes it is markedly embarrassed, confused and unintelligible; sometimes the articulation of sounds is obstructed by sputtering; in some cases, partial loss of speech exists with preservation of intelligence (aphasia); more rarely, intermittent loss of speech occurs, as we have already stated. The disorders of speech, resulting from disease of the brain, have nothing in common with stuttering properly so-called; for here, even in the most marked and rebellious forms, the tongue has preserved entire freedom of motion. Ladame has cited forty-five cases of disturbances of speech in which the tumor occupied the most varied regions of the brain. It has been determined, from a very large number of observations, that tumors of the ganglia and pons varolii are most frequently accompanied by disorders of speech, while, according to Ladame, the tumors of the convexity, pituitary region, and cerebellum, exhibit the smallest proportion.

From what has been already stated, we must consider the disturbances of speech as due to paralysis of the tongue (alalie) or, according to Leyden, to lesions of the olivary bodies and pons (anarthrie). Aphasic disorders of speech are only observed when the tumors occupy the island of Reil, or the parts which unite this with the frontal lobe and with the central and parietal convolutions. We shall mention cases of this kind under the tumors of the anterior lobes of the brain.

According to the situation and growth of the tumor, the symptoms which we have already mentioned are followed, after a longer or shorter period, by the "final" symptoms (Wunderlich), with loss of voluntary motion, of automatic excitation, and of the psychical functions; the patient then becomes comatose and soon dies.

Differential Diagnosis.

The formation of tumors within the brain may sometimes be mistaken for other cerebral diseases with similar symptoms. In the majority of cases, by taking into consideration the etiological conditions and the chief pathognomonic signs, we will not remain long in doubt concerning the diagnosis.

Cerebral tuberculosis (especially in the chronic forms) is differentiated from tumors by its more frequent appearance in young subjects, especially in children. The latter, apart from chronic hydrocephalus and, still more rarely, from cerebral hypertrophy, are not very liable to other chronic

cerebral diseases. We should also lay great stress upon the coexistence of tuberculous affections of the cranial bones, and of the mastoid process (with symptoms of caries, fistulæ, ulceration and otorrhœa), upon the evident influence of heredity, and upon the rapidity of its course (which generally occupies from three to nine months, and, in very rare cases, several years). Primary tuberculous products, limited in rare cases to certain portions of the brain (pons, cerebellum), manifest themselves during life by tumor symptoms.

Chronic hydrocephalus may result from disturbances of circulation arising from the presence of a tumor, and when its symptoms are not prominent, it may be difficult to distinguish them from the symptoms proper of the neoplasm. Chronic hydrocephalus, in children, often accompanies cerebral tuberculosis; in adults, the coexistence of diseases of the heart, kidneys, or spleen is in favor of hydrocephalus; in the latter, imbecility is more frequent and marked than in tumors, which are more often characterized by the chronic disturbances of sensibility and motion which we have already mentioned.

Cerebral apoplexy occurs in the course of diseases of the heart and vessels, or in disorders of the pulmonary circulation, and usually at an advanced age; it is also distinguished from tumors by its sudden development, or by the existence of very slight prodromata; the patient recovers quickly in favorable cases, though paralysis of one-half of the body persists. In tumors, the cerebral symptoms are generally of older date. The cephalalgia, vertigo, neuralgia, and spasms grow gradually worse; and these events precede the apoplectiform attacks (which are rare), or they even persist, after the occurrence of the latter, as symptoms of excitation or depression. Optic neuritis, which is usually double, belongs to cerebral tumors (not, however, unilateral embolic amaurosis, which is rarer).

Chronic cerebral softening, occurring in the course of a latent encephalitis, differs in many respects from cerebral new-growths, as has been especially pointed out by Durand-Fardel in his "Treatise on the Diseases of Old Age." We shall merely allude to the principal differential points. The paroxysms of cephalalgia are less frequent and violent in chronic cerebral softening than in tumors; the disorders of the special senses (amblyopia, amaurosis, anæsthesia in the territories of the cranial nerves) are observed much more frequently in tumors than in encephalo-malacia; in softening, on the other hand, we more often notice weakness of the psychical faculties, the appearance of contractures, sudden and complete hemiplegias and disturbances of speech under the form of aphasia (most frequently of embolic origin). Alternate and double paralyses also occur, according to Hasse, by preference in tumors, and very rarely in softening. We have already spoken in detail of the distinction between tumors and abscess of the brain.

Cerebral atrophy (in its acquired form) is almost always distinguished with readiness from tumors. It is manifested by premature decay of the intellectual faculties, with a progressive transition to imbecility, and tremor of the lips, tongue, and limbs, constituting the prodromal signs of paralysis, when the atrophy extends towards the spinal cord. The data necessary for a diagnosis will be found in the ensemble of the following symptoms: the paroxysms of cephalalgia, the affections of the special senses, absence of convulsive but not of epileptiform attacks (Erlenmeyer), the relatively short duration of the disease (1–3 years); hemiplegia or paraplegia is usually superadded to the deterioration of the intellectual faculties; the

nutrition of the muscles soon changes. When the cerebral atrophy is one of the sequences of a new-growth, the symptoms characteristic of the latter remain more prominent.

The *cerebral hypertrophy* of children presents some analogies to cerebral tumors by its long duration and the existence of cephalalgia, and of epileptiform attacks. Nevertheless, the rarity of this affection, the enlargement and strong pulsation of the large fontanelles (Mayr), the progressive dilatation of the skull, the presence of a bruit de soufflle, manifest evidences of rachitis upon the bones, the softness of the bones of the skull and legs (Betz), the laryngeal spasms, and the condition of asphyxia which usually accompanies them—all these phenomena render it easy to differentiate, in children, the disease in question from a cerebral tumor or chronic hydrocephalus.

Cysticerci of the brain and their distinctive characteristics will be discussed in detail in the chapter on cerebral parasites. We have already mentioned the most important points with regard to the symptoms of aneurism of the arteries at the base, and of tumors of the base of the skull (which are, in general, very difficult to distinguish) in the considerations upon meningeal apoplexy.

Syphilis of the brain usually has many cerebral symptoms in common with tumors. Certainty of diagnosis is of especial importance in this affection, on account of its relations to prognosis and treatment. The previous history, the presence of evident manifestations of the diathesis, the peculiar pains in the bones and nerves, the epileptiform attacks supervening at a mature age after previous irritative symptoms, their disappearance under specific treatment, all these phenomena, joined to prolonged and careful observation, render the diagnosis positive. We shall again refer to this subject more in detail in the chapter on cerebral syphilis.

The differential diagnosis between the initial symptoms of ataxia and certain cerebral tumors (of the peduncle or cerebellum) will be fully discussed at the proper time.

Diagnosis of the position of Tumors.

We shall now begin a detailed study of cerebral tumors with regard to their situation, and, with this end in view, shall collect the results of the latest works upon the subject, as well as of our personal observations (the number of which reaches fifteen). The classification which we have adopted is as follows:

I.	Tumors of the	Convexity of the	Brain.
II.	"	"	Anterior Lobes.
III.	"	"	Middle Lobes.
IV.	"	"	Posterior Lobes.
V.	"	"	Motor Ganglia of the Brain.
VI.	"	"	Optic Thalamus and Tubercula Quadrigemina.
VII.	"	"	Middle Cerebral Fossa and Region of the Gasserian Ganglion.
VIII.	"	"	Pituitary Region.
IX.	"	"	Cerebral Peduncles.
X.	"	"	Pons Varolii.
XI.	"	"	Cerebellar Peduncles.
XII.	"	"	Cerebellum.

I. Tumors of the Convexity of the Brain.

The severity of the symptoms depends upon the depth to which the tumors penetrate, and upon the degree of irritation to which certain deeper portions of the brain are directly or indirectly exposed. In animals we may remove a large part of the cerebral and cerebellar hemispheres without the slightest sign of irritation becoming manifest. Frogs, birds, and even rabbits tolerate removal of the cerebral lobes without presenting any symptoms of paralysis, but when the dog is subjected to this experiment, he immediately becomes paralyzed. In individuals who have been trephined, we may raise portions of the hemispheres with the curette without their knowledge. Abscesses and tumors, especially those of soft consistence, may exist in the brain without causing serious disturbance. In a case which I observed in a tuberculous subject at the Vienna General Hospital, a tuberculous deposit as large as a hazel-nut was found upon the convexity of the brain on the right side, without any especial symptoms being evident during life.

According to the recent investigations of Charcot and Pitres, tumors and lesions limited to the anterior portions of the convexity of the cortex and those in the sphenoidal and occipital lobes, etc., do not give rise to persistent paralysis.

Destructive lesions which are parallel to the principal directions of the fibres in the cerebral hemispheres are generally (as also in the spinal cord) less serious, according to Valentin, than lesions which are perpendicular to the course of the fibres. If, nevertheless, the primary activity, as far as this is possible, becomes re-established after a short interval, this can only take place by collateral paths, since regeneration of cut fibres has not been observed in animals. When, after excision of considerable portions of the two cerebral hemispheres, the animals react to stimulation of the skin by cries or attempts to escape, or when birds touch accurately with the beak those points at which they feel the presence of a parasite, these movements are not performed under the influence of the brain, but are presided over by the ganglionic masses and their anastomosing fibres in the medulla oblongata and spinal cord. Such movements of reaction only disappear when the medulla oblongata is removed. According to Flourens (Recherches experimentales sur les propriétés et les fonctions du systéme nerveux dans les animaux vertébrés, Paris, 1824, p. 100) the successive excision of the cerebral hemispheres by deeper and deeper layers, causes abolition of the different sensations of the animal; still later, the destruction of the brain is followed by deafness. The sensations reappear at the end of some time, and are suddenly manifested under the most varied influences.

Cephalalgia is one of the most frequent symptoms of tumors of the brain. It is situated sometimes in the frontal region, sometimes in the occipital region, or upon the side of the head, and in the latter event the tumor is often upon the same side. Sensory disturbances are very infrequent. In Finger's case (Prag. Vierteljschr., 57. Bd., 1860) the headache was accompanied by painful sensations and formication in the right arm; these symptoms disappeared after a time, and were replaced by anæsthesia of the limb. At the autopsy, a tuberculous tumor of the size of a walnut was found upon the convexity of the left cerebral hemisphere; the pathological changes extended to the deeper portions of the brain, lesions of which are accompanied by anæsthesia of the limbs. The motor disturbances are much more frequent and characteristic, and are usually manifested as symptoms of irritation. Lebert has found them present eleven times in thirteen cases, and Ladame, in his collection, has noted them twelve times in seventeen cases. These symptoms consist of epileptiform

attacks, convulsions of one-half the body, or only of one limb. Paralyses of a hemiplegic type are very rare, and are due to softening which has extended downwards across the motor tracts.

A patient, aged 26 years, suffering from Pott's disease, was affected from time to time, during the past year, with spasms in the limbs upon the right side. Consciousness was not involved, but, under the influence of very strong and general convulsions, the patient lost the power of speech. Later, the attacks occurred at intervals of a few days, but were markedly moderated and retarded by the use of atropine. At the end of several months a whitish tumor developed upon the right knee, and disorders of the mental faculties then occurred, with weakness of memory, confusion of ideas, and a rapid loss of flesh. Upon autopsy several tuberculous tumors of the size of a walnut were found upon the convexity of the left cerebral hemisphere.

Disturbances of the special senses are quite rare in tumors of the convexity. Lebert and Ladame have noted amblyopia and amaurosis in some cases; in Fischer's case the coexistence (for three years) of internal otitis, with difficulty of hearing, led Traube to make a diagnosis of cerebral abscess. The intelligence, also, sometimes presents symptoms of irritation (exalted ideas, furious delirium, etc.). Vomiting is a less constant symptom than constipation. The fever and strabismus, which sometimes occur, should be referred to an intercurrent meningitis.

We may, therefore, mention as characteristic symptoms of tumors of the convexity, the frequency of convulsions and epileptiform attacks, the rarity of paralyses and sensory disturbances, and symptoms of irritation on the part of the intelligence.

II. Tumors of the Anterior Lobes of the Brain.

The important part played by the anterior portion of the hemispheres, in the production of voluntary movements, has only been demonstrated by recent investigations. Histological researches have shown that the

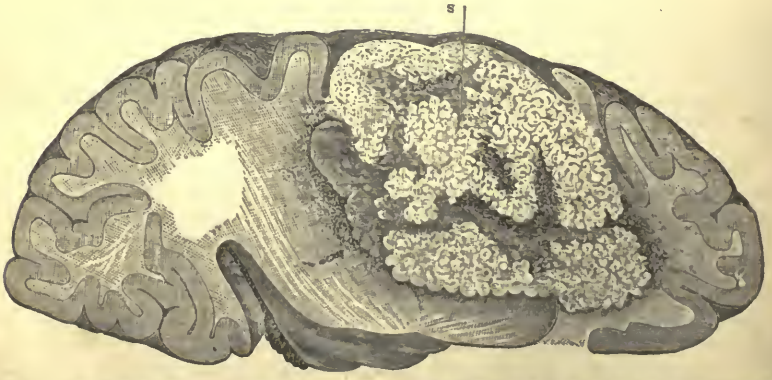

Fig 9.—Vertical section of left cerebral hemisphere. *s*, a glio-sarcoma of anterior lobe, extending backward into posterior lobe.

foot of the cerebral peduncle, which is chiefly composed of fibres from the antero-lateral columns of the spinal cord, is distributed, in great part, to the anterior lobes. Fritsch and Hitzig's experiments on the results of galvanic irritation have shown that, in dogs, the motor centres for the mus-

cles upon the opposite side of the body exist in the lateral portions of the frontal lobe. The centre for the muscles of the neck is situated most anteriorly; a little to the outside is found the centre for the flexors and rotators of the anterior limb; and to the inner side is the centre for the extensors and abductors; within and below is that for the movements of the posterior limbs, and, at the junction of the inferior and middle third of the anterior central convolution, is the centre for the facial muscles. According to the most recent researches, the centre for the ocular muscles is situated to the inside of the centre of those muscles which are innervated by the facial, and are grouped around the eye. These centres are connected with one another and with the cerebral ganglia.

Having determined the role which the anterior portions of the hemispheres play in voluntary movements (although these facts cannot be applied to man unreservedly on account of the imperfect decussation existing in animals), we must now refer to the part which the frontal and temporal regions of the brain, by their connections with the island of Reil, take in the central formation of language (*vide* pp. 76, 77); disorders of speech existed in the proportion of 19 to 100, in the tumors of the anterior lobes collected by Ladame. Later and more extended statistics have increased this proportion considerably.

Meynert and myself have observed two cases of aphasia in tumors of the anterior lobes, the principal symptoms of which I shall here describe.

1. Meynert's observation refers to a female idiot, aged 65 years, who was affected with paralysis of the right side of the face, of the tongue and lower limb, and was aphasic to such an extent that she was unable to repeat her name. At the autopsy Meynert found encephalitis, with softening of the walls of the left lower horn; at the point where this lower lobe penetrates into the substance of the brain, a hard vascular tumor was situated, of a reddish-white color, encapsulated in an encephalitic cicatrix; its volume was that of a hen's egg, and the posterior half was in a condition of cheesy degeneration. This tumor was wedged in between the insula and the inferior and external portion of the left lenticular nucleus. The island of Reil (which was in a condition of encephalitis, and the pia mater of which was adherent to the temporal lobe, operculum, and posterior part of the operculum)was pushed to the outside. The external capsule, claustrum, white substance of the insula, and the third segment of the lenticular nucleus, were in great part replaced by the tumor. The posterior portion of the lenticular nucleus, optic thalamus, internal capsule, and the periphery of the centre of Vieussiens, were swollen, diffluent, and œdematous (weight of brain, 1,378 grm.).

2. The case which I observed was that of a man, aged 41 years, who had suffered for several years from headache occurring in violent paroxysms, vertigo, intermittent muscular cramps, loss of memory, and apathy. The hitherto robust patient took little nourishment, and in the last months answered all questions with yes or no, after long reflection and with manifest effort. No other words could be pronounced by this patient. The pupils were moderately dilated, the tongue heavily coated, the pulse 72. Six days after entrance into the hospital, paralysis of the right half of the body supervened, with dilatation of the right pupil, and the pulse rose to 102; the heart sounds were normal.

The patient died two weeks later; at the autopsy a sarcoma as large as a hen's egg was found upon the frontal portion of the left operculum; upon the left prominence of the latter was a second tumor as large as a pea. The operculum was adherent to the insula by the inflamed pia mater; its parenchyma was softened, and several small, recent hæmorrhages were observed in its neighborhood. The right hemiplegia resulted in this case from the destruction of the fibres passing into the lenticular nucleus. The cerebral ganglia were unaffected.

Headache is one of the most frequent symptoms in tumors of the anterior lobes of the brain; it is sometimes diffused, and sometimes limited to the frontal region. Disorders of the psychical faculties are observed

in the majority of these tumors, and comprise all stages of intellectual dis turbance, from weakness of memory and judgment to hypochondria and dementia. These disorders may depend upon the pressure exercised by the tumors, upon softening and inflammation due to their presence, or upon the atrophy and discoloration of certain portions of the cortex of which we have previously spoken. In one situation, these various factors may involve the converging systems which preside over the association of ideas, and in an another position they may cause interruption of conduction in the intermediate portions of the radiating fibres which transmit the impressions of special sense, and, finally, lesions may exist in portions of the cerebral cortex which preside over the formation of ideas. In a patient of Broca (Gaz. des Hôp., 148, 1862), softening was found extending through the gray substance of the anterior lobes; in two other cases of melancholia and weak-mindedness, published by Duchek, abscesses were found extending to the cerebral cortex.

In a case published by Meschede (Virch. Arch., XXXV. Bd., 3 H., 1866), the patient, aged 30 years, suffered, since childhood, from epilepsy and afterwards from imbecility, kleptomania, and erotic propensities; finally from intercurrent attacks of furious delirium. At the autopsy, an osteoma, 1½ inches long, 1¼ inches wide, and nearly an inch thick, was found in the anterior and inferior portions of the left cerebral hemisphere (under the microscope, the osteomatous growth was found surrounded by soft myxomatous tissue). In addition, a split-shaped cavity, half as large as a pea, was found in the left Ammon's horn, communicating with the left lateral ventricle, and containing a tissue rich in vessels, which was regarded as an arrest of development (?).

Stewart published a case (Quart. Journ. of Calcutta Med. and Phys. Society, 1857) in which an osseous tumor situated in the left frontal sinus had given rise to headache, lasting several years, to hypochondria and somnolence, and finally to coma which continued several weeks.

The motor disturbances which are so frequently observed in tumors of the anterior lobes (23 times in 27 cases, Ladame) are sometimes of an irritative character (epileptiform attacks), and sometimes consist of paralyses of one-half the body, rarely of one side of the face. Generally, these symptoms are produced by tumors which penetrate deeply and provoke secondary irritation, œdematous swelling or softening of the cerebral ganglia and adjacent parts. In Mesnet's case, a hard, blackish tumor, as large as a billiard-ball, was found in the right anterior and middle lobes, and had given rise, during life, to a tendency to deviate towards the right in walking. Among the sensory disturbances, which are of very rare occurrence, we may mention neuralgias in the limbs (as in Andral's case), or anæsthesia (Bouillaud and Meissner's patients); in the first case, loss of sensibility was found upon the opposite side, and, in the second case, upon the side corresponding to the site of the tumor; but complications were present which evidently obscured the symptoms.

The special senses are only involved in a few instances. Here and there we find mention made of alterations of smell or taste, without any minute examination having been made. In five cases reported by Ladame, amblyopia (which, in one case, was alternate) or complete amaurosis was observed. If we examine the published cases more carefully, it will be found that in Plater's case, in which amblyopia attacked the left and right sides successively, a tumor as large as a hen's egg was present in the left cerebral hemisphere, and had flattened the optic nerves. The same event occurred perhaps in Jentzen's case, in which a tumor, occupying a similar position, had given rise to softening of the surrounding parts. In the three remaining cases, the anterior and inferior portions of the an-

terior lobes were affected by the tumors, which had also compressed the chiasm. In Eisenschnitz's case, a girl æt. 6 years, presented blindness, with considerable swelling of the left eye, violent frontal headache upon the same side, frequent vomiting, and later, loss of consciousness, slight convulsions, dilatation and paralysis of the right pupil. On autopsy, a glioma was found in the left retina, and a secondary growth of the same character, as large as a goose egg, existed upon the roof of the left orbital cavity; it was strongly adherent above to the dura mater, and within, to the sheath of the optic nerve. A considerable extravasation of blood had also occurred into the left lateral ventricle.

From the preceding statements we may accordingly give the following as the chief characteristic symptoms of tumors of the anterior lobes: diffused or frontal headache, symptoms of irritation or depression of the psychical faculties, convulsions, epileptiform attacks, hemiplegia, the frequency of disturbances of speech (generally assuming the characters of aphasia), the rarity of disorders of sensibility and of the special senses.

III. Tumors of the Middle Lobes.

In tumors of this region, headache usually occurs upon one-half of the head, on the side corresponding to the situation of the growth; it is rarely frontal. Motor disturbances are very frequent; in one-half the cases collected by Ladame, they existed under the form of hemiplegia; in several cases convulsions and epileptiform attacks occurred. In the majority of examples belonging to this category, the ganglia were also affected by the diseased process. In Cruveilhier's, Wegeler's, Green's, Vanroosbroeck's and Lebert's cases, the corpus striatum and optic thalamus were more or less seriously implicated.

Sensory disorders existed 10 times in Ladame's 27 cases; 4 times, anæsthesia occurred upon one-half the body on the side opposite to the tumor; in only two cases was the anæsthesia in the territory of the trigeminal nerve, once upon the same side as the tumor, and in the other case, upon the opposite side.

The cutaneous anæsthesia of the limbs was evidently due to lesions of the sensory fibres which pass out from the foot of the cerebral peduncle, and of which we have already spoken, while the trigeminal anæsthesia was caused by compression exercised in the course of the nerve. As symptoms of sensory irritation, Deliouse found, in one case, neuralgia of the ophthalmic branch with inflammation of the eye upon the same side.

Of the organs of special sense, the eye is most often affected (amblyopia or amaurosis), in consequence of compression of the optic nerves; audition is more rarely involved (thickening of the acoustic nerve in Abercrombie's case); strabismus is also sometimes observed. Intellectual disturbances, which usually appear as apathy or imbecility, are as frequent as in tumors of the anterior lobes; disorders of speech, when present, are readily understood from what has been already stated.

Thus, tumors of the middle lobes, like those of the anterior lobes, are also characterized by motor and intellectual disturbances. Nevertheless, the disorders of the special senses, and especially of sight, are more frequent in tumors of the middle lobes; a still more frequent symptom is cutaneous anæsthesia upon the side opposite to the tumor, affecting either one limb or half the body.

IV. Tumors of the Posterior Lobes.

The latest histological researches have shown that there is much less extensive union of the fibres of the corona radiata of the posterior lobe with the motor ganglia than in the anterior lobe. On the other hand, Gratiolet's investigations have shown that the cortex of the occipital lobe gives origin to the external sensory fibres of the foot of the cerebral peduncle, which correspond to the posterior spinal columns and to the expansions of the optic nerves. Hitzig and Ferrier have shown, in the course of their experiments upon electrical stimulation, that removal of the posterior lobes in animals produces no effect upon motion, a fact which is, however, not entirely applicable to man.

Among the pathognomonic signs of tumors of the posterior lobes, we may mention the appearance of psychical disorders, which are much more common than in growths of the anterior and middle lobes. The symptoms of motor irritation consist of convulsions and epilepsy; the symptoms of depression are manifested by incomplete hemiplegia or pareses limited to certain limbs. Cephalalgia is usually diffuse, more rarely limited to the posterior part of the head. Sensory disturbances have been observed in only a small number of cases, but careful examination will probably prove their greater frequency. In an old observation by Starkey, and a more recent one by Meschede, double amaurosis existed in addition to headache, vertigo, epileptiform convulsions, and anæsthesia of the limbs (Starkey). The vertigo which Immermann attributes, in tumors of the posterior cerebral fossæ, to real oscillations of the trunk, may be due to irritation of the organ presiding over the sensation of equilibrium, and which is situated in the temporal bone, *i. e.*, the semicircular canals.

When the tumors occupy several lobes at the same time, it goes without saying that the somewhat confused distinctions which we have established, completely disappear. Nevertheless, the striking symptoms which we have enumerated, such as disturbances of motion and sensation, of the psychical faculties and of the organs of special sense, preserve their importance in the majority of cases, and force us to suspect the existence of cerebral tumors.

V. Tumors of the Motor Ganglia of the Brain.

(Corpus Striatum and Lenticular Nucleus.)

The fibres of the foot of the cerebral peduncle, which have their central origin in the corpus striatum and lenticular nucleus, are connected with the centrifugal fibres of the pyramids, and since the ganglia receive fibres of the corona radiata proceeding from the cerebral cortex, they serve for the transmission of voluntary impulses to the anterior roots of the spinal cord. Nothnagel's experiments have shown that the lenticular nucleus in animals is chiefly a motor apparatus; and Ferrier, in his later experiments, has seen very marked pleurosthotonos develop upon the opposite side after electrical irritation of one of the corpora striata. The ganglia in question also contain the fibres of the cranial motor nerves. The surrounding medullary substance gives passage, as we have shown above, to the central apparatus for the transmission of sensibility towards the external and posterior regions of the optic thalamus, and to the por-

tions which unite these with the posterior and temporal regions of the brain.

The loss of the power of motion accompanying tumors of the cerebral ganglia is often preceded by symptoms of irritation (muscular spasms, tremor, and disturbances of co-ordination), which will claim our careful attention.

Duchek (Mediz. Jahrb., 1 H., 1865) observed at the onset, in a case of a tubercle as large as a hazel-nut, occupying the left corpus striatum, choreiform, inco-ordinated movements of the muscles of the face and limbs upon the right side. Lind observed tremor of the hands in a tumor as large as a hen's egg, which had taken the place of the left corpus striatum. The symptoms of motor irritation usually precede those of depression; alternation between the two sets of symptoms may continue as long as the conducting power is not entirely abolished; and, if this condition persists for a long time, we should rather suspect the presence of a tumor, developing slowly and pushing aside the surrounding parenchyma, than cerebral softening.

A musician, 26 years of age, suffered, in the spring of 1865, from periodical, violent pains, which extended from the vertex to the fourth cervical vertebra (the latter was very painful upon pressure). Later, his condition became complicated with headache, manifest loss of memory, frequent vomiting and temporary loss of consciousness. These symptoms were followed by convulsions, tremors and paresis of the left half of the body, and by frequent hiccough. Rude respiration was heard over both lungs. A diagnosis was made of probable tuberculous tumor of the brain, and this was confirmed on the autopsy. The tail of the right corpus striatum and the optic thalamus were occupied by a yellowish, dentated tumor, larger than a walnut, and extending into the third ventricle. The central portion was in a condition of cheesy degeneration. Small granulations, as large as a poppy-seed, were deposited around the chiasm in a mass of exudation; granulations as large as a millet-seed were found in the lungs.

An analogous case of a sarcomatous tumor occupying a portion of the lenticular nucleus, has been mentioned under tumors of the anterior lobes.

The motor disturbances chiefly consist in hemiplegia of the face and extremities upon the opposite side; epileptiform attacks are more rarely, and convulsions still more rarely observed.

In Lind's case, the tumor, which occupied the left corpus striatum, had also produced disturbances in the other hemisphere, and had given rise to paralysis of both lower limbs. Whenever the opportunity offers to observe cases of this nature, we should carefully examine the spinal cord in order to determine whether secondary degenerations have not developed in the antero-lateral columns.

In an observation recently published by Schueppel, a hæmorrhagic myxosarcoma (sarcoma with interstitial myxomatous tissue), as large as an apple, was situated in the corpus striatum, without giving rise to paralysis of the limbs. We must conclude, therefore, that if the fibres which transmit motor stimuli are not destroyed, but simply pushed aside, the power of motion will not be seriously affected. In the growths in question, the special senses remain intact. The disorders of sight which have been mentioned as occurring in some cases, are due to destruction of the chiasm or tubercula quadrigemina.

Intellectual disturbances are frequent, and are usually of a depressive character. Speech itself, in the sixteen cases collected by Ladame, was affected seven times; in three cases, slowness of speech existed; in two, there was difficulty in the articulation of words; and in two, a loss of speech. In accordance with the classification of the disorders of speech made in

preceding chapters, I think that, in this category of tumors, they must be chiefly regarded as motor disturbances of the tongue, or as slowness of speech resulting from weakness of intellect. As we have stated above, the fibres of the corpus striatum and lenticular nucleus are contained in the central portions of the pyramids and in the foot of the cerebral peduncle. They give rise to fibres which enter the nuclei of the facial and hypoglossal nerves, and which decussate across the median line of the trunk of the brain, as Meynert has directly proven. Aphasic disturbances of speech must be due to lesions of those fibres which enter the lenticular nucleus from the cortex of the insula. From the preceding statements, we can also explain how central lesions may diminish or abolish the influence of ideas, and even act upon the roots of certain cranial motor nerves.

We may summarize the symptoms of tumors of the cerebral motor ganglia as follows: hemiplegia (very often preceded by symptoms of motor excitation); convulsions; disorders of speech, especially in the articulation of words; facial paresis; disturbances of intelligence and integrity of the organs of special sense, the functions of which are very rarely involved.

VI. Tumors of the Optic Thalamus and Tubercula Quadrigemina.

According to Meynert, the two ganglia of the tegmentum of the cerebral peduncle give origin to the posterior spinal columns. Reflex stimuli are transmitted through the medium of the tegmentum to the anterior roots, and, in addition, the optic thalamus and tubercula quadrigemina are connected with the optic tract and corpora geniculata.

In diseases of the optic thalamus (which merely gives passage to the optic nerve), sight is not altered and special motor disturbances rarely occur. According to Schiff (Lehrb. d. Physiol. d. Menschen, p. 343–47), section of the posterior portion of the optic thalamus upon the left side in animals causes a deviation of the head to the right, while the legs are directed to the left, the left anterior leg in adduction, the right in abduction; in walking forwards, the animal describes a circle towards the right. Schiff attributes these anomalies of position and movement to paralysis of the abductors and corresponding adductors. According to Ferrier, electrical irritation of the optic thalamus exerts no influence upon motion.

In addition to serving as origin to the tegmentum of the cerebral peduncle, the tubercula quadrigemina also give rise (according to Gratiolet, in the superior pair) to the roots of the optic nerve, the fibres of which, after traversing the internal corpus geniculatum, radiate backwards into the cortex of the occipital lobe, while the external geniculate body, according to Meynert, furnishes the external root of the optic irradiation, and is connected with the corona radiata and with the base of the posterior tubercle of the optic thalamus. The optic nerve is therefore connected with the cerebral cortex, directly through the geniculate bodies, and indirectly by the two reflex ganglia, that is to say, by the optic thalamus and corpora quadrigemina. The reaction of the pupil to the light depends, therefore, upon the continuity of the connections between the retina and the tubercula quadrigemina through the medium of the optic nerve; these connections may be continuous, by reflex means, with the oculo-motor nerve and its ciliary branches. Destruction of the tubercula quadrigemina in animals causes, according to Flourens, blindness of the opposite eye; abolition of sight in one eye gives rise, according to Magen-

die, to atrophy of the tubercula quadrigemina upon the opposite side. According to the more recent researches of Adamueck, the motor innervation common to both eyes, proceeds from the anterior tubercula quadrigemina. The right tubercle governs the movements of both eyes to the left, the left governs the movements to the right. After prolonged irritation, the head is also turned to the same side as the eyes. If the two tubercula are separated by a deep incision, motion only follows upon the side which is irritated. Irritation of the free surface of the tubercula produces movement of both eyes to the opposite side; the movement is more marked, at the same time, upwards and inwards or downwards and outwards. The old experiments of Flourens upon blindness and paralysis of the iris, produced upon one side by the destruction of one of the tubercula quadrigemina, have been recently corrected by Knoll, who has shown that these phenomena are due, not to destruction of the tubercle, but to lesion of the optic tract. According to Knoll, irritation of one of the tubercles is followed by dilatation of both pupils, especially of the pupil on the side which has been irritated. According to the recent experiments of Ferrier, electrical irritation of the tubercula quadrigemina causes strong opisthotonos, trismus and dilatation of the pupils; the limbs are stiff and extended. These phenomena are especially well marked upon the side opposite to the irritation.

Tumors of the optic thalamus have, until recently, been regarded as causes of hemiplegia, but the motor functions of the thalamus are now strongly questioned. An observation, recently made by Meynert, is opposed to the views hitherto adopted. The patient, a boy four years of age, suffered from headache, vertigo, paralysis of the oculo-motor and trochlearis nerves and crossed paresis with tremor of the limbs on the left side. For a long time the patient had the habit (although not continuously) of inclining the head to the left, the left arm being flexed and the right arm extended. A diagnosis was made of a tubercular tumor in the right peduncle extending across the tegmentum into the optic thalamus. Upon autopsy, a tubercle, larger than a pigeon's egg, was found at the base of the brain in the anterior perforated space. The tumor had compressed the foot of the cerebral peduncle, the optic tract, and the wall of the third ventricle upon the right side and also the optic thalamus, and had thrust its posterior tubercle to the outside.

The abnormal attitudes of the patient, which were observed in Schiff's experiments and aided the diagnosis of a disease of the optic thalamus, are attributed by Meynert, not to paralysis, but to a perversion of the muscular sense. In fact, when the attention of the patient is diverted, the peculiar position of the arms disappears. It appears to me simpler to regard these pathological movements as caused by irritation of the reflex centres of flexion and extension of the two arms, and which lie side by side, in the optic thalamus.

Tumors of the tubercula quadrigemina are of much greater rarity. In Ladame's work only two cases are mentioned, which occurred in children, aged respectively fifteen months and three years, and which were due to isolated tuberculization of the tubercula quadrigemina. Henoch's case was characterized by right hemiplegia, paralysis of the right facial nerve in its palpebral and labial branches, internal strabismus of the right eye, contraction of the right pupil, and intermittent spasms in the healthy as well as the paralyzed limbs; sight was intact. At the autopsy, in addition to tuberculosis of the left lung, bronchial and mesenteric glands and spleen, granulations were found in the fissure of Sylvius and in the choroid plex-

uses of the ventricles, and a tubercle, half as large as a bean, in the left posterior tuberculum quadrigeminum (Berl. klin. Wschr., 1864, 13). In Steffen's case (eod. loc., 20, 1864), headache, double ptosis, eclamptic attacks (occurring throughout the body, but of short duration), and dulness of the sensorial faculties (without disturbances of vision) existed during life; the symptoms of pulmonary tuberculosis were also present. The autopsy showed the existence of tuberculosis of the lungs, bronchial and mesenteric glands; the tubercula quadrigemina were transformed into a rounded, fissured, yellowish, tuberculous mass.

To these two cases occurring in children, I am able to add a case of medullary tumor of the tubercula quadrigemina, which I had the opportunity of observing in an adult at the Vienna General Hospital.

A weaver, aged 30 years, complained, upon admission to the hospital, of violent headache, which had appeared a year previously, but had been continuous only for the last two months, of weakness of memory, dimness of vision in both eyes, and a feeling of extreme lassitude. The patient presented a cachectic appearance, answered questions with difficulty, the gaze was fixed, the pupils strongly dilated and reacted slowly. He stated that surrounding objects appeared dim. The gait was feeble, and walking was soon followed by a feeling of fatigue.

The force of the grip was also very much diminished; the additional symptoms were somnolence, intermittent convulsions of the limbs, and cough without any appreciable objective cause. The heart acted normally, the pulse being 60; there was slight constipation at the end of several days; the patient fell into a condition of stupor, from which he could only be aroused with continually increasing difficulty. He died seven weeks after admission, in a condition of general paralysis.

At the autopsy, the meninges were found infiltrated with serum, the cerebral convolutions were flattened, the fornix was strongly arched upward, and the ventricles distended into sacculated cavities. A medullary tumor, about as large as a nut, was found upon the corpus quadrigeminum, extending to the middle commissure; it separated the optic thalami and stretched into the fourth ventricle by a small, conical prolongation.

If we attempt to gather some characteristic symptoms of these tumors from the extremely few cases which we have recorded, we will be aided by the fact that pathology agrees (although in an imperfect manner) with the anatomical and physiological researches to which we have referred above. The symptoms of motor irritation, the later disturbances in the paths which transmit motor impressions, the dilatation of the pupil on the opposite side (Henoch's case), and the paralyses caused by pressure of the tumor upon the motor oculi nerve, all these symptoms are readily explained by the experiments which have been referred to. But we do not attach any especial importance to the symptoms of paralysis of the third pair of nerves, even when they have a progressive course (contrary to the opinion of Henoch and Steffen), for we shall see hereafter that analogous symptoms occur in tumors of the peduncle.

As regards the blindness produced in animals by extirpation of the tubercula quadrigemina, nothing similar was observed in the first two cases which we have cited. Despite the extensive changes in the tubercula, vision was unaffected. But if we consider that in these two cases the fibres of the optic nerve were not entirely destroyed, but merely pushed aside by the tumor, and that, according to Knoll, it is not the destruction of the tubercula quadrigemina, but lesions of the optic tract, which are most important; if we, furthermore, reflect that, even after the destruction of the tubercula, communication with the cortex of the brain is still possible through the geniculate body and that visual perception is thus rendered possible—we can satisfactorily explain the slight alterations in the sense of sight.

In confirmation of the blindness observed in physiological experiments, we may refer to the dimness of vision present in my case and noticed by the patient himself. In this connection, I may also quote Friedreich's case, in which the tubercula were compressed by a sarcoma as large as a hen's egg, occupying the right optic thalamus. During life the patient suffered from paresis of the extremities on the left side, from strabismus of the left eye, lagophthalmos, amblyopia, and alternating dilatation and contraction of the pupils. The left side of the face was paralyzed and sometimes subjected to spasms; partial spasms were also present in Henoch's patient.

The diagnosis of a disease of the tubercula qaudrigemina is far from being free from obscurity. All that can be said is, that the most striking symptoms consist of convulsive spasms, paralyses of the limbs and of the motor oculi nerves, changes in the condition of the pupils, paresis of the face and frequent disorders of vision.

VII.—Tumors of the Middle Cerebral Fossa, and of the Region of the Gasserian Ganglion.

Passing now to the base of the brain, we shall carefully study the tumors of the middle cerebral fossa, which are accessible to diagnosis at some points. Tumors thus situated, may, according to their size and the manner in which they develop, extend to the Gasserian ganglia and the fibres which pass out from them, to the roots of the olfactory nerves, pituitary gland, chiasm, to the ocular, facial, and acoustic nerves, and even to the pons varolii and cerebellar peduncles. They may also send prolongations into the foramen rotundum, foramen ovale, and internal auditory canal. The symptomatology may thus become very complex and the diagnosis may present great difficulties.

Nevertheless this class of tumors is characterized by certain symptoms, which often permit an exact localization of the lesion during life. These symptoms consist in affections of the trigeminal and facial nerves and in nutritive disturbances of the eye upon the affected side. The affection of the trigeminal is manifested, at first, by symptoms of irritation under the form of prosopalgia, which are followed, after destruction of the fibres, by anæsthesia limited to one-half the face (sometimes anæsthesia dolorosa). The paralysis of the trigeminus usually extends to the external and internal branches, and then produces, in addition to paralysis of the masseter muscle, insensibility of the skin and mucous membranes, and blunting of the olfactory and gustatory senses in the corresponding nares and half of the tongue. In consequence of the increase of the lesions of the trigeminal nerve, destructive inflammation of the eye develops. Inflammatory redness of the conjunctiva and iris, opacity, purulent infiltration and central ulceration of the cornea and perforation and atrophy of the eye occur in the same manner as after experimental section of the trigeminus. We shall enter, in detail, upon the causes and nature of this ophthalmia, in the section upon diseases of the cranial nerves.

In a case reported by Beveridge (Med. Times and Gaz., 921, 1868), complete loss of sensibility occurred upon the left half of the face, from the eyebrow to the chin, and involving the conjunctiva, cornea, left nares, and the left half of the tongue; audition was impaired upon the same side. Little by little, sight began to grow feeble upon the left side, and finally hypopyon developed and perforation of the eye ensued; the

left side emaciated considerably. The patient died after paresis of this side and paralysis of the left muscles of mastication. At the autopsy, a hard, conical tumor, an inch in length, was found between the pons varolii and the petrous portion of the temporal bone. The trigeminal nerve was more compact and harder than normal, and, after passing under the tentorium of the cerebellum, entered the ganglion of Gasser, which was considerably hypertrophied, mixed with fibrous tissue and covered by the strongly adherent dura mater. This mass not only involved the Gasserian ganglion, but also the ophthalmic branch, the origin of the superior maxillary nerve and a portion of the inferior.

In a more recent case, described by Borland (Bost. Med. Journ., Vol. VII., 1872), the symptoms observed during life were ulcerative lesions of the right eye, ptosis, right facial paralysis with salivation, followed by enfeebled hearing and paralysis of the left leg. At the autopsy, a glioma, one and a half inches in diameter, was found upon the left side under the tentorium cerebelli, with sclerosis of the right sensory root of the trigeminal nerve, infiltration of the right Gasserian ganglion with pigment cells and amyloid bodies, and compression of the pons, cerebellar peduncles, and medulla oblongata.

In calling attention to the characteristic electrical reactions of the muscles of the face in paralyses due to tumors at the base of the brain (Klin. Beitr. z. Sympt. u. Diag. d. Tumor. d. Hirnb. u. d. Pedunc., Med. Jahrb., XXI. Bd., 1870), I have demonstrated that the faradic excitability of the muscles of the face and of the branches of the facial nerve is abolished and that the galvano-muscular reaction is increased (in comparison with the healthy side), while the isolated branches of the facial nerve present a diminution of galvanic excitability. The following notes refer to a case of this kind, which is remarkable for the intra-cranial though extra-cerebral character of the lesions:

A porter, 20 years old, was received into the second medical division of the Vienna General Hospital. The patient states that he has suffered since Nov., 1868, from vertigo, frontal headache, diplopia, and stiffness of the right side of the face. Upon admission (May, 1869), I discovered complete paralysis of the right half of the face (with lagophthalmos and salivation), paralysis of all the ocular muscles upon the right side, with the exception of the superior rectus; in addition, softening of the cornea (keratomalacia) and anæsthesia of the trigeminal nerve (externally, following an oblique line running from the labial commissure to the zygomatic arch and occiput; internally, upon the mucous membrane of the cheek and buccal cavity on the right side). There was also complete abolition of the faradic excitability of the muscles of the face and branches of the facial nerve upon the right side, with increase of the muscular reaction to the continuous current (compared to the healthy side). The subclavicular and lateral cervical glands, extending to the parotid, were infiltrated and firm. The left leg became weak after any prolonged exercise, but both hands were equal in strength. The anæsthetic cheek was, at times, the seat of violent pains; during one of these attacks I noticed marked redness around the right ear; the temperature in the right external auditory canal was 36.2° C. and in the left only 35° C. The ensemble of symptoms, the characteristic effects of electricity upon the paralyzed side of the face, together with the enlargement of the cervical glands, led me to diagnose a carcinoma (syphilis, scrofula, and tuberculosis having been previously excluded) situated at the base of the brain around the Gasserian ganglion. In the absence of motor disturbances in the limbs, I concluded that the tumor did not affect any of the organs of motor conduction (pons and cerebral peduncles). The patient died in a condition of stupor towards the end of the month. At the autopsy, a tumor, larger than a five-franc piece, was found at the internal periphery of the middle cerebral fossa upon the right side, and from which a medullary juice flowed upon section. It passed through the cavernous sinus to the orbital walls, and sent prolongations through the foramen ovale and foramen rotundum into the Fallopian aqueduct. The trigeminus (except a small portion which was preserved in the Gasserian ganglion), the motor oculi communis, patheticus, motor oculi externus and petrosus superficialis major nerves, were matted together by the tumor (under the microscope it was found to be composed of a stroma of connective tissue and a few cellular elements). The cervical glands and a small growth in the liver presented similar changes.

We may summarize the diagnostic signs of tumors situated in the middle cerebral fossa and around the Gasserian ganglion as follows: chronic head-symptoms, facial neuralgia, often changing to anæsthesia and limited to one-half of the face; simultaneous paralysis of adjacent motor nerves or of the anterior sensory nerves; peculiar electrical reactions upon the paralyzed side of the face; finally, inflammation and suppuration of the eye-ball. The appearance of hemiplegia of motion and sensation in the limbs with crossed paralysis of the cranial nerves, disorders in the articulation of speech, dysphagia, and partial rotation of the head or trunk to one side, show that the tumor has extended to the pons and, varying with the symptoms, to the cerebellar peduncle.

VIII. Tumors of the Pituitary Region.

The tumors of the pineal gland usually assume considerable dimensions and, by their size, growth, and lateral development, may compromise various organs at the base of the brain. The anterior perforated space with the olfactory region, the chiasm, roots of the optic nerves, mamillary bodies, posterior perforated space, cerebral peduncles and even the pons and adjacent portions of the cerebellum, may be compressed or destroyed. The cavernous sinus, sphenoidal fissure, the nerves passing through it, and the ventricles, are very often affected by the pressure exercised by the tumor or by its prolongations. Danger may also arise from softening of the parts adjacent to the tumor, and this may even extend (as in Biermer's case) to the cerebral ganglia and the centrum ovale of Vieussens. The effects of tumors of the pituitary gland may extend to the fourth ventricle and give rise to the production of diabetes.

Among the initial symptoms of these growths are the periodical headaches, which especially occupy the frontal and temporal regions and may even extend to the suborbital region and to one or the other eyeball. Disorders of vision also occur among the severe initial symptoms, and appear under the form of amblyopia or amaurosis either of one eye or more frequently of both (atrophy of the optic nerves). Sensory irritative symptoms are infrequent and temporary. Motor symptoms of irritation and depression are isolated (convulsions, contractures, hemiplegia, or paraplegia), and are not characteristic of this group of tumors. Disorders of the special senses are manifested, and must be often regarded as symptoms of hyperæmic irritation. These symptoms are roaring in the ears, flashes of light before the eyes, and hallucinations of sight. The enfeeblement of the sense of smell which sometimes occurs may be due to lesions of certain parts adjoining the tumor; in fact, the olfactory region (with the decussating fibres of the roots of the olfactory lobe) is situated above the anterior perforated space and beyond the Ammon's horn, so that certain portions of the anterior commissure originate from roots situated in the olfactory region. Among the psychical disturbances we may mention weakness of memory and apathy; there is no noteworthy disorder on the part of speech. Tumors of the pituitary gland may be mistaken, in some cases, for orbital growths which also cause amaurosis and exophthalmos. In intra-cranial tumors, according to Michel, the amaurosis precedes the exophthalmos, and the reverse holds good in orbital tumors. In the latter, also, which are most frequently situated upon the sides of the eyeball, the exophthalmos is generally accompanied by strabismus.

I shall now report the history of a case which appears to me to be

of great interest, both because the disease occurred in an intelligent *confrère*, and especially because it was attended by a complication not hitherto described, viz., intense diabetes mellitus.

At the end of June, 1859, before the close of the Italian campaign, Dr. W., a military surgeon, aged 34 years, was forced to return to Vienna on account of headache and continually increasing weakness of sight. The visual disturbance consisted in a progressive narrowing of the field of vision, starting from the periphery, at first in the right eye and then in the left, so that finally only those objects could be seen which were placed directly in front of the pupils. In the following years the sight became entirely lost. Ophthalmoscopic examination by Prof. Jaeger showed bluish discoloration of the optic discs. In May, 1861, the patient complained of an increase of weakness in the legs, though he was able to walk down a flight of stairs. In the following months the power of motion rapidly diminished and the patient was thereby confined to his room. Polyphagia and polyuria then developed as new and unexpected symptoms, and progressive emaciation occurred, despite a very excellent appetite. The urine was clear and pale; the daily quantity measured three to four kilogrammes; the specific gravity varied from 1038–1040. Trommer's and Boettcher's (nitrate of bismuth) tests gave an abundant precipitate. The sensorium was unaffected. In October, 1861, the patient was unable to leave his bed; the lower limbs were paretic, the upper ones unaffected. Towards the close of the year very obstinate ciliary neuralgias were added to the previously mentioned symptoms. Opiates and hypodermic injections of morphine only produced slight relief; inhalations of chloroform were more successful. When the pains were most severe, the patient used from 60–90 grammes of chloroform daily. The sad condition of the patient was rendered still more gloomy during the first few months of 1862, by an advanced degree of emaciation. Three days before death (beginning of May) the pulse became accelerated, and changes occurred in the quantity and color of the urine; during the last days of life no trace of sugar could be detected. At the autopsy, a hard tumor (sarcoma), larger than a walnut, was found in the region of the hypophysis; the sella turcica was eroded; the ephippium had disappeared, with the exception of a very small portion which was adherent to the posterior clinoid eminence; a prolongation of the tumor had entered the sphenoidal fissure. Advanced fatty degeneration had occurred in the optic nerves; nothing abnormal could be detected in the fourth ventricle with the naked eye; no microscopical examination could be made. The kidneys and liver were very much congested.

Let us analyze the characteristic symptoms observed in this case. The frontal headache, the amblyopia and amaurosis progressing from the periphery to the centre, at first upon one side, then on both; the ciliary neuralgia, caused by the entrance of the morbid tissue into the sphenoidal fissure; the symptoms of progressive paralysis of the lower limbs (due, perhaps, in great part, to the diabetic muscular weakness); all these symptoms agree entirely with the clinical history presented by other observers.

But the extreme glycosuria constitutes a new and interesting complication. In the absence of microscopical examinations of the floor of the fourth ventricle, we are unable to form any conclusions with regard to the lesions which produced this symptom. According to the experiments of Claude Bernard, lesions of the floor of the fourth ventricle will cause the appearance of sugar in the urine (from lesion of the centre of the vascular nerves of the kidneys, according to Schiff). Tumors of this region may also produce glycosuria. If we consider, in addition, that the tuber cinereum is found in the immediate neighborhood and in advance of the pituitary gland, and that the infundibulum merely represents a prolongation of the gray substance of the third ventricle and, by contiguity, of the fourth ventricle into the medulla oblongata, we may admit that tumors of the pituitary region, by an increase of pressure, may give rise to paralysis of the medullary centres of hepatic innervation and to consequent hyperæmia

of the liver resulting in diabetes. An observation made by Pavy, who found sugar appear in the urine after compression or lesion of the vertebral plexus, also favors this interpretation.

IX.—Tumors of the Cerebral Peduncles.

In order to comprehend the symptoms due to the formation of tumors in the cerebral peduncles, we must take into consideration the anatomical relations and physiological signification of the parts which compose them. The considerable development of the cerebral hemispheres in man accords with the volume of the foot of the cerebral peduncle which takes its origin in the hemispheres; extirpation of one hemisphere causes, according to Gudden, atrophy of the cerebral peduncle on the same side. A portion of the fibres which start from the cerebral peduncle is directed, according to Broadbent, directly towards the cerebral cortex; the largest portion of the fibres of the pes pedunculi terminates centrally, as we have already shown, in the corpus striatum and lenticular nucleus. The motor fibres which traverse the lenticular nucleus and which radiate towards the anterior lobes, spring from the internal portion of the pes pedunculi and are continued through the pons varolii into the antero-lateral columns. The sensory fibres contained in the external portion of the pes pedunculi, and which are directed towards the occipital lobe, are continuous with the posterior spinal columns. Decussating fibres, starting from the foot of the cerebral peduncles, also enter the motor nuclei of the medulla oblongata. Experimental section of one of the cerebral peduncles produces movements of "manége," the convexity of the circle which the animal describes in walking, being upon the side of the section. The movements of "manége" (rolling and turning) are explained by Schiff in the following manner: the paralysis of the fibres, which act in an harmonious manner in rotatory movements, forces the animals to direct their movements towards the opposite side and to describe a circle by the summation of the contractions which affect unequally the anterior and posterior portions of the body. Incision of the peduncle, immediately in front of the pons, causes the animals to fall upon the opposite side, motion being preserved in the limbs of the side operated upon. We have already discussed lesions of the posterior third of the cerebral peduncle under diseases of the optic thalamus. The recent experiments of Afanasieff (Wien. med. Jahrb., 9–12, 1870) upon section of the cerebral peduncle, have resulted in the production of paralysis of the motor oculi of the same side, incomplete paralysis of the face and limbs upon the opposite side, and also in a diminution of sensibility.

The latest experimental investigations have also demonstrated the influence of the cerebral peduncle upon the movements of the bladder and upon the vascular nerves. Budge first demonstrated (Henle und Pfeufer's Zschr., 21. Bd., p. 14) and has since confirmed by new researches (Pflueger's Arch. II. Bd., 1870, p. 511–17), that irritation of the peduncle is followed by contractions of the bladder. As these movements can be obtained after removal of the posterior column, but not after section of the anterior column below the irritated spot, he concluded that the motor nerve fibres of the muscular tissue of the bladder passed from the peduncle through the restiform bodies, medulla oblongata, and anterior columns of the cord. The more recent researches of Afanasjeff have also shown that section of the peduncle influences the functions of the bladder by

increasing the obstructions to the flow of urine and by abolishing the influence of the will upon micturition. Budge has likewise proven (Centralb. f. d. med. Wiss., 35, 1854) that the central excitation of the vaso-motor nerves proceeds from the peduncle, and is transmitted through the anterior columns and roots to the communicating branches and to the sympathetic nerve. Irritation of the cerebral peduncle produces contraction of all the arteries in the body. After section of the peduncles, Afanasieff observed contraction of the arteries, at first as a symptom of irritation, and then followed by arterial dilatation as a phenomenon of relaxation. The contraction of the arteries, in animals, continues from ten to fifteen days, and is accompanied by a reduction of temperature (1.5°–2° C. in the rectum).

The symptoms obtained in experiments on animals present great similarity with the morbid signs observed in man in peduncular tumors, as will be seen by the following notes of a case under my own observation (published in Med. Jahrb., XIX. Bd. 1870):

A peasant woman, 39 years of age, states that during the last two years she has suffered from headache, vertigo, weakness of sight, and paralysis of the limbs upon the right side. Careful examination shows the existence of very marked ptosis of the left lid. On the right side the respiratory muscles of the face are paralyzed (with normal galvano-faradic excitability), and hemiplegia of the limbs is noticed, with considerable diminution of tactile sensibility and sensibility to pain upon the right halves of the face and body (upon the upper limb the helices of an induction apparatus must be brought together to 20 or 24 millimetres in order to obtain contractions and sensations as well marked as on the left side). Speech is stuttering and the movements of the tongue are markedly embarrassed. When the tongue is protruded, it deviates to the right. With the ophthalmoscope an old neuro-retinitis is observed in the right eye and the acute stage in the left.

The patient complains of distressing vertigo, which increases rapidly on walking or when she is tired. This is also very marked when the patient sits up in bed, so that she is forced to resume the horizontal position. She can only lie upon the left side for a few minutes at a time, on account of the violence of the vertigo. During the following weeks the patient who was four months advanced in pregnancy, experienced improvement in speech, the other paralytic symptoms remaining unchanged. On two occasions she had spasms of the extensors of the upper and lower limbs (without loss of consciousness), lasting a few minutes. She also had frequent desire to urinate, especially at night, a symptom which the patient had not experienced in previous pregnancies. She aborted a dead, macerated fœtus, about six months old; the abortion was followed by fever and albuminuria, with symptoms of pneumonia on the right side. Towards the close of life the paralysis involved the motor oculi nerve of the hitherto unaffected side, and we were led to make a diagnosis of a peduncular tumor stretching from one side to the other.

Autopsy: A tumor, larger than a bean, was found between the cerebral peduncles, below the bifurcation of the basilar artery and the hypophysis, the growth being surrounded by a zone of serous softening in the right peduncle. A minute examination by Meynert showed the presence of a cyst, as large as a bean, in the internal portion of the left cerebral peduncle, which had also destroyed the roots of the left oculo-motor nerve as they traverse this point. The innermost portion of the right cerebral peduncle and the adjacent portion of the tegmentum were softened and congested. A tumor was situated upon the anterior portion of the posterior perforated space. The growth was larger than a bean, compressed the right oculo-motor nerve, and took the place of the mamillary bodies and posterior portion of the tuber cinereum. Anteriorly it was composed of a hard white substance (fusiform cells and numerous bundles of intertwining connective tissue) and, posteriorly, of a soft, vascular tissue (small nucleated cells, recalling the structure of the ependyma, or large, epithelial cells containing proliferating elements). This tumor may be classified among Virchow's glio-sarcomata.

From the succession of symptoms, it appears that the cyst in the left cerebral peduncle gave rise to the paralysis of the left oculo-motor, to the

right hemiplegia, and to the partial paralyses of the face and tongue. The tumor on the right side, which complicated the symptoms during the last periods of life, produced the paralysis of the right oculo-motor nerve which occurred a little before death; the amblyopia was due to the compression of the chiasm.

If we consider the symptoms of peduncular tumors separately, we will find that most patients complain of headache and vertigo, the latter being perhaps caused by diplopia.

Motor disturbances have occurred in all the cases hitherto observed. At the beginning of the affection these symptoms are of an irritative nature and later, when the paths for the transmission of motor impulses become involved, paresis or complete paralysis of the limbs occurs upon the opposite side. When the lesion involves the fibres of origin of the facial nerve (which leave the pons in an ascending direction and decussate, passing only in part through the centre of the pes pedunculi), partial facial paralysis will occur upon the side opposite to the lesion. On account of the intra-cerebral character of the facial paralysis, electrical exploration reveals normal or only slightly changed reaction to the faradic and galvanic currents. The oculo-motor paralysis, according to the united testimony of most authors, occurs upon the same side as the tumor. In small tumors of the peduncle, paralysis of the third pair may be wanting, as in Andral's case, in which a cyst, as large as a pea, was found in the centre of the substance of the cerebral peduncle and therefore at a distance from the origin of the oculo-motor nerve and from the edge of the peduncle. According to Herrmann Weber, the third pair is only involved when the internal and inferior layers of the nervous tissue are affected near the origin of the nerve. In the absence of symptoms of paralysis on the part of the nerves supplying the ocular muscles, it is impossible to differentiate the affection from a tumor of the optic thalamus. When the paralysis of the third pair exists on the same side as the hemiplegia, we must, according to Brown-Séquard, admit the existence of multiple morbid foci.

If the tumor finally extends across to the other side, paralysis of the oculo-motor nerve on that side will be added to that already existing. Very characteristic examples have been observed by Ruehle, Weber, Spanton (Med. Times and Gaz., May, 1863) and by myself. We very rarely find, in man, the manége movements observed in animals after experimental destruction of one of the cerebral peduncles. We may consider, however, as the analogue of these movements, the permanent rotation of the head to the side opposite to the lesion, observed in Stiebel's patient, and the tendency noticed in Paget's patient to fall forwards upon the head. In a case published by J. Hoffmann (Diss., Breslau, 1860), a tubercle in the peduncle, as large as a cherry-pit, had produced forced movements upon the paralyzed side of the body, in addition to hemiplegia, paralysis of the oculo-motor, and atrophy of the optic nerve upon the corresponding side.

Sensory disturbances are also often observed in tumors of the cerebral peduncle. The irritative symptoms consist of formication and neuralgic pains in the limbs, the depression symptoms, of anæsthesia. The sensory paralysis usually affects the limbs at the same time as the motor paralysis (as in Weber's and Spanton's patients). The sensory disturbances generally recover more readily, as Weber has shown by means of Sieveking's æsthesiometer (Med.-Chir. Trans., XLVI. Bd., p. 121, 1863).

The disturbances of sensation appear upon the side of the body oppo

site to the tumor. They usually occur upon opposite halves of the face and body and are due to an anatomical lesion of those fibres, which, after leaving the posterior spinal columns, pass into the external portion of the pes pedunculi and ascend into the medullary substance situated behind the lenticular nucleus.

With regard to vaso-motor disorders, Weber has observed in one case (extravasation into the inferior and internal portions of the left peduncle) an elevation of temperature upon the right (paralyzed) side of the body. In a case published by Fleischmann (W. med. Wschr., 6–9, 1871) of softening of the left cerebral peduncle from a tuberculous nodule in the left optic thalamus in a boy two years of age, right hemiplegia of the face and limbs occurred in addition to paralysis of the left oculo-motor nerve and abnormal quotidian elevations of temperature (1°–1.2° C. in 12 hours).

With regard to the differential diagnosis, we can merely state that certain diseases with analogous symptoms may be easily mistaken for peduncular tumors. Among these are circumscribed meningitic processes at the base of the brain, of which we have previously spoken at some length. In such cases the successive affection of the oculo-motor and facial nerves on both sides, the appearance of paralyses of the patheticus or external motor oculi, and the diminution or abolition of the electro-muscular contractility, will aid in forming a diagnosis. Certain forms of ataxia, in which ptosis and ocular paralyses are combined with cephalalgia and, in the beginning, with weakness of a lower or even an upper limb on one side of the body may, for some time, be mistaken for a cerebral tumor. In these doubtful cases the appearance of rachialgia, sciatica, brachialgia, intercostal neuralgia, the abnormal increase of the galvanic excitability of the nerves, the pathological modifications of the law of contraction, in addition to the fatigue occurring so readily in the upright position, the genital excitation, the condition of the sphincters—all these symptoms, when carefully observed, will enable us to form a correct diagnosis. We may, therefore, summarize the characteristic symptoms of tumors of the cerebral peduncles as follows : cephalalgia, vertigo, alternate hemiplegia with sensory disturbances, paralysis of the oculo-motor upon the same side as the tumor, the frequent tendency of this nerve to be affected also upon the opposite side, the less complete paralysis of the opposite side of the face, frequent neuro-retinitis, difficulty of micturition, abnormal temperature and absence of intellectual disturbances.

X.—Tumors of the Pons Varolii.

The pons varolii, the size of which depends upon the development of the pes pedunculi, attains in man a more considerable thickness and volume than in any other animal. Among the fibres composing the pons, we find, anteriorly, the longitudinal motor fibres which come from the spinal cord and pass upwards (continuation of the pyramids). Apart from these motor fibres, the pons contains, in its posterior part (as Clarke first demonstrated), sensory fibres which start from the external portions of the pes pedunculi and decussate in the posterior column.

Perpendicular sections show, according to Meynert, that these longitudinal fasciculi form the anterior and posterior halves of a shell enveloping the nucleus, which gives rise to the convex shape of the pons, that is to say, the deep transverse fibres. The nerves which pass towards the brain, after their fibres have for the most part decussated lower down in the pons, also pass through the pons varolii. According to Schiff, many of

the vaso-motor nerves reach the cerebral peduncle and optic thalamus through the pons. To their paralysis and its secondary consequences is due the death of the animals in section of the pons, despite the disappearance of all the other disorders which were due to the lesion.

Transverse section across the longitudinal fibres of the pons (in the most anterior portions, in front of the origin of the trigeminal nerve) produces, according to Schiff, deviation of the anterior limbs (as in section of a cerebral peduncle) with extreme flexion of the body in a horizontal plane towards the opposite side and very imperfect movement of the posterior limb (on the other side). Rotation in a very small circle (not the manége movements) develops in consequence of this paralysis. Both motor and sensory disturbances are produced in animals on account of the incomplete decussation of the fibres in the medulla oblongata, a fact which is at variance with that observed in man. According to Brown-Séquard (Lancet, I., 1871), excision or lesion of the pons, as well as of the cerebral or cerebellar peduncles, is followed by ecchymoses into the lungs and bronchi or even by considerable hemorrhages; certain isolated spots become anæmic from the vascular spasm. The vaso-motor nerves of the lungs do not pass through the pneumogastric nerves, but through the cervical portion of the spinal cord and the first thoracic ganglion of the sympathetic.

Among the morbid signs of tumors of the pons we may first consider the motor disorders, since these are the most characteristic. General convulsions are rarely observed as symptoms of motor irritation. They may be due to irritation of the surface which unites the inferior border of the pons to the superior border of the acoustic tubercle, and which represents Nothnagel's convulsive centre. Paralyses constitute a much more constant and important symptom. They appear both in the cranial and spinal nerves, and are generally characterized by the fact that the cranial nerves are paralyzed on the same side as the tumor and the spinal nerves upon the opposite side (alternate hemiplegia of Gubler). Unilateral affections of the pons are most frequently accompanied by paralysis of the facial, acoustic, oculo-motor communis, trigeminal and hypoglossal nerves, and by lesions of the optic nerve. Complete or incomplete hemiplegias, upon the side opposite the tumor, are the most frequent. They existed 12 times in 26 cases collected by Ladame; paresis of one or the other upper limbs and symptoms of paraplegia are very rare phenomena. The limbs were not paralyzed in a few cases, in which the longitudinal fibres of the pons were undoubtedly unaffected by the tumor and had been pushed to one side.

Manége movements do not make their appearance unless the lesion extends to the middle cerebellar peduncle, which is connected with the transverse fibres of the pons. The movements of partial rotation are caused, according to Schiff, by a partial lesion of the most posterior of the transverse fibres, and which is followed, in animals, by rotation of the cervical vertebræ (with the lateral part of the head directed downwards, the snout directed obliquely upwards and to the side). Symptoms of a similar nature are also observed in man. Thus, in Peyrot's patient (cholesteatoma of the posterior portion of the pons and the posterior lobe of the cerebellum), the head was affected by an irresistible rotatory movement. In a personal case, torsion of the neck occurred with rotation of the head forwards and to the left.

1. In a patient, under my care, increasing paralysis of all the limbs had existed for a year; the walk was uncertain and tottering. The mental faculties were con

siderably weakened, speech was unintelligible, the words confused and indistinct from frequent sputtering, the pupils dilated, the gaze fixed and expressionless. Autopsy: The middle third of the anterior segment of the pons, especially above, as far as the lower limit of the tegmentum, was of a grayish color, partly gelatinous, partly firm. The new-growth (composed of large cancerous cells with processes and bands of connective tissue) had proliferated into the internal portion of the cerebral peduncle as far as the anterior portion of the optic thalamus.

Paraplegia may also develop, either from the compression of the medulla oblongata or the secondary degeneration of the spinal cord (as in Luys' case). This symptom has not been hitherto studied with sufficient care. In a personal case, which I shall report at a later period, weakness occurred first in the right and then in the left half of the body; the tumor of the pons had compressed the fourth ventricle and olivary bodies. Facial paralysis frequently occurs in tumors of the pons. In Ladame's 26 cases, it occurred 11 times and always upon the same side as the tumor, while the paralysis of the limbs existed upon the opposite side (alternate hemiplegia). According to Brown-Séquard, when the lesion of the pons is situated in the centre above the decussation of the facial nerves, the facial paralysis occurs upon the same side as the hemiplegia; when the lesion is peripheral and below the decussation of the facial, the facial paralysis is alternate with reference to the hemiplegia. It appears to me more reasonable to believe that if the compression takes place at the decussation of the fibres of the facial nerves, which occurs above the nucleus of origin, the facial paralysis will be entirely alternate; but if the tumor compresses the roots of this nerve, its paralysis will occur upon the same side (I shall discuss the electrical reaction hereafter). When the lesion affects the inferior nucleus of the facial, the alternate facial paralysis will be only partial.

In an article upon the characteristic signs of tumors at the base of the brain (Wien. med. Halle, 1863, 6–9), I published the first observation in which facial paralysis, due to disease of the pons, was accompanied by diminution and, finally, by abolition of the farado-muscular contractility. Duchek has also described (Med. Jahrb. 1 H., 1865) a case of tumor of the pons, in which he observed diminution of electro-muscular contractility upon the paralyzed half of the face and body. My own observation in this regard deserves especial attention on account of the complications which were present.

2. The patient, 38 years of age, complained of headache which had lasted five months, in the temporal and occipital regions, and upon which a diminution of motion and of the visual function gradually supervened. Upon examination the patient presented double amaurosis (atrophy of the optic nerves); both pupils are dilated. The face is paralyzed upon the right side, the velum palati on the left side is thinned, more elevated and, in the pronunciation of vowels, is drawn upwards more strongly than on the right side. The ocular conjunctiva, nares, vault, and velum palati, the tonsils, mucous membrane of the cheek, gums, tongue, and upper teeth, are completely anæsthetic on the right side; the lower teeth and the floor of the mouth have preserved their normal sensibility. The anæsthesia of the right cheek is bounded behind by a vertical line starting from the concha of the ear and extends upon the scalp, forehead, and face, as far as the lower jaw. Upon faradic exploration, the electro-muscular contractility is found to be entirely abolished upon the paralyzed half of the face. The left lower and upper limbs are paretic. Taste and smell have disappeared upon the right side, but hearing and speech are preserved. In the next six months paralysis of the right external oculo-motor nerve and amaurosis occurred. The left half of the body remained paretic, while complete hemiplegia appeared upon the right side. In the last two weeks right pleurisy supervened, and the abolition of motion rapidly became complete.

Autopsy: A nodular tumor, larger than a nut, was found upon the left half of the

pons which was partially flattened and compressed: in the centre it contained bands of hard connective tissue of a yellowish color, which disappeared towards the softened and reddish-gray periphery. The tumor also extends upon the cerebellar peduncle as far as the left cerebellar hemisphere which, together with the left olivary body, has suffered compression. The fourth ventricle is compressed laterally. The trigeminal, facial, and acoustic nerves are enclosed in a thick layer of the new-formed tissue.

I have learned, by more recent investigations, that the electrical phenomena which we have described in facial paralysis from tumors of the pons, should be rendered complete by the following considerations: to the loss of faradic excitability of the muscles and nerve-fibres of the face, is added an increase of the galvano-muscular contractility with diminution or loss of the galvanic excitability of the branches of the facial. The following observation of a tumor of the pons is especially interesting on account of these characteristic symptoms:

3. In the beginning of June, 1871, a woman, aged forty-five years, entered the service of Dr. Scholz. She had suffered for four years from headache and vertigo and, for the past four months, from paralysis of the left cheek and of the limbs upon the right side of the body. Upon examination I found complete facial paralysis upon the left side and an increase of the cutaneous sensibility to physical and electrical irritants, with loss of farado-muscular contractility and of the excitability of the branches of the facial nerve. The galvano-muscular excitability is notably increased (the right side gives no reaction to a slight current), especially to the descending current at the negative pole and upon interruption of the current. There has been loss of hearing (the tympanum is normal) upon the left side since the beginning of the disease (according to Politzer the labyrinth was diseased from compression of the acoustic nerve). When the tongue was protruded, it deviated to the right.

The right eye is sensitive, the ocular conjunctiva strongly injected, the cornea is softened, the right limbs are paralyzed, with diminution of the electro-muscular contractility, and the electro-cutaneous sensibility is much weaker than on the left side. In order to distinguish the points of the æsthesiometer apart, they must be separated, on the palm of the left hand, to 11.2 mm.; on the right hand, to 38.2; on the left forearm, to 43.5; on the right forearm, to 61.3; on the left leg, to 41.7; on the right leg, to 59.2. These phenomena persisted unchanged during a stay of three weeks in the hospital. Towards the end of the month the patient returned to her relatives. Baerwinkel has lately published (Arch. f. klin. Med., XII. Bd., 1874) a case of tumor of the pons, in which electrical exploration upon the side of the facial paralysis gave similar results.

When the tumors of the pons extend to the middle cerebellar peduncles a deviation of the cervical vertebræ is observed in man, similar to that noted in the above-mentioned experiments of Schiff. When the fifth pair of nerves are subjected to prolonged compression, ophthalmia occurs from lesion of the trigeminus, as in the cases which I have published above and which presented the symptoms of an affection of the pons and adjacent structures.

4. An assistant surgeon, thirty-four years of age, presented, upon admission, paralysis of the right limbs (which had lasted eight months) and left facial paralysis. Later, paralysis and anæsthesia also occurred in the domain of the left trigeminus, external oculo-motor and acoustic nerves. The left eye could not be rotated outwards past the vertical line, the left upper lid remained open. The masseter and temporal muscles were also paralyzed, hearing and smell weakened on the left side, and taste upon the left half of the tongue. A burning sensation was felt at times upon the anæsthetic portions of the cheek; speech is stuttering; the head is always directed to the left and forwards. Contractions and pains occur from time to time in the right limbs. About four days before death inflammation and tumefaction of the left conjunctiva occurred, with an abundant purulent secretion. Upon the following day the cornea was of a dull white color and became more and more opaque. It assumed a cadaverous appearance, became completely opaque and diffluent and finally

softened and ulcerated, allowing the crystalline lens and aqueous humor to escape. Upon the autopsy a tumor as large as a walnut was found upon the left half of the pons varolii and the left cerebellar peduncle.

The pons is often secondarily affected by the extension of growths which have developed in adjacent portions of the brain; the symptoms are then complex. Thus, in an observation by Sarne (Gaz. des Hôpit., 196, 1869) a child, four years old, who suffered from epileptiform convulsions, presented, as the first morbid symptoms, disturbances of speech and paralysis of the left oculo-motor followed by paralysis of the left side of the face and of the right limbs. Towards the close of life the child suffered from disturbances of deglutition and from paralysis of the right oculo-motor nerve. Upon the autopsy a cheesy tubercle, the size of a walnut, was found upon the pons varolii, occupying the entire left side and a portion of the right, and extending in front across the left peduncle and as far as the centre of the right peduncle.

Finally, we must mention paralyses of the ocular and tongue muscles among the motor disturbances in pons tumors.

With regard to the first we are of Larcher's opinion (Essai sur la path. de la protubér. annulaire, thèse, Paris, 1869), according to whom the diverging strabismus, which occurs in rare cases, is due to an extension of the lesion to the cerebral peduncle, while the converging strabismus (paralysis of external oculo-motor) is much more frequently caused by tumors of the pons itself. The disorders of speech, frequently mentioned by Ladame, Da Venezia and Larcher, generally affect the articulation of words. The lesion of speech is of a motor nature from compression of the roots of the hypoglossal nerve, and must not be mistaken for aphasia. Leyden designates, by the term anarthria, those disturbances in the articulation of words which follow a lesion of one of the motor centres situated below the tubercula quadrigemina (pons varolii and olivary bodies). According to Leyden and Meissner the voice is retained for a long time in birds, even after extirpation of the ganglia of the brain and base.

Sensory disturbances exist, according to Ladame, in about one-third of the cases. On careful examination they will undoubtedly be found even more frequent. The larger proportion of the sensory disorders are manifested upon the side of the body opposite the seat of the tumor. I have observed, in this particular, a characteristic phenomena which has not been previously described. As may be seen in the third observation of tumors of the pons, which I have reported on page 127, in addition to the alternate motor paralysis, crossed sensory disturbances also occurred (hyperæsthesia of the left cheek, anæsthesia of the right (paralyzed) limbs). I had previously reported this characteristic symptom (Traité d'électrothérapie, 2e édition, 1872) and Meynert confirmed the observation (Sitzber. d. W. Gesells. d. Aerzte, Jan. 31, 1873) in a case of encephalitis of the pons and of the right middle cerebellar peduncle. The alternation of the sensory paralyses depends, according to Meynert, upon a lesion of the root of the fifth pair in its central course which, before its decussation, passes through the pons and medulla oblongata. The posterior spinal columns, however, are directed upwards across the posterior portion of the pons and participate in the decussation in the pyramids. In my fourth observation crossed sensory disturbances were also present with alternate motor paralysis. In the exceptional instances in which the anæsthesia occurs upon the same side as the tumor, the latter, as Ladame has already remarked, has compressed the medulla oblongata; in other cases the tumor has directly compressed the trigeminus.

The mental faculties are very often impaired in tumors of the pons (about one-half the cases according to Ladame). The symptoms most frequently observed are loss of memory, apathy, and stupor, which are usually due to cerebral compression. Cephalalgia is very frequent, but its position varies a great deal and is of slight value in localization, being sometimes limited to the frontal or occipital regions and sometimes general and diffused. Disturbances of deglutition and vomiting are frequently observed among the later phenomena due to tumors in this region.

We may summarize the most important and striking symptoms of tumors of the pons varolii, as follows: absence of convulsions, crossed paralysis of motion, often also of sensation; frequent abolition of the faradomuscular contractility and increase of the galvano-muscular reaction, with complete unilateral facial paralysis; disorders of the special senses (especially amblyopia or amaurosis), difficulty in the articulation of speech, frequent dysphagia, converging strabismus.

XI.—Tumors of the Cerebellar Peduncles.

The cerebellar peduncles maintain intimate anatomical relations with the pons and cerebellum. The fibres of the middle cerebellar peduncle surround the pons in a transverse direction and, upon the sides of the latter, they are directed upwards, decussating, in greatest part, towards the inferior layers of the lateral lobes of the cerebellum. It therefore follows that lesions of the transverse fibres of the pons also act upon their continuation in the middle cerebellar peduncles, and that, on the other hand, degenerations of the cerebellum also cause lesions therein.

Schiff's beautiful experiments have established, in a striking manner, the truth and importance of the relations which we have pointed out. If the middle cerebellar peduncles are incised at the side of the pons, the animals, when they attempt to walk, will perform a rotatory movement directed towards the side of the lesion. The paralysis is therefore on the opposite side and its action is crossed. If the incision is made laterally through the lobes of the cerebellum the animal will turn to the side opposite the lesion. The paralysis is therefore upon the corresponding side, and the action is direct. This explains the apparent contradiction between the opinions of Magendie and Hertwig, on the one hand, and Longet and Laffargue on the other, with regard to the direction of the rotation.

Very recently Curschmann (Klin. u. Exper. z. Path. d. Kleinh., Schenkel, D. Arch. f. kl. Med., XII. Bd., 1873), by incision of the parts which unite the two cerebellar peduncles between the acoustic nucleus and the hemisphere of the cerebellum, has caused the production, not of a forced rotation, but of a forced lateral decubitus in such a manner that the animal fell upon the injured side and maintained this position until death, which occurred in a few hours. It resumed the same attitude when placed upon the other side or in any other untrammelled position. But in lesions of the acoustic prominence the animal always presented very marked rotatory movements around the vertical axis, from the sound side toward the side of the lesion, with coexisting deviation of the eyes. The eye upon the side of the lesion was directed downwards and forwards, that on the other side, backwards and upwards.

Curschmann obtained an autopsy upon a female phthisical patient, 39 years old, who had suffered from prolonged headaches, vertigo, and convulsions. She always lay upon the left side, with strong rotation of the head to the right and forwards, and

assumed this attitude as soon as she ceased to execute other movements. A foyer of softening, due to basilar tubercular meningitis, was found in the right cerebellar peduncle at the junction of the superior and middle peduncles.

Physiological experiments serve to confirm these pathological observations. The movements of manége, or the rotation around an axis, are shown in a characteristic manner in many cases. We do not know at present whether these motor impulses are due to defective consciousness (orientation) of the body with regard to its relations to surrounding objects or, as I incline to believe, to involuntary muscular contractions caused by lesions of certain central organs (as in the experiments which we have mentioned above). I have reported in detail, under tumors of the pons, a personal observation of partial rotation around the vertical axis with deviation of the head to the left (tumor of the right half of the pons, extending to the left cerebellar peduncle). I do not think it will be superfluous to add some cases drawn from the older literature as well as from more recent works, in order to obtain the most striking symptoms of the disease now under consideration.

Friedberg has published an interesting study in Wagner's Arch. f. Heilkunde (2 J., p. 385–432), upon the semeiotic importance of involuntary movements of manége and involuntary rotation around the vertical axis of the body. We shall here describe the most important observations contained in this work.

Among these cases (which include tumors, hemorrhages, and spots of softening) are some in which the rotation occurred from the side on which the lesion was situated towards the opposite side. Thus, in Serres' case (rotation from right to left around the vertical axis of the body, followed later by an attack of apoplexy and left hemiplegia) a cavity filled with blood-clots, with signs of cerebral softening, was found at the point of penetration of the middle cerebellar peduncle into the right cerebellar hemisphere. Belhomme's patient had lost consciousness, squatted down and turned rapidly around the vertical axis of the body (most frequently from left to right). Upon autopsy two depressions were found upon the two middle cerebellar peduncles. These depressions (more marked on the left side) were caused by two exostoses growing from the ridge of the sella turcica. A boy 4½ years old, observed by Minchin, had suffered at first from headache, periodical convulsions, disorders of deglutition and respiration; he was then affected with rotatory movements from left to right around the vertical axis of the body, and finally became hemiplegic on the right side. In this case (as in the following one of Friedberg) the rotation occurred first in the cervical, then in the dorsal, and finally in the lumbar vertebræ. A tubercle, as large as an almond, was found at the base of the left cerebellar hemisphere, inserted into the surface, with softening of the adjacent parts, probably as far as the cerebellar peduncles. Friedberg's patient had received a blow upon the anterior part of the head, causing a fracture with depression of the right parietal bone and subsequent meningitis. After the meningitis had subsided, movements of manége (forming a circle with the convexity directed towards the right) appeared in paroxysms, followed at a later period by rotatory movements from left to right around the vertical axis, and by polyuria and glycosuria. Autopsy: The dura mater, softened and discolored, was driven against the left inferior lobe of the cerebellum by a fragment of necrosed bone from the internal table of the occipital bone. Opposite this point the left cerebellar arachnoid was opaque, the pia mater strongly injected, and between the two a thin layer of fibrinous exudation. The injection of the pia mater extended also to the left cerebellar peduncle, especially to its internal part.

In Krieg's case rotatory movements occurred from left to right; at the autopsy a hæmorrhagic extravasation was found in the cortical substance of the right cerebellar hemisphere. Gustorff's patient suffered, after a fall, from several attacks of circular movements, with violent paroxysms of vertigo. A fibrous cyst, as large as a hen's egg, was found in the left cerebellar hemisphere; the cyst contained a small quantity of fluid and a bluish-black body, as large as a nut, infiltrated with blood and of a brawny consistence.

In other cases observed by Petit, Serres, and Weidler, lesions of the cerebellar peduncles and cerebellum were noted although rotation of the body around a vertical axis did not occur during life. Friedreich's patient suffered from violent pains in the left eye and from left facial neuralgia; in addition, he had right hemiplegia, weakness of sight and audition on the left side, with purulent ophthalmia. Upon autopsy a tumor as large as a hazel-nut was found in the left cerebellar peduncle. In an observation which I have published (Wien. med. Halle, 1863, 1869), the patient, who had suffered for several years from headache and frequent attacks of vomiting, was seized with left hemiplegia, facial paralysis, amblyopia and paresis of the external rectus upon the same side; the motility of the limbs and sight were less altered upon the right side. Loss of speech (with preservation of intelligence) occurred from time to time, lasting two or three days, and the patient remained in this condition until death, sometimes deprived of speech, sometimes barely able to stammer a few words. A tumor as large as a walnut (with softening of adjacent parts) was found in the left middle cerebellar peduncle; the optic nerves were compressed, flattened and hard.

Among the observations made in modern literature we may mention a case reported by Vigla (Gaz. d. Hôp., 72, 1866), in which the patient was affected with violent frontal headache, loss of sight (shining spots upon the fundus of the left eye) and of hearing; the head was directed backwards on account of contracture of the neck. While walking, involuntary movements occurred which were directed backwards and to the left. Autopsy: a hard tumor, the size of a walnut, was found upon the inferior surface of the right cerebellar hemisphere, towards its inner part; upon the right side atrophy was noticeable in the cerebellar peduncles, olivary bodies, pyramids, seventh and eighth pairs of nerves, and in the chiasm and right half of the tubercula quadrigemina, which were of a yellowish color; atrophy of the left optic nerve was also observed.

Bilot's case (Correspondenzbl. f. Psych., 3–4, 1867) was characterized by violent occipital headache and deviation of the left labial commissure. The left eye could only be half opened, the head was inclined to the left and forwards and, while walking, the body was markedly inclined to the right (with a feeling of oscillation). At a later period disturbances of respiration and hyperæsthesia (especially in the right limbs) occurred. Autopsy: the cerebellum was increased in volume, especially upon the left side, and contained a tubercle five centimetres long; the cerebellar peduncles, and medulla oblongata were pushed to the right and flattened; on the left side the olivary bodies and roots of the pneumogastric nerve were atrophied.

In Krauss's patient, who was four years of age (Allg. med. Centralzeit., Sept., 1867), uncertainty of the gait was noticed from the beginning, with weakness and tremor of the left limbs. The later symptoms were inability to walk, involuntary micturition, strabismus and amaurosis. Autopsy: a tumor, as large as a hen's egg, was situated at the base of the brain, occupying the position of the two halves of the pons, the cerebellar peduncles and the cerebellum, especially on the left side, partly destroying the vermis inferior and the middle cerebellar peduncles, filling the floor of the fourth ventricle without affecting its walls, and strongly compressing also the posterior portion of the chiasm.

A boy, three years old, was placed under my care, presenting an abnormal development of the anterior portion of the head and weakness of intellect; he did not speak, and suffered from periodical spasms of the extensor muscles. The head was constantly turned towards the left and forwards; the gait was unsteady and skipping, and associated with a tendency to fall upon the left side. When the child stands upright he turns two or three times around the vertical axis of the body. I diagnosed a tumor of the cerebellar peduncle with coexisting ventricular hydrocephalus.

The variations which have been observed, in different cases, with regard to the direction of the rotation, may be due, if we may judge from the above-mentioned experiments of Schiff, to localization of the disease of the cerebellar peduncles either within the region of the pons or that of the cerebellum. In accordance with Curschmann's experiments we

should, in pathological cases belonging to this category, examine the cerebellar peduncles and their appendages with great care, and should not overlook lesions of the acoustic prominence (acoustic nucleus). It still remains for us to dispose of the theory that the rare examples of anomalous movements, which we have passed in review, are due to diseases of the cerebellum itself. Magendie and Schiff's experiments have shown that these peculiar movements are not observed in lesions of the cerebellum unless the middle cerebellar peduncles are involved at the same time. Clinical observations also support this view. In Gavarret's and Belnomme's cases mention is only made of a lesion of the peduncles of the pons. In Curschmann's recent observation, quoted above, the left cerebellar peduncle was alone affected, the cerebellum and peduncles of the pons being intact. In Serres', Gustorff's and Friedberg's patients the peduncles of the pons were affected in addition to the cerebellum. It is true that in Minchin's and Krieg's published cases mention is only made of cerebellar lesions; but in these cases the middle cerebellar peduncles were not examined. Thus, physiological research, as well as pathological observation, prove that affections of the cerebellar peduncles are the principal cause of the motor disturbances under consideration.

From the preceding observations it appears that the most frequent signs of tumors of the cerebellar peduncles are as follows: headache, vertigo, disorders of the special senses, hemiplegia, unsteady gait with a tendency to fall upon the side, and partial rotation around a vertical axis, with lateral rotation of the head. The symptoms which are especially characteristic are: involuntary movements of manége or involuntary rotation around the vertical axis of the body.

XII.—Tumors of the Cerebellum.

The study of the cerebellum has, of late years, occupied the attention of anatomists and physiologists, as well as of pathologists, and a large number of observations are presented to our consideration. Recent histological researches have demonstrated that the cerebellar cortex is in crossed communication by means of the superior cerebellar peduncles, with the cortex of the brain (especially with the corona radiata). The middle cerebellar peduncle contains fibres which pass out from the cerebral ganglia through the cerebral peduncle (centrifugal fibres), in addition to commissural fibres between the two halves of the cerebellum. The inferior cerebellar peduncle originates from the motor and sensory portions of the restiform body. A portion of the posterior spinal column also originates in the cerebellum. Finally, the inferior cerebellar peduncle is connected with the acoustic nerve (which terminates in the cerebellum) and with the optic nerve.

With regard to the physiological functions of the cerebellum, we know since the time of Flourens, Carpenter, etc., that the organ plays an undoubted part in co-ordination of movements. While a bird, which has been deprived of one of the cerebral hemispheres, performs coordinated movements when it is pinched or when it attempts to escape, the animals, from whom the cerebellum has been removed, have an uncertain, wavering gait and often make missteps or stamping movements. Schiff attributes these movements to a paralysis of the vertebral column, which is, however, said to result from lesion of the cerebellar peduncles. According to Leven and Ollivier, isolated lesions of the cerebellum in animals

produce rotatory movements, general muscular weakness, strabismus, and sometimes incomplete hemiplegia. The animals, however, always recover at the end of one or two weeks. Lesions of the cerebellum and medulla oblongata, in addition to the preceding symptoms, cause the animal to fall and give rise to involuntary evacuations of urine and fæces, spasms, disorders of deglutition and respiration; death always occurs at the end of one or two days. According to Lussana the cerebellum is the centre of the muscular sense and, for this reason, lesions of the organ cause interference with the precision of movements. Despite the diversity of opinions upon the functions of the cerebellum it is evident that cerebellar lesions produce marked disturbances of co-ordination. These disturbances may vary greatly, according to the depth to which the lesion has involved the hemispheres and vermis, and also according to the portion of the posterior columns or of the fibres of the cerebellar peduncles, which has been affected. In a case of absence of the cerebellum, observed by Cruveilhier (Anat. pathol., Vol. I., liv. XV., p. 5), the patient suffered from weakness of the limbs, was unable to utter any sounds, and presented a high degree of imbecility, although the functions of special sense were preserved. Lussana's patient (atrophy of the cerebellum) presented an alteration of the muscular sense (Mi manca la terra sotto i piedi). The symptoms mentioned by other observers, such as hemiplegia or paraplegia, partial paralyses, and various anomalies in the movements of locomotion, indicate the frequent occurrence of complications in the appendages of the cerebellum.

Among the morbid symptoms of cerebellar tumors we may mention, in the first rank, cephalalgia, which, according to Leven and Ollivier and also according to Ladame, is most frequently situated in the occipital region, and is said by Friedreich to be increased by pressure upon the neck. The majority of authors coincide in the statement that the psychical functions and faculty of speech are only rarely affected. The most frequent and constant symptoms consist in disturbances of motion. According to the authors above mentioned, irritative motor symptoms are very frequently observed. These consist of general convulsions, which Lussana attributes to a coexisting lesion in the pons or spinal cord, but which Brown-Séquard considers to be due to lesions of the substance of the cerebellum. As symptoms of depression we may mention general muscular weakness, oscillations and uncertainty in walking, partial tremors of the limbs and paralyses, which vary in form and intensity. In addition to general muscular weakness we very often observe, according to Leven and Ollivier, limited paralyses (especially hemiplegia on the side opposite to the cerebellar lesion); paraplegia is rare. Movements of rotation and movements of manége were present in one-third of the cases collected by Leven and Ollivier. They are most frequently incomplete, and consist in torsion of the trunk, involuntary lateral inclination or in turning the head backwards. In three cases complete manége movements were noted. In the three cases cited by Ladame from Mettenheimer, Cazin, and Berenius, the symptoms were the same as those obtained in experimental lesions of the cerebellar peduncles. True disturbances of co-ordination are rarely observed. The examination of the spinal cord has been neglected in the majority of cases and too little attention has been paid, at the same time, to changes present in other portions of the brain. In a work by Tuerck, upon the primary degenerations of certain columns of the cord (Sitzber. d. Kais. Akad. d. Wiss., XVI. Bd.), a case of cancer of the vermis inferior, as large as a hen's egg, is mentioned, which occurred in a boy six years of

age (the history of the affection was not reported). Considerable degeneration, in a longitudinal direction, was observed in the posterior columns and the posterior roots; the author did not consider the degeneration secondary to the cerebral lesion. In a case recently published by Eisenschnitz (Jahrb. f. Kinderheilkunde) a girl, eight years old, was affected with double amaurosis, frequent epileptiform attacks, oscillation during walking (movements intact during the recumbent position) and frequent masturbation. Upon autopsy a soft, friable tumor, as large as an egg, was found in the right anterior cerebral hemisphere, resting against the falx cerebri; in addition, the cortical substance of the left cerebellar hemisphere, in a layer about one millimetre in thickness, was completely transformed into a hard, friable mass of a greenish-yellow color, with blood-red spots upon cut section.

In the Vienna General Hospital I observed a patient, forty-eight years of age, who, after suffering previously from headache, was suddenly seized, about twenty months before admission, with inability to walk and with loss of speech (without disturbances of the special senses); the previous health had been good. Upon examination I found: ptosis and paresis of the lower muscles of the face on the left side, speech stuttering, the tongue deviated to the left, and moved with difficulty, disorders of deglutition, and frequent regurgitation of solids and liquids. The upper limbs were paralyzed and anæsthetic, the lower were merely paretic, their sensibility being only slightly changed. Micturition and defecation were performed involuntarily. The patient fell into more and more profound apathy and died at the end of two weeks, with very rapidly developing bed-sores, high fever, and cyanosis of the face. Autopsy: The base of the right cerebellar hemisphere, with the exception of the flocculus, internal portion of the tonsil and the semi-lunar lobe, was replaced by a tumor as large as an apple, rough, hard, and giving exit to a little fluid upon section. It was of a uniform white color, with some spots of a dull yellow hue. The tumor penetrated the inner meninges at a spot hardly as large as a hazel-nut, and adhered closely to the dura mater at the anterior border of the foramen magnum. Chronic hydrocephalus, old tubercles at the apex of the right lung, and the lesions of Bright's disease in the left kidney, were also present.

Paralysis of the facial, oculo-motor, and hypoglossal nerves occurred in this case, in addition to the progressive paralysis of sensation and motion in the limbs. The peculiar character of the paralyses was perhaps due to the gradual compression of the cervical portion of the spinal cord. Among the special senses, sight and hearing were most frequently affected. The blindness (with final atrophy of the optic nerves, Galezowski) is due to the extension of inflammation to the optic track and geniculate bodies. The deafness is produced by the compression exercised by the tumor upon the acoustic nerve or by a lesion of the nucleus of this nerve. In a case reported by Gaston Sieffert (tubercle of the right cerebellar hemisphere) he was able to detect, with the ophthalmoscope, the presence of tubercular granulations in the right choroid. In the cases published by Edes (Boston Med. Journ., 1863) and by Tilling (St. Petersb. Zeit., 1872), the following symptoms were observed: neuro-retinitis with retinal hemorrhages, nystagmus and paralyses, some of which were direct, the others crossed with regard to the cerebellar lesion, on account of the compression of the medulla oblongata by the tumor.

The disorders of deglutition, the acceleration of the pulse, and the respiratory disturbances which occur in cerebellar tumors, must be attributed to a paralysis of the functions of the medulla oblongata. The irritation of the genital organs, noticed in some cases, proceeds, according to Longet, not from the cerebellum but from the subjacent medulla oblongata.

Finally, we may state, with regard to the differential diagnosis, that cerebellar affections are distinguished from ataxia by the frequent head-

aches (usually in the occipital region), by convulsions and by motor disorders which, with a little care, can be readily differentiated. The latter, according to Cyon, are caused by the vertigo. The patient experiences a sensation as if objects were revolving around him and lacks a point of support upon the ground. The ataxic, however, who is unable to stand on his feet, is free from all fear of falling when he is supported or is in the recumbent posture. In experiments upon animals, the motor disturbances only make their appearance when the cerebellar hemispheres and the vermis are excised to a certain depth, or when the portions that originate in the cerebellar peduncles are removed in great part. According to Schiff and Valentin these lesions cause weakness of the muscles of the vertebral column.

I am of the opinion that, in cerebellar affections, the central action, which presides over the fixation of the vertebral column and over the co-

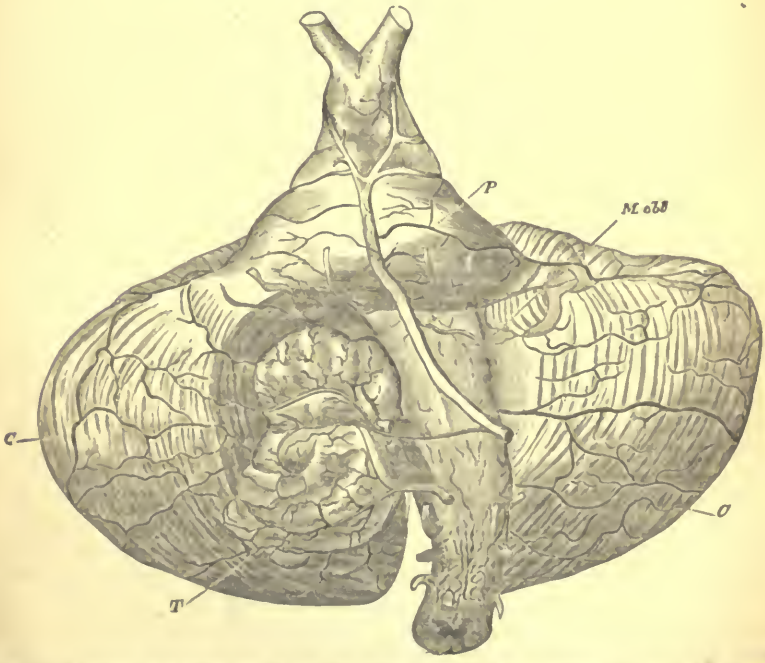

FIG. 10.—Tumor of the cerebellum, which has pushed the medulla oblongata to one side and produced softening in it. *T*, tumor; *C*, cerebellum; *M obl*, medulla oblongata; *P*, pons varolii.

ordination of the movements of the trunk, is interfered with and that this gives rise to oscillations of the trunk sufficiently marked to disturb the sensation of equilibrium and to cause vertigo, swaying and uncertainty of gait. In a more advanced degree, the vertical position and all movements of locomotion are affected. The movements of manége are caused, according to Immermann, by a predominance of the disorders of innervation upon one half of the trunk.

The differentiation between cerebellar diseases and sclerosis of the posterior columns is made by the ensemble of the following symptoms: in cerebellar affections, hemiplegic symptoms are very common, the para-

plegic forms are rare; movements of manége or of rotation (usually incomplete) occur; there are no disorders of coordination properly speaking, and no bilateral shooting pains; cutaneous hyperæsthesia is frequently observed; there are no symptoms of irritation or weakness of the genital functions and, finally, no anomalies of galvanic excitability.

We may summarize the principal signs of cerebellar tumors as follows: occipital headache, convulsions of an epileptiform character, oscillations, uncertainty of gait, intense vertigo, indications of rotatory movements, amblyopia or amaurosis, convergent strabismus, absence of psychical disorders.

CHAPTER XII.

PARASITES OF THE BRAIN.

Encysted parasites of the brain have only been known to physicians during the past century. Rendtorf (De hydatitid cerebr. tum. Berol., 1822) and Aran (Arch. gén., Sept., 1841) first gave an exact description of hydatids in the human brain. As a complement to the study of tumors developing in different regions of the brain, we shall now examine from an anatomical and clinical point of view, the animal parasites which occur in the cranial cavity, viz., the cysticercus (cysticercus cellulosæ) which is the more frequent, and the echinococcus (echinococcus hominis) which is of much rarer occurrence.

a. *Cysticercus of the brain* (*cysticercus cellulosæ*).

The cysticerci of the brain, which develop after the introduction of their embryons (ova of tænia), are observed both in the meninges and in the cerebral parenchyma. In general they are found lodged in the depth of the tissues and, exceptionally, in a free state. Among 88 cases, Kuechenmeister (Zeitschr. f. prakt. Heilk. 3.27, 1866) found the vesicles free in only nine, of which five occurred in the ventricles. The cysticerci were found in the meninges (especially in the pia mater) 23 times; upon the surface of the cerebral hemispheres, 59 times; in the white substance of the brain, 19 times; in the corpus striatum, optic thalamus, and adjacent commissures, 32 times; in the ventricles, 18 times (more frequently in the right lateral ventricle); in the cerebellum, 18 times; in the pons varolii, 4 times; in the medulla oblongata, twice.

According to Kuechenmeister and Ferber, the cerebral ventricles present the most favorable soil for the development of the cysticerci, and vesicles an inch in diameter or even as large as a pigeon's or hen's egg, are found in this position. They very rarely extend from one ventricle to the other, nor do several large vesicles occur in juxtaposition. The space between the meninges and the convolutions also appears favorable to the development of the parasites; the white substance of the brain yields, in this respect, to the cortical portions.

It is a peculiar anatomical fact that the parts which are rich in vessels, such as the ventricles, the ganglia and their commissures, the pia mater and the cortical substance, are the favorite site of the cysticerci. This fact appears to indicate, in my opinion, that the ova of these parasites, introduced into the digestive tract from without, are carried into the brain by the current of blood and deposited, by preference, in those regions which are richest in vessels. But, a certain number of the parasites may

pass through the walls of the vessels and, by following the meshes of the connective tissue, reach other portions of the brain or even other soft portions of the economy. Among 88 cases of cerebral parasites collected by Kuechenmeister, the cysticerci were found eleven times in other portions of the body.

The cerebral cysticercus is usually inclosed in a very soft spherical envelope in which the animal is seen, with the naked eye, as a small white tubercle. Under the microscope, the neck of the parasite becomes visible with the circle of characteristic hooks, curved inwardly. The cerebral tissue around the cysticercus is usually unchanged. Sometimes the meninges, at the point of insertion of the cyst, are merely affected to a slight extent or the adjacent cerebral parenchyma presents slight grades of pallor, œdema, atrophy, softening, ecchymoses or hæmorrhagic extravasations. In rare cases a firm capsule is present, with inflammatory thickening of the surrounding tissue. In the exceptional cases in which the cerebral ventricles contain free vesicles, the cavity is enlarged, the ependyma thickened and the adjacent cavities contain an accumulation of serum. In animals the vesicle is found shrivelled after death, its walls and contents opaque and granular, and the whole reduced to a pasty mass. In order to distinguish these masses from similar calcareous concretions following encysted cerebral abscesses and tubercular or syphilitic productions, we must be able to discern the characteristic hooks under the microscope. Thus, in Westphal's case (Berl. klin. Wschr., 43, 1865), Cohnheim found the remains of cysticerci in a calcareous mass of the fourth ventricle and, in addition, the headless ampullæ of cysticerci were present at the base, in the two fissures of Sylvius, in the pons, upon the sides of the medulla oblongata, between the origins of the nerves and on the cord, especially upon the cauda equina.

The cerebral cysticercus occurs very frequently in certain localities and rarely in others. These variations depend upon the importance attached to hog-breeding in different countries. According to Cobbold and Manning (Med. Times, Jan.–Feb., 1871), it occurs with greatest frequency in those localities in which cows pasture in fields which are strewn with the excrement of men and animals who live in cities. Cerebral parasites are usually found in individuals belonging to the lower and dirtier classes of society, and men are much more liable to infection than women. Kuechenmeister states that cysticerci are rarely observed in the first ten years of life ; the greatest frequency occurs from twenty to sixty years. According to Graefe (Arch., 12 Bd., 1866, oral communication from Virchow) cerebral cysticerci are found in Prussia in two per cent. of all autopsies.

The symptoms of cysticerci of the brain present great variations, according to the situation, size and number of the vesicles, and according to the extent to which they have affected the cerebral parenchyma. The coexistence of parasites in several portions of the brain complicates the symptoms, as in tumors. In quite a considerable number of cases they are manifested by very slight symptoms and the cerebral cysticercus remains latent, especially in children.

Among the frequent symptoms we may mention headache, vertigo, motor and psychical disturbances. Among the latter, the symptoms of irritation predominate in the beginning and often, also, for a certain length of time. Griesinger (Arch. d. Heilk., 5 H., 1862) first called attention to certain irritative phenomena on the part of motion and of the psychical faculties. In Kuechenmeister's statistics, which include eighty-

eight cases, epilepsy was noted twenty-four times, simple muscular spasms or convulsions six times, mental diseases twenty-eight times.

The epilepsy due to the presence of cysticerci takes an unusually rapid and precipitous course (in about half the cases, according to Griesinger). The attacks, at first infrequent or subacute, become more and more frequent and severe, and lead to death, after having finally caused grave cerebral symptoms, delirium, drowsiness and prostration of the vital powers. The epilepsy caused by cysticerci is more frequent in the male sex and occurs, according to Kuechenmeister, when the two hemispheres, the ventricles, pons, and medulla oblongata, have become involved. We must also mention certain irritative motor symptoms which occur in very rare instances. Thus, Choulant-Dommer (cysticerci of the vermis superior and middle lobe of cerebellum) and Griesinger (parasites in middle cerebellar peduncle and cerebellum) have observed spasmodic rotatory movements of the neck and head, with violent attacks of vomiting.

The mental disturbances, which develop in lesions of different parts of the cerebral cortex, appear under the form of delirium, maniacal attacks, illusions, and later as melancholy, somnolence, stupor and mental decay. A patient, forty-eight years of age, was received at the Vienna General Hospital, who, for ten years, had suffered from epilepsy, with very long intervals in which consciousness remained intact. During the week preceding death, the epileptic attacks succeeded one another very rapidly (80 to 100 daily). At the autopsy, nearly sixty cysticerci, larger than a pea, were found in the cortex of the two cerebral hemispheres, and another foyer in the right corpus striatum.

Paralyses of the limbs are rare symptoms in cysticerci of the brain. Among Kuechenmeister's eighty-eight cases, the crysticerci were situated seventeen times in the corpus striatum and anterior commissure, but well-marked hemiplegia was, nevertheless, observed only in three cases. Paraplegia appears exceptionally and only in lesions at the base. Paralyses of the muscles of the eyes and neck are also rare, but the sphincters are more frequently affected (nine times). From the preceding statements, it would appear that cysticerci of the brain usually separate the motor fibres without injuring them materially. The proof of this hypothesis should be sought for in future investigations, by minute examinations of the parenchyma. The large vesicles may, however, interfere with the motor fibres either directly or indirectly. More or less complete paralyses may also be caused by circulatory disturbances, following epileptiform attacks. Sensory disturbances are rare and depend upon secondary changes in the sensory cerebral fibres. The blindness, observed in a few cases, is caused by the presence of parasites in the chiasm, pons or cerebellum.

With regard to diagnosis, the probabilities, according to Griesinger, are in favor of cerebral cysticerci when, after previous headache, vertigo, vomiting and weakness of the limbs supervene, with paroxysms of muscular tremor and epileptiform attacks, especially when the latter occur towards the age of forty in previously healthy individuals, without any history of hereditary disease, traumatism, syphilis, or diseases of the large vessels. The epilepsy caused by cysticerci is at first subacute, as we have already stated. It then runs a rapid course and the attacks increase in number and violence, especially upon the approach of the fatal termination. In Ferber's case (Arch. d. Heilk., 6 H., 1862), despite the absence of epilepsy, the diagnosis was made during life from the other symptoms. If, after the above-mentioned prodromata, an alteration of the mental faculties supervenes with complex symptoms of depression (hear-

ing and sight enfeebled, photophobia, strabismus, anomalies of the pupils, headache, vertigo, somnolence, unilateral pains in the limbs, uncertain gait), we should suspect cysticerci of the brain, according to Griesinger, if we are able to exclude general paralysis of the insane.

Sometimes other data are present which render the diagnosis of cerebral parasites probable; thus if we learn, for example, that the epileptiform attacks and mental disturbances in question appeared in individuals who had previously suffered from tænia, if these symptoms appear in dealers in pork, butchers, etc., or if we find that, in addition to these phenomena, vesicular masses appear under the skin or in the muscles (as in Bonhomme's and Tuengel-Ferber's patients) which may be excised and whose characteristic appearances may be recognized under the microscope.

In the majority of cases, the development of cerebral cysticerci terminates in death after frequent and violent convulsions, more rarely after the symptoms of paralytic insanity (Joire). Death may be due to apoplexy, œdema, ventricular dropsy or inflammatory softening. In certain cases, the parasites perish in the brain without having produced any noteworthy symptoms, or the disorders which have been produced may then subside. But we are ignorant as to the causes of this fortuntae termination, and treatment does not possess the slightest efficacy in this direction.

b. *Echinococcus of the Brain.*

The echinococcus is rarely found in the cerebral parenchyma. Among 363 cases of echinococcus collected by Davaine, this parasite was found twenty times in the brain and sixteen times in Cobbold's 136 cases. Of forty observations collected by Morgan (Manchest. Med. and Surg. Rep., 1, 1870), the echinococcus was situated ten times in the cerebral lobes, eight times in the cereflellum (but only twice in the cerebellum alone), four times in the ventricles, twice in the corpus callosum, once in the pons, etc. Echinococcus cysts (contrary to cysticerci) attain large dimensions and not unfrequently grow to the size of a walnut or orange. In Morgan's case, the cyst, which was as large as a cocoanut, weighed 647 grammes. In Rendtorff's case (hydatids of the right hemisphere and of the lateral ventricle in a child of eight years), the total weight amounted to 1,050 grammes. The cysts attain their greatest volume in the cerebral hemispheres and in the lateral ventricles, especially in children, in whom the unossified skull yields more readily to internal pressure and in whom the cerebral parenchyma is better able to tolerate serious lesions. The hydatids are most frequently isolated but, in rare cases, several cysts or an agglomeration of hydatids are found in the brain. The cyst is composed of an external fibrous membrane, rich in blood-vessels, which encloses the parasites, and of a second adjacent envelope, soft, amorphous and transparent. Its internal surface contains groups of small nuclei or buds, as large as millet-seeds, each of which is provided with the characteristic ring of hooks. The cerebral echinococcus, with its hooks and scoleces (immature tænia) is very rare. The internal cavity of the cyst is usually filled with a liquid which is either clear or contains floating débris and secondary vesicles. These are called acephalocysts, and their buds are destitute of hooks. The external envelope is also sometimes wanting. Local reaction is manifested by congestion, inflammation,

softening and atrophy of the adjacent parts, in consequence of the compression to which they have been subjected. More rarely, we find partial scleroses or hæmorrhages; but dropsy and dilatation of the ventricles, anæmia and flattening of the cerebral parenchyma or thinning of the cranial bones in children, are of much more frequent occurrence. Sometimes, also, the echinococci perish in the brain, shrivel up and undergo calcareous degeneration.

The cerebral echinococcus in man is rare in certain countries, while in others, such as Iceland and Australia, it is frequently met with. In these countries the frequent transmission of the parasite to man is effected by the cattle and shepherd dogs. According to Morgan, the affection is more frequent in men than in women. The echinococcus is rare in the first fifteen years of life, increases in frequency from fifteen to twenty-five years, and then again becomes less frequent.

The symptoms of cerebral echinococcus are not generally characteristic. The most constant symptom is headache; then follow vertigo, vomiting, tremors, epileptiform attacks (19 times in 40 cases, according to Morgan) and visual disorders (in 27 cases) under the form of optic neuritis or white atrophy; intelligence is rarely affected. Two cases have been recently observed in which the echinococcus cysts made their way from the cranial cavity to the outside, and in which attention has been drawn to certain other signs. In Reeb's case (Recueil de mém. de méd. et de chir. milit. 27, 1871) of a boy five years old, affected with chorea and atrophy of the optic nerves, a fluctuating tumor was found upon the left parietal bone, making its way through a fissure in this bone. At the autopsy, vesicles of echinococci were found in the two posterior lobes and in the ventricles; the parasites had destroyed the dura mater and were making their way outside of the cranial cavity, between the lambdoid and sagittal sutures. A still more recent case is that described by Westphal (Berl. klin. Wschr. 18, 1873), in which a young man, twenty-seven years old, who had previously suffered from headaches, presented the following symptoms: vomiting, photophobia, disturbances of sight (blindness and right exophthalmos), and later, paresis of the left side and projection of the right parietal region. After temporary œdema of the eyelids and conjunctiva on the right side, two breaches were found in the bone of the frontal region, through which the tumors appeared. Upon incision more than 90 vesicles, varying from the size of a pea to that of the fist of an adult, with easily recognizable buds and hooks, flowed through the opening and through the left nares. The case terminated in recovery.

The diagnosis of intracerebral echinococci can only be made with a certain degree of probability (as in cerebral cysticerci).

When the hydatids are situated in the cranial cavity, Westphal states that the diagnosis must be made in accordance with the following circumstances: tumor-symptoms appearing and disappearing alternately, œdema of the eyelids, losses of substance in the bones, appearance of circumscribed tumors, explorative puncture. The duration of the symptoms in the cases collected by Morgan averaged one and a half years.

The disease nearly always terminates in death. Complete recovery only occurred in Moulinié's patient, fifteen years old (after trephining), in Fletcher's patient, twenty-three years old (after frontal incision), and in the above-mentioned case of Westphal. In a boy of ten years observed by Berncastle, very marked improvement occurred after a discharge of fluid from the ear. The treatment may be inferred from the foregoing facts. Morgan has had good results in sheep by the introduction of a

trocar through the ethmoid or through a soft part of the skull. In accordance with this he has proposed to trephine the skull in man and to puncture the brain with an exploring trocar. But on account of the slight degree of certainty to be attached to symptoms of cerebral echinococci, and of the almost absolute impossibility of recognizing its real situation even after palpating and percussing the previously shaven skull, it is only in very exceptional cases that we will be able to carry Morgan's suggestion into practice.

CHAPTER XIII.

DIATHETIC DISEASES OF THE BRAIN (CEREBRAL TUBERCULOSIS, CARCINOMA AND SYPHILIS).

THE diathetic affections of the brain, tuberculosis, carcinoma and syphilis, constitute the last chapter in our studies of cerebral disorders. With regard to syphilitic products, we might with propriety classify them among tumors on account of the similarity of their symptoms. But the large number of their peculiar characteristics and the more complete knowledge which we have obtained concerning them from recent researches, enable us to devote a special chapter to cerebral syphilis.

a. *Tuberculosis of the Brain.*

We are already acquainted with the acute forms of tuberculosis of the cerebral meninges (pp. 21–26). We will now occupy ourselves with the consideration of chronic tuberculosis of the brain and surrrounding parts. Tubercle of the brain is found both in the white and gray substance, more frequently in the latter. It occurs in the form of spherical masses, as large as a grain of hemp-seed or a lentil, isolated or conglomerate, and then attaining the volume of a small walnut or even of a hen's egg. Cerebral tubercle develops in various portions of the brain, in the cerebral hemispheres, motor ganglia, peduncles, pons, cerebellum and sometimes in the choroid plexuses. The tubercular masses present a yellowish color and a consistence which is sometimes firm, sometimes cheesy. When they form large agglomerations, the tumors are rough, contain several layers, and are very vascular. Cerebral tubercles are formed of rounded cells, partly fatty and atrophied, contained in a delicate fibrous framework with nuclear proliferations in the vessels.

At a more advanced period, the cerebral tubercle softens and disintegrates. Several years after recovery has taken place, the tuberculous tumor, especially in children, is found shrivelled, calcified, or of a doughy consistence and encysted. At times, the adjacent cerebral tissue presents no noteworthy changes. But when the tumor develops rapidly, it gives rise to phenomena of irritation or compression such as hyperæmia, small hæmorrhages, inflammatory degeneration or softening, or even atrophy and partial sclerosis. The accumulation of serum in the dilated ventricles is also a frequent complication.

Chronic tuberculosis of the pia mater or tuberculosis of the bones of the skull frequently coexists with cerebral tubercles. The former may exist as independent affections. Chronic tuberculosis of the pia mater is characterized by the presence of granular masses, varying from the size of a poppy-seed to that of an almond, and isolated or collected in groups. They develop upon the pia mater of the cerebral hemispheres and of the

cerebellum, but most frequently at the base. These small tumors adhere closely to the dura mater, which becomes thinned in consequence of the atrophy and loss of substance in the adjacent portions of the cranial bones. In other cases, they are directed towards the brain and their point of departure can only be recognized by the extensive adhesions of the pia mater. Tuberculosis of the cranial bones may be primary and may give rise to local caries, necrosis and fistulæ, or the rarefaction of the osseous tissue may be secondary to tubercles of the brain or meninges. Tuberculization often originates in the temporal bone. It proceeds from a tuberculous affection of the cavity of the tympanum or internal ear and, when it invades the Fallopian aqueduct and facial nerve, gives rise to facial paralysis. Caries of the mastoid process may also, as we have shown in the first chapter, give rise to meningitis, encephalitis or cerebral abscess, or to the grave symptoms of thrombosis of the sinuses. Sometimes the tuberculization starts from the petrous portion of the temporal bone or more rarely from the bones of the orbital cavity or nasal fossæ, extends through the meninges to the cerebral parenchyma, and tuberculous products develop abundantly at the base, in the cerebellum or in the adjacent cerebral lobes.

Tuberculous products in the brain are nearly always accompanied at a later period by tuberculosis of the lungs, bronchial and mesenteric glands, and very often by tuberculosis of the pia mater. Death may result from miliary tuberculosis, tubercular meningitis, and sometimes from inflammatory softening in the surrounding cerebral tissue.

According to Fleischmann (Jahrb. d. Kinderheilk., II. Bd., 1872), cerebral tubercles in children are accompanied, despite the absence of circulatory disturbances, by hæmorrhages into the cerebral cortex, pleura, pericardium and kidneys, and by spots of emphysema in the lungs. The hæmorrhages occur upon the side opposite to the situation of the cerebral affection, like the analogous changes observed by Brown-Séquard in his experiments, upon the effects of lesions of the pons and cerebral peduncles. We can readily comprehend that the changes in the different nerve-fibres, produced by the cerebral lesion, may also involve the vascular nerves which traverse the organs at the base of the brain together with the other fibres.

The chief cause of cerebral tubercles is the tuberculous diathesis. They are much rarer in adults than in children. Ladame found them 64 times among 87 cases of tumors in children; and Rilliet and Barthez, among 312 autopsies on tuberculous children, observed cerebral tubercles in 37 cases. They occur with the greatest frequency from the first year of life until the second dentition, that is to say, during the period in which the cerebral development is most active. Different observers do not agree with regard to the influence of sex. Cases of chronic cerebral tuberculosis are very frequent at the age of puberty, but they become infrequent after the fortieth year. Cerebral tuberculosis of adults may be primary and sometimes develops in several parts of the brain or at the base, without the coexistence of tuberculosis in other organs. In the majority of cases, however, especially in old people, cerebral tubercles develop after the diathesis has been manifested in other viscera. Injuries and exposure appear to favor the outbreak of cerebral tubercles in individuals predisposed to the affection.

The symptoms of cerebral tubercles present a great variability according to the situation, size and manner of development of the tumors. Isolated and small tuberculous products, developing slowly at the convexity of the cerebrum or cerebellum or in the neighborhood of the ganglia, may remain latent. A tubercle larger than a hazel-nut and which had

manifested no especial symptoms during life, was found upon the convexity of the right hemisphere in a patient who died of phthisis at the Vienna General Hospital. A similar occurrence has also been observed in children. We merely notice in them a change of temper and slight headache or isolated spasms, and even if we suspect tuberculosis we are unable to diagnose cerebral tubercles, since similar irritative symptoms are habitual among children in various other diseases.

Larger tubercles, occupying certain portions of the brain, often give rise, on account of their isolation and continuous growth, to perfectly well-defined symptoms of tumor. In order to establish their special symptomatology, we must rely chiefly upon the pathognomonic signs to which we have called attention in detail in the preceding chapter on tumors of the different regions of the brain. Chronic headache, with exacerbations and mental disturbances, are present from the beginning. In adults, the latter consist of apathy and absent-mindedness and, in children, of an easy and indolent disposition. When the tuberculous products appear early and are accompanied by hydrocephalus, the intellectual development is retarded. Furthermore, disorders of the special senses, especially of sight, frequently occur.

With the ophthalmoscope, we may recognize optic neuritis and, very frequently also, tubercles in the choroid, upon one or both sides, in the region of the papilla and yellow spot. Manz first discovered them in this situation (Arch. d. Ophth., IV. Bd., 1858); later, according to Graefe and Leber (Arch. f. Ophth., XIV. Bd., 1868), Cohnheim observed them in 17 cases of miliary tuberculosis. They develop in the choroid coats of guinea-pigs after inoculation with tubercle. Graefe and Leber have given the same description of tubercles of the choroid (with regard to their situation, number and parallactic deviation) in the tubercular meningitis of adults and of children. In two cases published by Fraenkel and Leber (Berl. klin. Wschr. 4, 1859), the tubercles of the choroid were visible in the children before the patients were affected with any disturbance of intelligence or sight. In one of these cases, the diagnosis was confirmed by the autopsy. Not long ago, Bouchut and Sieffert (*loc. cit.*) demonstrated, upon the living subject, the existence of choroidal tubercles in cases of tubercular tumors of the peduncle, base of the brain, and cerebellum.

Motor disturbances are among the most striking symptoms in these cases. The irritative motor symptoms consist of muscular spasms, unilateral or more extended convulsions, epileptiform attacks and rotatory movements (observed in tubercles of the cerebellum and right cerebellar peduncles by Minchin, Bilot, and Vulpian). The case which I reported under the head of tumors of this region, also presented symptoms of this nature.

The phenomena of depression are manifested by various paralyses. Hemiplegia is the most frequent form. From its possible combination with paralysis of certain cranial nerves, the alternation between the paralyses of motion and sensation in the face and limbs, the different electrical reactions, the existence of disorders of co-ordination, ophthalmia depending upon lesion of the trigeminal nerve, and from disturbances of the special senses, we may obtain the data necessary to localize the cerebral lesion (as shown by the examples reported in the preceding chapter).

The diagnosis of cerebral tubercles is nearly always surrounded with great difficulties. We can only recognize it with certainty in the living in the few cases in which the symptoms are grouped in an especially favorable manner.

When a patient presents cerebral symptoms, we may attribute them to

the presence of tuberculous products in the brain or neighboring parts, under the following circumstances: coexisting pulmonary disease, often hereditary; caries of the vault of the skull, or petrous portion of temporal bone (rare); tuberculous ozæna (syphilis excluded) with fistulous canals opening externally; in children, signs of tuberculization in the lungs and bronchial or mesenteric glands; scrofula, rachitis, tendency to moist eruptions; finally, tubercles in the choroid recognizable with the ophthalmoscope. The termination of chronic tuberculosis of the brain is nearly always fatal. Although we sometimes meet with cerebral tuberculous products which have become transformed into calcareous matter, these cases are too rare to sensibly diminish the gravity of the prognosis. The cerebral lesion itself with its sequences, hydrocephalus, inflammatory softening, hæmorrhages and basilar meningitis, is the most frequent cause of death. When the cerebral affection is less threatening, the patients may succumb to pulmonary tuberculosis. Children usually die from the internal hæmorrhages, which we have mentioned above, or from pneumonia, bronchitis and acute pulmonary œdema. Our previous remarks suffice to show the slight efficacy of treatment. With regard to such symptomatic measures as may be adopted, *vide* the statements made in preceding chapters.

b. *Carcinosis of the Brain.*

The brain does not constitute a frequent site of carcinoma. Among 285 cases of cancer of the different organs, collected by Chambers (Brit. Review, July, 1853), the brain was only affected twelve times. The cancer may be primary and isolated in the brain tissue without involving other portions of the body. On the other hand the carcinoma which originates in the dura mater, especially at the base, or in the cranial bones, coincides with multiple manifestations in other organs and in neighboring lymphatic glands. When cerebral cancer is secondary it is usually multiple and, according to Rokitansky, it not infrequently develops symmetrically in homonymous parts of the brain.

Cancer is especially frequent, and usually primary, in the cerebral lobes, in the pons (eight times in Lebert's ninety-six cases), in the cerebellum and central ganglia. Carcinomata very often originate from the dura mater, especially of the base and cerebral fossæ; or they may enter the brain from the orbits (more rarely the nasal fossæ) and from the pterygoid fossæ or sphenoidal sinuses. The new growth frequently originates in the osseus tissue of the sella turcica or petrous portion of the temporal bone, etc., from whence it extends either in an inward or outward direction. Sometimes the neoplasms develop in the cranial coverings and enter the skull through the sutures and foramina or through the rarefied tissue of the cranial bones.

Medullary cancer of the brain, which is the most frequent variety, appears under the form of rounded, rough tumors, with several lobes, of a soft consistence and varying in color, according to their vascularization, from yellowish-white to reddish-brown. Fibrous cancer is usually smaller and smoother, of a harder, more compact structure and poorly supplied with blood-vessels. The soft tumors adhere to the subjacent parts and are detached with difficulty. They give rise to attenuation or hyperplasia of the adjacent osseous tissue, to thickening of the meninges or neuroglia, and may also cause inflammatory softening or hæmorrhages into the surrounding brain tissue. Cerebral cancers contain extremely variable pro-

portions of blood. From lesion of the vascular walls, more frequently from rupture, they may allow an ichorous fluid to escape; or, in consequence of atrophy, fatty degeneration of their elements and disappearance of their vessels, they may thicken in part like tuberculous matter.

The symptoms of cerebral cancer vary greatly. The local symptoms resulting from the action of the growth upon the surrounding parts, are the most characteristic. Large tumors, rich in cells and blood-vessels, may be subject to hyperæmic enlargement, giving rise to periodical increase of the cerebral compression, to anæmia and epileptiform attacks. Soft multilobular tumors, which are rich in cells, compromise the functions of the brain by their rapid development, the hard, scirrhous tumors, by their consistence. The patients do not always present the signs of the cancerous cachexia. The diagnosis of cerebral carcinoma is only possible when the symptoms of a tumor of the brain appear at the same time with evident cancerous degeneration in other organs, or when the tumor symptoms coincide with infiltration and hardness of the lymphatic glands and parotid and when we can exclude other diathetic lesions.

The prognosis is always very grave. Death is caused by the lesions produced by the cancer in the brain itself, by its spread to other organs, and by the rapid growth of the cancer in the brain (especially frequent, according to Rokitansky, in the melanotic variety). Treatment consists in palliating the painful symptoms by subcutaneous injections of morphine, hydrate of chloral, etc.

c. *Syphilis of the Brain.*

Syphilis may also extend its terrible ravages into the nervous centres. In syphilitics who present cerebral symptoms, the specific lesion may be situated in the cranial bones, in the meninges and dura mater or in the cerebral parenchyma. Thickenings, tumors, gummata and spots of softening play the most important part in the cerebral manifestations of syphilis.

Pathological Anatomy.

Before examining the changes produced by syphilis in the interior of the cranial cavity, we shall first discuss those produced in the bones of the skull. Syphilitic affections may appear upon the internal surface of the skull under the form of tophus or of characteristic osseous cicatrices with more or less abundant osteophytes. Syphilitic caries due to periostitis and osteitis usually invades the flat bones (very rarely the base of the skull, Heitz) and is accompanied, according to Rokitansky, either by a diffuse sanious secretion or by active proliferation of the connective tissue, and by osseous sclerosis with partial necrosis. In another and equally frequent form of caries, the dry caries or inflammatory atrophy of the osseous cortical layer (knochenrinde of Virchow)—a form which constitutes foci, with central atrophy and peripheral hypertrophy—suppuration never occurs. The losses of substance resulting from atrophy of the osseous tissue are filled by gummy products which develop from the periosteum, or by the dura mater which has become thickened and adherent to the underlying meninges. Sometimes, according to Breslau, the thinning of the flat bones of the skull is due to atrophy from resorption of syphilitic exostoses or tophus.

Syphilis of the meninges consists in chronic inflammation, in infiltration

with osseous products, or in the presence of gummata. The inflammation of the dura mater (usually produced by a syphilitic affection of the pericranium or of the bones of the skull) appears as an external or internal pachymeningitis. It causes firm thickening of the dura mater and adhesions to the cranium and subjacent meninges, and sometimes leads to puffiness or fatty degeneration of the inflammatory products and ossification of the new connective tissue membranes (osteophytes). The pia mater is also often inflamed. This meningitis which is usually chronic. (Flechsig, Ueber Mening. luet. Inaug. diss., 1870) rarely extends over a considerable space. It is generally circumscribed (especially at the base), and is evidenced by changes in the meninges and by exudations and connective tissue products, the retraction of which causes strangulation and partial atrophy of the cranial nerves at the base.

A second form of meningeal syphilis is constituted by gummy tumors (syphilomata). These neoplasms originate in the dura mater or pia mater, and sometimes in the cranial bones. They appear, according to E. Wagner (Arch. f. Heilk. III. u. IV., Jahrg. 1862 u. 1865), as diffuse, soft infiltrations of a reddish gray color, or as rounded (sometimes irregular) prominences attaining the size of a fist, or even as nodosities, under the form of diffuse infiltration. The syphiloma is composed of abundant nuclei and cells, disseminated throughout the new-formed connective tissue and looking like white blood-globules. Later, it becomes filled with regular alveoli. According to Rindfleisch, syphilomata develop in the brain at the expense of the lymphatic sheaths and along the vessels, and they produce spots of softening by compression of the vessels and arrest of the circulation. The syphiloma may dry down or may cause a loss of substance or the formation of a cavity. The latter contains the débris of a yellowish cheesy matter in the midst of which is a little serous fluid. The syphilitic changes in the cerebral parenchyma vary in their character. Circumscribed cerebral inflammations may accompany specific meningitis or may follow in the train of syphilitic tumors. Primary specific inflammation of the cerebral parenchyma has not been hitherto observed in adults, but, according to Virchow, congenital interstitial encephalitis only occurs in syphilitic children. Under the same circumstances we also find thickening of the ependyma and small foci of fatty degeneration in the ventricles which are enlarged and contain more serum than normally. Syphilomata rarely occur in the cerebral parenchyma. Nevertheless, Virchow, Westphal, Charcot, etc., have seen them upon the surface and in the white substance of the hemispheres, in the optic thalamus, optic tracts, pituitary gland, cerebral peduncles, pons and cerebellum. They frequently give rise to partial sclerosis or hemorrhagic softening of the surrounding cerebral substance. They may cause nuclear proliferation in the nerve-cells of the adjacent cerebral cortex and considerable enlargement of the cells of the connective tissue. Under the etiology of the subject we shall discuss the specific changes in the cerebral vessels. Finally, the optic nerves may become inflamed and the nerves at the base of the brain may undergo fatty degeneration and atrophy.

Etiology.

Cerebral disturbances from constitutional syphilis may sometimes develop in the first few months, or in the year following the infection. Usually, however, several years elapse before cerebral syphilis makes its

appearance. It occurs with the greatest frequency in middle age. The congenital lesions described by Virchow are rare and their existence has been questioned for some time past.

Several years ago attention was directed to the important part played by specific changes of the vessels in syphilitic cerebral affections. Since Passavant's observations (Virch. Arch., XXV. Bd.) upon syphilitic thickenings of the basilar artery by the formation of exudation and thrombosis, Clifford Albutt (St. George Hosp. Rep., III., 1868) has several times found infiltrations and small ulcerations of the middle coat of the large cerebral arteries in cases of syphilis. Syphilitic thrombi and emboli have been found in all the large arteries at the base. More recently Heubner has pointed out the influence exerted by syphilis upon large cerebral arteries. He has observed nuclear infiltrations of the tunica adventitia and obliterations starting from the internal coat in the vessels at points corresponding to adhesions of the meninges and brain. The same changes have been found in the central artery of the retina, together with infiltration of the optic nerve. Virchow (Krankh. Geschw., II. Bd., p. 444) has found gummy inflammation of the walls of the aorta. Hertz (Virch. Arch., Bd. 57, 1873) found a large aneurism of the ascending aorta, composed of several cavities with thickened walls, which were formed of several layers and filled, in part, by a yellow, cheesy mass.

But Heubner (Die leutische Erkrankung der Hirnarterien, Leipzig, 1874) has especially recognized the importance of the syphilitic neoplasms which develop within the large arteries at the base of the brain. These arteries are often affected in consequence of syphilitic changes in neighboring parts but sometimes, also, independently. At first the vessels lose their transparency and assume a whitish-gray color. They are hard and enlarged and finally acquire a cartilaginous consistence. Upon section, the lumen of the vessel is found diminished by a fifth or even a third of its normal calibre. Layers of a new-formed substance are noticeable, at first semi-lunar and then circular in shape. This substance is of a white or grayish color and at first of a soft consistence, but later it becomes as firm as cartilage. The new growth develops between the elastic layers of the internal coat (fenestrated membrane) and the endothelium. It is at first composed of endothelial cells, which continually proliferate and are finally transformed into a compact felted tissue, formed of fusiform and stellate cells. Rounded cells are deposited in this tissue, originating in the vasa vasorum and forming a granular substance. The new-formed tissue develops toward the centre of the vessel and along its longitudinal axis and thus diminishes, little by little, the calibre of the principal artery and its branches. Later it becomes organized or cicatrizes, and is changed into fibrous tissue. By the obstruction produced in the current of blood, it gives rise to thrombosis, cerebral softening and syphilitic cerebral inflammations, especially in the carotid, in the branches which pass to the lenticular nucleus and corpus striatum, in the middle cerebral artery, and in the vessel which supplies the corpus callosum.

The degeneration of the walls of the large arteries and of the cerebral vessels (which may be considered as a syphilitic endarteriitis) gives rise to multiple thromboses and emboli. The latter originate, in very rare instances, from the penetration of gummy tumors into the cardiac cavities (Oppolzer). The progressive development of the specific vascular degenerations explains the long duration of the prodromata, the changes and narrowing of the cerebral arteries, and the consequent disturbances of circulation and nutrition. They are the causes of the foyers of circum-

scribed softening, of the partial ruptures of the vessels with apoplectiform attacks, and of the symptoms of repeated thrombosis with frequent remissions. The degenerations and obliteration of the small vessels, which often pass unnoticed upon autopsy, give rise to less serious consequences. In Hertz's case, quoted above, the small cerebral vessels, even in the healthy regions and remote from the softened ganglia, were in a condition of fatty degeneration, and contained microscopic aneurisms.

Symptomatology.

The onset of cerebral syphilis is usually announced by prodromic symptoms, lasting several months, such as headache, vertigo, insomnia, neuralgia in the limbs, disturbances of intelligence and memory. These disorders, at first variable and intermittent, become gradually more and more severe or, more rarely, sudden paroxysms develop and terminate in disorders of motion, sensation, and of the mental faculties.

The motor disturbances, which are the most frequent, consist of symptoms of irritation or of paralysis. Among the first, we may especially mention apoplectiform, epileptic or maniacal attacks, due to meningeal exudation or to the compression exercised upon the cortical vessels during their passage across the pia mater or to the vaso-motor cerebral excitation provoked by the tumors. The violent initial attacks, which are sometimes accompanied by delirium and illusions, are generally of short duration. The apoplectiform attacks which follow the cerebral symptoms or which occur suddenly, with loss of consciousness, are accompanied by paralyses, which afterwards become hemiplegic in character, either gradually or after new attacks. The epileptiform convulsions are more frequent and persisting. They may appear in infants suffering from congenital syphilis; in adults they develop little by little, after syphilitic lesions of the skin and bones. They either appear as convulsions, progressing from the periphery to the centre and limited to one-half of the face and body, with moderate disturbance of the sensorium; or they involve the whole body, are attended with complete loss of consciousness and affect motion to a greater or less extent by their increasing frequency and intensity.

Paralyses must be regarded as among the serious effects of cerebral syphilis. From a recent abstract made by Braus (Monographie u. Hirnsyphilis, Berlin, 1873) it appears that paralyses occurred eighty-two times in one hundred cases of cerebral syphilis, and in the following proportions: paralysis of the ocular muscles, thirty-four times; paralysis of the face, twenty-seven; of the tongue, twenty-two; of the bladder, seventeen; of the intestines, fifteen; hemiplegia, thirty-one; paralysis of one limb, eighteen; paraplegia, eight. Paralysis of the cranial nerves (oculo-motor communis and externus, patheticus and facial) are the most frequent. Then follow paralyses of one-half of the body. Isolated and unsymmetrical paralyses of the extremities are much rarer. These paralyses are sometimes incomplete and temporary, sometimes more complete and permanent. The variations depend partly on the resorption and retraction of the products of exudation, and partly upon the changes in the vascular walls and on the various conditions of the thromboses and collateral circulation. These multiple paralyses are caused in general by the presence of gummata at the base of the brain, by chronic basilar meningitis with strangulation of the cranial nerves from retraction of the exudation, and by syphilitic endarteritis of the basilar and vertebral arteries. In one of these cases re-

ported by Ziemssen (Virchow's Arch., XIII. Bd., 1858), faradic contractility was abolished in the completely paralyzed muscles and considerably diminished in the incompletely paralyzed ones. According to my own observations this abolition of farado-muscular reaction coincides with increase of the galvano-muscular excitability.

Hemiplegia occurs in about one-third of the cases of cerebral syphilis and varies in its development. It may appear slowly, as in cases of tumor when complicated with contractures, diminution of the electrical excitability of the muscles, and disorders of speech (foci of softening in the ganglia from obliteration of the middle cerebral artery). At other times the hemiplegia occurs suddenly with apoplectic symptoms or combined with epileptiform convulsions which sometimes mask the progress of the paralysis. Finally, hemiplegia, pursuing a progressive course, may appear as a complication of psychical disorders (as in hemiplegia with delirium).

Crossed paralysis occurs in the rare cases of syphilitic lesions of the pons. Paraplegia results most frequently from incomplete double hemiplegia in symmetrical lesions of the central ganglia, in lesions of the median portion of the pons, in compression of the medulla oblongata by diseased products at the base, and in degenerations of the spinal cord.

Cerebral syphilis sometimes, also, gives rise to *sensory disturbances*. In addition to headache, which is rarely absent, and to the frequent osteocopic pains, we observe, in the beginning, circumscribed trigeminal hyperæsthesia and neuralgia in the limbs. Hemiplegia may also be complicated by violent pains and various abnormal sensations. At a later period, numbness of the paralyzed limbs or unilateral anæsthesia of the scalp, face, tongue, and buccal mucous membrane often develop. Complete abolition of sensation upon one-half of the body is a much rarer event.

The most frequent and severe disturbances of the organs of special sense occur in the visual apparatus. When they first appear, under the form of rapidly increasing weakness of sight, they may rapidly recover under the influence of specific treatment. When they have given rise to optic neuritis, recognizable by the ophthalmoscope, they terminate in blindness. The visual disturbances are explained, according to Heubner, by the signs of inflammation which appear in the central artery of the retina. Amaurosis may be caused by the extension of morbid products from the pons, peduncles or tubercula quadrigemina to the optic tracts, or by specific infiltration of the chiasm and optic nerves. Taste, smell, and hearing may be diminished upon one side from anæsthesia of the trigeminus. The psychical disturbances, which are sometimes temporary and sometimes persisting, present no less interest. Braus found them present forty-five times in one hundred cases. They are almost always preceded by violent headache, lasting weeks or months, and which is especially severe at night. The affection is characterized by mental weakness, with or without exalted ideas, or by melancholia or mania, passing into imbecility. Frequently, all the symptoms of paralytic insanity make their appearance. Among forty-five patients of this category, Jaksch (Prag. med. Wschr. 1–20, 1864) found coexisting hemiplegia in 21 cases (some of the cranial nerves were also implicated). At a later period the paralysis extended to the tongue and to the muscles of deglutition and respiration. At the autopsy changes were observed in the meninges and cortex, the thickened meninges adhering to the cortical substance. In addition, foyers of softening were present in the superficial and deep parts of the brain, and specific lesions in the cranial bones and liver.

Disorders of speech have been noticed several times, either as aphasia

or as motor paralysis of the tongue. In Bouchard's patient (Gaz. med. 45, 1866), who suffered from apoplectiform hemiplegia and aphasia, the autopsy revealed the presence of softening of the second and third left frontal convolutions, with gummy growths in the meninges of the parietal lobe and in the pituitary gland. Leyden (Berl. klin. Wschr. 7–9, 1867) found, upon autopsy (in a case characterized by sudden paralysis with considerable disorder in the articulation of speech), spots of softening in the corpus striatum and optic thalamus and in the pons.

The later course of syphilitic cerebral diseases is very variable. In favorable cases recovery occurs at the end of several weeks or months. Death may occur in three or four years, rarely much later (Todd and Engelshed). Recovery is often interrupted by relapses, which may occur after intervals of several years, and may terminate in death by the development of new changes in the brain or by the extension of lesions to other organs.

Diagnosis and Prognosis.

In order to recognize the syphilitic origin of a cerebral affection we must not only enter carefully into the previous history of the patient, but also into his present condition. In suspected cases we must not alone examine the genital organs, skin, lymphatic glands, skull and long bones, but also the lateral and posterior portions of the buccal cavity, the base of the tongue, tne fauces and nasal fossæ, the epiglottis, larynx, and even the fundus of the eye with the aid of the ophthalmoscope. We must consider the possible occurrence of induration of the testicle and epididymis, of onychia and dactylitis, etc., and also examine the vascular system, the heart and the liver.

In young people and adults headaches occur, at first slight and intermittent and then becoming severe, with exacerbations, especially at night. Insomnia, ptosis, ocular paralyses and dilatation of the pupils, or visual disorders then develop, followed later by unilateral spasms of the face or limbs or by epileptiform convulsions together with the signs of optic neuritis. At this time, or even later, hemiplegia makes its appearance.

Multiple paralyses of the cranial nerves do not aid us much in the diagnosis of cerebral syphilis in the absence of other specific symptoms, since similar paralyses occur in circumscribed meningitis and tumors at the base. But when epileptiform convulsions are superadded or when apoplectiform attacks occur after rapid improvement of the paralyses, we may attribute the symptoms to cerebral syphilis, especially if the patients are young or middle aged.

In meningeal inflammation due to cerebral syphilis, the pulse may not be frequent and the temperature but slightly raised, despite the severity of the symptoms. Antisyphilitic treatment will produce rapid disappearance of the cerebral phenomena, but if medication is abandoned too early they will soon reappear (Poncet). The diagnosis of syphilitic mental diseases is not possible except from the coexistence of other symptoms of syphilis, of the headaches which we have described above, and from the effects of antisyphilitic treatment.

The cerebral symptoms of congenital syphilis usually appear under the form of epileptiform convulsions, occurring during the first ten years of life. In these cases we find ulceration of the palate and uvula, enlargement of the bones of the nose, periostitis, ozæna and hoarseness. A peculiar appearance of the superior median incisors (wedge-shaped with crenated

edges) is regarded as characteristic by Hutchinson. A history of syphilis may usually be obtained from the parents. With regard to prognosis, the invasion of cerebral syphilis must be regarded, in general, as a serious condition. The acute irritative symptoms are usually less grave and persisting, since they can be more readily overcome than the chronic symptoms of depression.

The prognosis is so much the more grave when the nervous centres are more largely involved. Among 21 cases collected by Gros and Lancereaux (Des affect. nerv. syphilit., Paris, 1861), in which the syphilis had involved the brain and cord after the bones and fibrous tissues, 17 deaths occurred. On the other hand, among 51 cases in which the brain was the principal seat of the syphilitic changes, death only occurred in 12. But the remaining 39 cases must not be regarded as definitely cured, since relapses occurred in a portion and, perhaps, in the majority.

Syphilitic mental disturbances offer a very favorable prognosis. But sometimes the syphilis may be recovered from, while the mental disorder persists. It may also happen, as in a case of recovery from dementia paralytica, published by Flemming (Path. u. Ther. d. Psychosen, 1859), that the patient dies four or five years later from secondary syphilis without any return of the psychical disorder. In favorable cases the mental alienation usually lasts several weeks or months (in one case it continued more than two years). Among the fatal cases we may mention the following forms, viz., dementia paralytica, mania, melancholia, and insanity, without paralysis or loss of intelligence, but with voluntary starvation.

Finally, we may add that the motor and sensory disturbances may disappear after apoplectiform or epileptic paroxysms, unless the increasing severity of the latter lead directly to a fatal termination. But if we examine with care we will find slight feebleness of movement, alterations of the temperament, slight disorders of speech, etc. A relapse may occur with apoplectic or inflammatory symptoms, even after the cerebral symptoms have disappeared for several years. The patients usually die from the cachexia or from an extension of the diathetic disorders. Taken all in all, therefore, the prognosis of cerebral syphilis must be regarded as very doubtful.

Treatment.

The treatment consists in antisyphilitic measures, regulated according to the condition of the patient. The majority of observers prefer the external and internal administration of mercury, generally in the form of the iodides. In opposition to the view that tertiary symptoms are caused by mercurialism, we may state that distinct symptoms of cerebral syphilis have been observed in patients who have never taken mercury.

Convalescents whose nutrition has suffered from the disease itself and from prolonged antisyphilitic treatment, derive great benefit from the cautious employment of hydrotherapeutic measures (moist frictions, the use of wet blankets until the return of heat, followed by half-baths whose temperature is progressively lowered). Electricity may be employed to relieve such paralyses as may have remained permanent.

CLASS II.

DISEASES OF THE MEDULLA OBLONGATA.

I.—DISEASES OF THE MEDULLA OBLONGATA.

CHAPTER XIV.

The medulla oblongata, situated between the posterior portion of the pons and the inferior termination of the olivary bodies, is intimately connected, by its structure and vessels, with the adjacent portions of the brain and spinal cord.

We shall first say a few words on the special functions of the medulla oblongata, so far as they have a bearing upon pathology. In accordance with the most recent researches, we need no longer admit the existence of circumscribed medullary centres for the various reflex acts. The transmission of reflex actions is accomplished by much more extensive apparatus, on account of the wide-spread communications in the spinal cord between the motor and sensory paths. Even when the reflex centres of the medulla or of the brain have been removed and reflex excitability is increased by the administration of strychnine, reflex actions may nevertheless be produced. Prokop Rokitansky (Med. Jahrb., I. H., 1874) has ascertained that the spinal cord contains respiratory centres below the fourth ventricle. Wilh. Schlesinger (eod. loc.) has shown that the centres of the vascular and uterine nerves are also contained in the floor of the same ventricle. The centre of the vascular nerves contains a centre of excitation and one of depression. They may be stimulated by reflex means by acting upon the brain, or directly by acting upon the blood. The centre for the arrest of the heart's action, which corresponds to the fibres of the pneumogastric and is merely a reflex centre, is also situated in the medulla oblongata. The centre for the retardation of the heart's action, the fibres of which pass through the cervical sympathetic, is also contained in the medulla. The convulsive centre of Nothnagel is contained in the pons varolii, but on account of the intimate relations of the inferior border of the pons with the adjacent border of the medulla oblongata, it is probable that the latter participates during life in the production of the convulsive phenomena. The medulla contains the fibres of origin of the sensitive cranial nerves and the fibres of the tegmentum of the peduncle which are interrupted, at this level, by cells which serve for the transmission of impulses to the motor fibres. Finally, the medulla

oblongata also contains the centres of pupillary dilatation, of the movements of deglutition and mastication, of the salivary secretion, and the centre of synergic movements of the body (the latter depend, to a great extent, upon the pons and tubercula quadrigemina).

a. *Anæmia and Hyperæmia.*

Direct observation is very poor in materials bearing upon bulbar anæmia. It affects by preference the gray substance (which is richer in capillaries) and which becomes markedly paler than normally. The vessels of the pia mater are moderately distended with blood, but the superficial nerve meshes are strongly congested. The different regions of the medulla may be unequally affected by the anæmia, according to the condition of the vessels.

In certain diathetic diseases, the bulbar anæmia merely runs parallel to the cerebral and spinal anæmia. Under the influence of trifling causes, the patients become subject to dyspnœa, palpitation, nausea, spasms and prostration, owing to the morbid excitability and exhaustion of the bulbar and spinal centres. Irritation of the centre of the vascular nerves may arise from certain toxic agents (opium, chloroform, ergotine, nicotine, etc.) or from psychical excitement, as in hysteria, and may produce symptoms of bulbar and cerebral anæmia, with slowness of the pulse and respiration, spasmodic contraction of the cutaneous vessels, lowering of the temperature, loss of consciousness, etc. In epilepsy, judging from experimental data and clinical observation, we must consider the medulla oblongata as the point of origin of the vascular spasm.

Partial anæmia of the medulla oblongata may be the result of compression of large capillary zones by extravasations, abscesses, tumors, etc. Thrombosis and embolism may give rise to circumscribed anæmia and paralyses, from arrest of the arterial circulation.

The symptomatology varies, as Duret has shown, according to the point at which the obliteration exists, the nutrient arteries being distributed without anastomoses to distinct territories of the medulla oblongata. If the embolus occupies one of the vertebral arteries, it interrupts the circulation in the anterior spinal artery and, consequently, in the median arteries which it supplies, *i.e.*, in the nutrient arteries of the nuclei of the hypo-glossal, spinal accessory and inferior facial nerves. The symptoms of labio-glosso-pharyngeal paralysis will then suddenly develop.

Cases of this nature have been observed by Charcot, Joffroy (Gaz. méd., 1872), and Proust (Compt. rend. de la Soc. de Biol. 1869). In the last of these cases, hemiplegia occurred, with sudden paralysis of the face and tongue, and then complete aphonia and dysphagia developed, rapidly followed by death. At the autopsy obliteration of the superior half of the vertebral artery and the lower cerebellar artery was found, with softening of the corresponding cerebellar lobe.

The paralysis, according to Duret, is complete or incomplete, according as a single anterior spinal artery or two symmetrical ones, originate from the obliterated vertebral. In the latter case the lesion is unilateral. Hemiplegia is due to obliteration of one of the two arteries which supply the pyramids or the restiform bodies. Obliteration of the basilar artery, afferent vessels from which supply the pons, may cause paralysis of all the limbs. If the embolus is prolonged into the basilar artery, the sudden anæmia of the nucleus of the pneumogastric may cause sudden death. A

clot in the lower part of the basilar artery produces obliteration of the inferior and middle arteries of the pons, which supply the nuclei of the pneumogastric and glosso-pharyngeal nerves, causing rapidly fatal symptoms of cyanosis with stertorous breathing, rapid pulse, and relaxation of all the limbs. An embolus in the upper part of the basilar produces, according to Duret, disorders of vision, strabismus, ptosis, and paralysis of the superior facial nerve.

When the embolic lesions in the medulla oblongata quickly retrograde by the re-establishment of the collateral circulation, or by the disappearance of the embolus, the morbid symptoms may yield little by little. If the disorders of nutrition persist, spots of softening will form in the medulla and pons. They are usually situated in the median line and are triangular in shape, the base being directed towards the fourth ventricle along the course of the median arteries of the medulla.

Hyperæmia of the medulla oblongata is unknown except from a few observations upon the cadaver. This hyperæmia (with considerable dilatation of the vessels, especially in the posterior half of the cord, Schroeder v. d. Kolk) occurs in epilepsy, in infantile eclampsia or in eclampsia from intoxication, in hydrophobia, tetanus, etc. It is usually accompanied by intense congestion of the meninges and adjacent portions of the spinal cord. Although a large part of the blood found after death in the superior and inferior portions of the spinal cord is due to cadaveric hypostasis, nevertheless recent experimental investigations have shown that considerable hyperæmia of the cord may occur, especially in convulsive affections. Thus Landois (Centralbl. f. m. Wiss., 10, 1867) found in rabbits (in whom intermittent ligature of the superior vena cava had induced epileptiform attacks) a considerable venous hyperæmia of the tubercula quadrigemina, medulla oblongata, and spinal cord. Magnan (Arch. de Physiol., Mrch., 1873) saw intense congestion of the medulla oblongata and meninges in dogs who had become epileptics from the administration of absinthe.

Bulbar hyperæmia frequently accompanies embolic, hæmorrhagic, and inflammatory processes, and tumors which develop in the neighborhood of the medulla. It also occurs in severe typhoid fever, acute exanthemata, cerebo-spinal meningitis and fatal forms of chorea.

Temporary congestions are observed in the living from depression of the vaso-motor centres. In these cases the redness, heat, and sweating of the extremities, and the periodical hyperæsthesia evidently originate in a medullary lesion. Certain temporary symptoms of excitation, which appear as the first signs of bulbar paralyses, must be ascribed to circumscribed congestions (of the nuclei or nerve roots). Finally, in a case of typhoid fever (Hesky, Med. Presse, 1869), in which Cheyne-Stokes' respiration had been observed during life, very noticeable hyperæmia and firmness of the medulla oblongata were observed, especially upon the floor of the fourth ventricle, with hyperæmia of the neurilemma of both pneumogastrics.

b. *Apoplexy of the Medulla Oblongata.*

Medullary hæmorrhages may be *secondary*, from the extension of considerable hæmorrhages in the central ganglia towards the median line, with afflux of blood into the lateral, middle and fourth ventricles. They often follow hæmorrhages into the pons and cerebellum, which reach the base of the brain after destruction of the medullary substance. *Primary*

medullary hæmorrhages are caused by local lesions or by diseases of the medulla oblongata and cervical portion of the spinal cord.

The most important causes of spontaneous hæmorrhages into the medulla oblongata are the diseases of the afferent vessels.

Arteritis of the basilar artery is followed by thrombosis and then by foyers of softening and hæmorrhagic infarctions. We know that the symptoms vary according to the site of the obstruction. Aneurisms of the basilar artery are also a frequent cause of hæmorrhages into the pons and medulla oblongata. According to Lebert (Berl. klin. Wschr., 1866), among thirty-one cases of aneurisms at the base, vascular ruptures and hæmorrhages occurred seventeen times into the parenchyma of the pons and medulla oblongata, giving rise to the following symptoms, viz.: occipital headache, stiffness of the neck, vertigo, nausea, vomiting, noises in the ears, feeble hearing, disorders of speech, deglutition and respiration, and hemiplegia or paraplegia.

Traumatic lesions of the medulla oblongata are often followed by abundant hæmorrhages. Waters' patient (Med. Chir. Transact., XLVI., 1863), 23 years old, having received a violent blow upon the left side of the face and neck, manifested the following symptoms: at first temporary loss of consciousness, then embarrassment of speech, inability to swallow; incessent hiccough, deafness, incomplete right hemiplegia with elevation of temperature on the paralyzed side, dilatation of the pupils, nystagmus, accelerated pulse (up to 100). The patient died upon the second day after the receipt of the injury. At the autopsy a hæmorrhage was found in the upper part of the medulla oblongata, extending under the pia mater to the right and thirty-two centimetres downward. A transverse fissure was situated above, involving the restiform body and a part of the floor of the fourth ventricle, and a second one behind, reaching to the posterior pyramid and column. In another case, published by Leyden (Virch. Arch., Bd. 55), epilepsy and paresis of the legs with slowness of movements developed after a blow upon the head. These symptoms indicated a hæmorrhage into the medulla oblongata and cervical portion of the cord.

In rarer cases bulbar hæmorrhage may be due to caries of the first cervical vertebræ with suppurative meningitis and hæmorrhage into the medulla, as in Fullers' case (Lancet, Sept., 1870); or to tumors developing in the neighborhood of the medulla oblongata, which are sometimes accompanied by considerable hæmorrhages. We may finally mention that hæmorrhages into the adjacent portions, extending as far as the medulla oblongata, may give rise to some bulbar symptoms. In Desnos' case (Union med., 20, 1869), the patient was suddenly seized during sleep with extreme dyspnœa, crepitant râles in both lungs, followed by vomiting, contraction of the pupils, relaxation of the limbs, albumen in the urine, coma and death at the end of twenty-four hours. The diagnosis of uræmia was made. At the autopsy a hæmorrhagic extravasation as large as a hazel-nut was found in the posterior and superior portion of the pons, with perforation into the fourth ventricle. The kidneys were normal. The symptoms of bulbar apoplexy present considerable differences according to the origin and situation of the extravasation. The very large extravasations which extend from the central ganglia, ventricles or base of the brain to the medulla oblongata, give rise to violent symptoms of apoplexy, to coma and to rapid death. In only a few cases have certain final symptoms indicated the implication of the medulla oblongata.

In many bulbar hæmorrhages epileptiform spasms develop after a temporary loss of consciousness. This is confirmed by the recent experiments

of Westphal (Berl. klin. Wschr., 38, 1871). In guinea-pigs which had been rendered epileptic by blows upon the head, he found small, irregular extravasations, disseminated throughout the white and gray matter of the medulla oblongata and cervical cord, and collections of blood in the cavity of the spinal dura mater. Convulsions are not mentioned in some cases of bulbar apoplexy. Their existence depends on certain hitherto unknown localizations.

Other motor disturbances appear in bulbar apoplexies, such as more or less complete hemiplegia with diminution of sensibility. The paralysis sometimes assumes the paraplegic type or may extend, especially in fatal cases, to all the limbs. We may also mention disorders of the movements of the tongue, of speech and deglutition, the deviation of the palate and uvula, frequent and usually persisting hiccough, and finally, spasms in the muscles of the face, eyes, and neck.

In addition to dyspnœa, the Cheyne-Stokes' respiration often appears as a characteristic symptom of hæmorrhages into the medulla oblongata. This phenomenon consists of certain irregularities of respiration occurring in a perfectly regular order. From time to time the inspirations cease for a quarter or even an entire minute. They then begin gently, become more and more rapid and profound, and finally grow weaker and again cease. Schiff has found (Nervenphysiologie, 1859, p. 324), in his experiments upon animals, that the smallest hæmorrhage into the medulla or the slightest pressure exerted upon it, renders the respiration more infrequent and difficult. The animals presented the same alternation of diminution and increase of respiration, separated by periods of complete arrest.

Cheyne-Stokes' respiration which may be due to hæmorrhages, effusions, and tumors of the base, extending to the medulla oblongata (Traube, Merkel, Mader), is produced, according to Traube (Berl. klin. Wschr., 1869 and 1874), by an insufficient supply of arterial blood to the medulla oblongata. "The irritability of the respiratory centre being thus lessened, the normal quantity of carbonic acid present does not suffice to provoke an inspiration. In order that this shall be produced, considerable quantities of carbonic acid must collect, and in order that this latter event may occur, the intervals must be longer than in the normal condition. Hence the prolonged suspension of respiration. The carbonic acid necessarily accumulates at first in the pulmonary circulation, from whence a primary excitation of the respiratory centre occurs through the pulmonary fibres of the pneumogastric nerves. Later, the carbonic acid also accumulates in the general circulation, and the activity of the medulla oblongata is then called forth by the sensory nerves of other portions of the body."

We may finally add that, in considerable extravasations extending into the fourth ventricle, polyuria has been observed by Potain (Gaz. des Hôp., 1862), and albuminuria by Desnos (*loc. cit.*) and by Mader (Med. Wschr., 1869). We also know, from the experiments of Claude Bernard, that in lesions of the fourth ventricle, situated above the acoustic nucleus, the urine is secreted in small quantity but contains albumen.

The diagnosis of an apoplexy of the medulla oblongata cannot be established except in those cases in which an apoplectiform attack is accompanied by certain characteristic functional disturbances. These are: epileptiform attacks occurring either in the beginning or later; when consciousness is restored, signs of incomplete hemiplegia or paraplegiæ, with preservation of electro-muscular contractility and simultaneous disorders of sensation (at first hyperæsthesia, then circumscribed anæsthesia); dysphagia, repeated vomiting, hiccough, embarrassment of speech, deflection

of the velum palati and uvula. Independent of all these symptoms we must also, in making a diagnosis, take into consideration the intense dyspnœa (without material cause), the Cheyne-Stokes' respiration and albuminuria. In large hæmorrhages originating in the cerebral ganglia or ventricles, death occurs in coma at the end of a few hours, the bulbar symptoms being imperfectly defined.

Experience teaches that the prognosis is more favorable in traumatic cases than in those in which the hæmorrhage is due to disease of the vessels of the brain or base (especially frequent in old age). The prompt return of consciousness, the alternation of the paralytic symptoms, and the daily improvement in the symptoms, afford indication of gradual amelioration. But unexpected complications, fresh hæmorrhages, or inflammatory processes, may again place the patient's life in jeopardy. Even in the favorable cases in which the more serious symptoms disappear we may find paralyses and partial atrophy, perhaps after the lapse of weeks or months.

Venesection is rarely indicated in bulbar apoplexies (except in cases of very marked stasis, and in robust persons). We prefer local bleedings from the mastoid processes or neck. We should then continue the application of ice-bags to the occiput and neck for a long time. In addition, absolute rest, reduced diet, and regulation of the secretory functions are indicated. We may employ cool baths, hydrotherapeutics and electricity to combat permanent paralyses.

CHAPTER XV.

INFLAMMATIONS AND TUMORS OF THE MEDULLA OBLONGATA.

INFLAMMATIONS of the medulla oblongata, like all other processes of a similar nature, may terminate in diffuse suppuration or plastic exudation, or they may give rise to special morbid types by producing changes which are limited to the origins of the nerves in the medulla. We shall, therefore, first discuss the simple inflammatory processes and their various sequences, and shall then investigate an affection, presenting very peculiar symptoms and due to inflammation and successive degeneration of the different nuclei of origin in the medulla, and which has been the subject of important researches during the last few years.

a. *Inflammations and their sequences.*

Like the brain and spinal cord, the medulla oblongata is very frequently the site of inflammatory processes, due to traumatism, arteritis, thrombosis, tumors, etc., or conveyed from adjacent parts. Thus, in Abercrombie's case (l. c., Obs. 39), a child sixteen months old, who had fallen, three months previously, upon the occiput or neck, suffered from strabismus, right hemiplegia, intermittent convulsions, a pulse at first accelerated and then retarded, and obstinate constipation; intelligence remained intact. At the autopsy a purulent focus enclosed in a capsule was found in the parenchyma of the medulla oblongata, at its intersection with the pons varolii. The diseased process presented the appearances of a scrofulous abscess. Lebert has more recently observed a case of abscess of the medulla oblongata, in which the symptoms during life consisted of strabismus, a succession of paralyses upon the right side, convulsions, and disturbances in defecation. Several nerve nuclei were implicated in the purulent focus and, furthermore, the continuity of the motor columns was interrupted at different points although not completely abolished.

In a case published by Meynert (Prakt. Heilk., 1863), a woman, twenty years old, after suffering five days from malaise, was attacked with violent headache, slight ptosis, drooping of the right labial commissure, difficulty in the articulation of words, profuse salivation, and pulse accelerated and then slowed, without paralytic symptoms or disturbances of intelligence. The patient died at the end of two days. At the autopsy, in addition to suppurative basilar meningitis, an abscess as large as a hazelnut was found in the left half of the pons, with perforation of the fourth ventricle in the neighborhood of the nucleus of the facial and of the motor nucleus of the trigeminus. The motor fibres which, starting in the spinal cord, pass upwards through the longitudinal fibres of the pons, were intact. The paresis of the levator palpebræ was caused by suppuration of the posterior perforated space, between the points of emergence of the motor oculi nerves.

We must also mention among the consequences of inflammation, the formation of spots of softening, such as we have previously described, and also the development of connective tissue proliferation which usually affects the white substance of the medulla oblongata and spinal cord. Sometimes, as in tetanus, a viscous substance is produced which is rich in cells, and, at others, as in labio-glosso-pharyngeal paralysis, proliferation of the neuroglia occurs with numerous disseminated, granular cells. In cerebro-spinal sclerosis these form multiple foci, extending throughout the cerebro-spinal axis, and embracing the different columns, and even the nerve nuclei of the medulla, in the hyperplastic process.

These inflammatory processes, which usually extend beyond the medulla, cannot be recognized, even by their remote consequences, except in a small number of cases, in which epileptiform convulsions and other functional disorders already mentioned, indicate the participation of the medulla oblongata in the disease of the nervous centres.

b. *Labio-glosso-pharyngeal Paralysis* (*Paralysis of the bulbar nuclei, Nervenkernelaehmung of the author*).

The disease in question, although recognized in its principal characteristics by Trousseau (1841) and by Dumenil (Gaz. hebdom., June, 1856), was first described as a distinct morbid process by Duchenne (Arch. génér., Sept., Oct., 1860) and was called by him labio-glosso-pharyngeal paralysis. At a later period Wachsmuth, in a monograph which he devoted to the consideration of this disease (Ueber progressive Bulbaerparalyse und Diplegia facialis, Dorpat, 1864), succeeded, by means of induction, in associating its symptomatology with successive destruction of the nerve nuclei of the fourth ventricle and with secondary degeneration of the peripheral nerve trunks. But it needed other and more recent investigations to fully clear up the anatomical characters of the disease and its relations to certain allied forms. I have proposed to substitute the simpler and more exact title of paralysis of the nerve nuclei or bulbar nuclei for the complex terms hitherto employed.

Pathological Anatomy.

The most evident and long since recognized lesions are the atrophy of the cranial nerves which were paralyzed during life. In addition to atrophy of the roots of the hypoglossal and facial nerves, recognized by Trousseau, Dumenil, and Wachsmuth, the roots of the accessory, glosso-pharyngeal and pneumogastric nerves have, more recently, been found flattened, transparent, grayish, and in a condition of fibrous degeneration (Charcot, Leyden, Hun, etc.). The corresponding nerve trunks, sometimes, also present an atrophic degeneration in a centrifugal direction.

It is only since the most recent researches of Charcot and Joffroy (Arch. de Physiol., 1870), of Duchenne (Arch. génér., 1870), of Hun (Amer. Journ. of Insanity, 1871), and of Leyden (Arch. f. Psych., II. u. III. Bd., 1870 and 1872), that attention has been directed to changes in the nuclei of origin of the bulbar nerves situated in the fourth ventricle. Of the nerve nuclei arranged like a mosaic upon the floor of the ventricle, the degeneration is chiefly found in the nucleus of the hypoglossus (situated immediately in contact with the median line), when the disease

begins in the inferior half. When the process extends, it reaches the nuclei of the pneumogastric and spinal accessory, which are situated more to the outside. The nucleus of the glosso-pharyngeal, which is situated entirely to one side, is usually unaffected. If the disease passes beyond the transverse limit marked out by the medullary (acoustic) striæ, it will involve, in the upper part of the fourth ventricle, the nuclei of the facial and external motor-oculi nerves. The acoustic nucleus, situated more to the outside, and the motor nucleus of the trigeminus, hidden further upwards in the fossa cærulea, generally appear intact. The latter was found degenerated once by Duchenne and the sensory nucleus of the trigeminus in one case by Charcot.

The ganglion cells of the nerve nuclei then lose their stellate form and become shrunken, smaller and of a dull ochre color. The prolongations and nuclei are rudimentary or even completely atrophied. The cells are filled with pigment and granular matter, the nucleus and nucleolus present a vitreous, shining appearance (yellow degeneration of Charcot), and are separated from one another by large spaces. According to the latter author, the ganglion cells are ten to twelve times less frequent than in the normal condition. In the majority of cases similar degenerations are found in the cells of the anterior horns of the spinal cord, together with intense vascularization. The posterior horns do not take part in these changes, with the exception of Kussmaul's case (Volkmann's Samml. klin. Vortraege, 1873), in which Maier found some granular cells in the posterior horns of the cervical cord.

The hyperplasia of connective tissue, which is even found in the floor of the fourth ventricle, extends in a downward direction, along the internal portion of the anterior columns and the posterior part of the lateral columns, as in atrophic degeneration of apoplectic origin (Tuerck). In an upward direction it sometimes affects the fibres of the pyramids and their prolongations in the pons and cerebral peduncles (Leyden, Maier). The olivary bodies are more rarely involved, the posterior columns and the restiform bodies which are derived from them, escaping degeneration. The neuroglia undergoes proliferation and encloses atrophied nerve-tubes, filled with fatty granulations and with swollen axis cylinders. When the latter are atrophied, we only find pale nerve fibres, deprived of myeline, mixed with amyloid corpuscles and large stellate cells (hypertrophy and infiltration of the reticulum of the neuroglia). The vascular walls are thickened and strewn with fatty granules and shining nuclei, which are also found, according to Maier, in the vessels of the central gray masses with consequent diminution of their lumen. It is rare that these changes are confined to the motor nuclei, as in Charcot's, Duchenne's, and Joffroy's cases. In the majority of cases the sclerotic process and the vascular lesions accompanying it, secondarily affect the nervous tissue and nuclei.

The muscular tissue itself is more or less changed. The muscles present a whitish-red appearance and their fibres are either markedly thinned with preservation of the striæ, or they are affected with fatty or waxy degeneration. Considerable fatty tissue is found between the muscular fibres, especially in the tongue. Proliferation of the nuclei of the sarcolemma, and of the interstitial tissue, in addition to granular infiltration of the fibres, are found, according to Charcot, in the emaciated muscles of the tongue, pharynx, and larynx.

Etiology.

Cold, violent emotions, exaggerated muscular efforts, traumatic concussions of the head, and syphilis (Cheadle and Silver) are mentioned as causes of primary labio-glosso-pharyngeal paralysis. The large majority of cases occurred in patients from forty to seventy years of age, and few examples have been observed above or below these limits. The male sex is much more frequently affected. Since the etiological factors may affect all professions alike, we can readily comprehend why the disease occurs in all classes of society. Heredity has exerted no influence in any case hitherto reported. In the secondary forms, which we shall discuss further on, the pathogeny depends upon the primary disease.

Symptomatology.

The prodromata of labio-glosso-pharyngeal paralysis sometimes consist of pains in the head and neck, a feeling of compression in the chest and throat, especially in speaking, and rarely of apoplectiform attacks. Generally, the course of the disease is very insidious. The patients appear to be in good health, and the first disturbances in the movements of the tongue pass unnoticed. Little by little the patient encounters more and more difficulty in speaking and eating. These two functions are accomplished with unusual effort and induce considerable fatigue. The disorder of speech is especially noticeable in the pronunciation of those consonants which are uttered by expelling the expired air, when the anterior part of the tongue is applied against the upper incisors or the anterior portion of the arch of the palate (d, t, l, n, s), or in the pronunciation of those consonants which are essentially produced by the approximation of the posterior halves of the tongue and palatine arch (k, g, and the aspirate letters ch, j). At a later period paralysis appears in the territory of the facial nerve. The movements and occlusion of the lips become difficult, together with the pronunciation of the labials (b, p, f, v, w, m) and of the vowels o and u. The patient can no longer whisper, suckle, whistle or kiss. Paralysis of the muscles of the velum palati and of the pharynx then develops, with inability to close the nasal fossæ, nasal twang of the voice, frequent regurgitation of liquids and, at a more advanced stage, dysphagia. The latter symptom varies in its character according to the degree of paralysis of the tongue and inferior muscles of the pharynx. At the beginning of the paralysis of the constrictors of the pharynx, the patient endeavors, by violent efforts of deglutition, to swallow the portions of food which are retained in the mouth and gullet, and the arrest of large pieces of food may place him in danger of suffocation. If the tongue, which presides over the formation and insalivation of the alimentary bolus, is still able with its root to cause complete occlusion by lowering of the epiglottis, and if the larynx is not yet paralyzed, the patient can readily cause soft and liquid fluid to pass into the pharynx. But if the paralysis prevents the proper closure of the larynx, demonstrated by Bruns by means of the laryngoscope (contact of the arytenoid cartilages with lateral narrowing of the larynx), then solid particles are better swallowed than liquids. The latter readily penetrate into the larynx and provoke violent cough and a paroxysm of suffocation. The succession of the symptoms is inverted in some cases, and an early paralysis is ob-

served in the muscles of the velum palati and pharynx, followed later by disorders of speech and respiration (Duchenne, Eulenburg).

The progressive disturbances in the articulation of sounds, in deglutition and in the movements of the lower jaw, are accompanied by an abundant and troublesome salivation, which is not explained by the dysphagia alone. Increase of the salivary secretion is not alone of a paralytic character, as Cl. Bernard has observed after experimental section of the chorda tympani ; we must rather attribute it, on account of the character of the primary disease, to irritation of the salivary centre discovered by Gruetzner (Pflueger's Arch. f. Phys., 7. Bd., 1873) in the medulla oblongata, in consequence of central irritation of the fibres of origin of the chorda tympani and sympathetic nerves. In most cases the paralytic disturbances of speech and deglutition are also accompanied by feebleness and paralysis of the voice, terminating finally in its complete extinction. Laryngoscopic examination reveals paralysis of the vocal cords (Gerhardt, Fauvel, Ziemssen). In addition to this paralysis I have twice been able to detect the coexistence of anæsthesia of the pharynx and larynx, and this may, in exceptional cases, constitute one of the initial symptoms. In two of Kussmaul's patients, who were unable to speak but were still capable of moaning or laughing, the vibrations and immobilization of the vocal cords were abolished, and the glottis appeared like a gaping opening of an elliptical shape. Mechanical hyperæmia and catarrh may be produced by the penetration of mucus and débris of food into the larynx. Even the bronchi and lungs may be seriously implicated from the same causes.

While the paralytic disorders are increasing in the domain of the hypoglossal, facial, and spinal accessory nerves, the continued progress of the affection also terminates in functional disorders of the pneumogastric nerve. These consist of attacks of dyspnœa, occurring after exertion or even during the night, and they may result in sudden death. The slowing of the pulse, which is sometimes observed (according to Duchenne), and the sudden and fatal arrest of the heart's action, are due to irritation of the pneumogastric nerve. On the other hand, the final irregularity and excessive acceleration of the heart, observed by Mignard, indicate paralysis of the motor filaments of the pneumogastric nerve of the nuclei situated in the upper half of the fourth ventricle ; the disease especially attacks the inferior ganglion cells of the facial, and those of the motor portion of the trigeminal. The nuclei situated laterally and more anteriorly are more rarely affected by the extension of the lesions, though Eulenburg has observed paresis of one of the external motor oculi nerves and progressive feebleness of audition among the initial symptoms. Hérold has twice seen unilateral paralysis of the motor oculi communis. In certain cases the paralysis predominates in the upper branches of the facial nerve, or facial diplegia develops from lesions of the superior cells of the nucleus of this nerve. If the degeneration extends backwards it will involve the fibres of the pyramids. This gives rise to paralyses of the upper and lower limbs, of the muscles of the trunk, of the sphincters, and even causes impotence (as in one of my patients). The sensory disturbances consist, at the onset, of pains in the forehead, occiput or neck, of diminution of sensibility in the hands or legs, upon the mucous membrane of the mouth and fauces, and even complete abolition of reflex excitability. The intelligence and functions of special sense are unaffected; the appetite and digestion are also unimpaired. Fever does not develop unless caused by some complication.

The electrical and reflex excitability of the paralyzed muscles varies

according to the intensity of the nervous and muscular degenerations. The electro-muscular contractility was normal in the majority of cases observed by Duchenne, Leyden, etc., and by myself. Nevertheless, I have sometimes found it markedly diminished and, in some cases, it has been even completely abolished (Wachsmuth, Benedikt). Galvanic irritation may produce movements of deglutition by reflex means or through the medium of the hypoglossal nerve. Galvanization of the facial is followed, according to Schulz, by arrest of the salivary hypersecretion. Labio-glosso-pharyngeal paralysis may occur as an affection restricted to the bulbar nuclei, or it may follow, as a secondary process, other degenerations which have originated in the cervical cord or gray matter of the cord. The symptoms of labio-glosso-pharyngeal paralysis sometimes appear, during the course of the primary affection, in dementia paralytica, in disseminated cerebro-spinal sclerosis, and in softening of the pons and cervical cord, with secondary atrophy of the medulla oblongata (Gerhard's case, after injury to the skull).

In progressive muscular atrophy, which is caused, as we shall see further on, by degeneration of the cells of the anterior horns, the morbid process may extend through the cervical cord and involve the nuclei of origin situated in the floor of the fourth ventricle, or the disease, beginning in the bulbar nuclei, extends to the anterior horns, thus explaining the frequent complication with secondary muscular atrophy. The nervous lesions give rise then, according to their ascending or descending direction in the gray columns of the cord (or, according to Gerlach, in the gray meshwork of nerve fibres), to all the symptomatic forms of one and the same morbid process, the grouping and succession of symptoms depending upon the different points of origin and on the combinations of the lesions. Pathological anatomy and clinical observation corroborate the truth of this statement.

At a more advanced stage of the disease symptoms of inanition develop (nutrition being impaired and the patient being unable to satisfy his hunger), with general emaciation and muscular relaxation, which render all movements difficult. In the majority of cases severe medullary symptoms develop at a later period. The rachialgia and periodical neuralgias in the limbs are often the forerunners of the atrophy and paralysis. When the cervical cord is first involved, the lesions affect the thenar and hypothenar eminences, the interossei muscles, then the tongue (with fibrillary tremors) and the rotators and flexors of the head, which then falls upon the chest. When the lesions extend upwards the paralysis involves the diaphragm, the muscles of the trunk and of the upper and lower limbs, in which contractures are also produced. This association of bulbar and spinal paralyses completes the sad picture of the disease in its most severe form. Incapable of speaking and moving, the unhappy patient must be fed by others, and is irretrievably lost. He can manifest his mental sufferings with the eyes alone, and presents the most terrible picture of human decline conceivable. The disease may last from one to three years. The patients die from suffocation, marasmus, inanition, apnœa, paralysis of the diaphragm or heart, or from pulmonary hypostasis.

Diagnosis and Prognosis.

The paralysis of the bulbar nuclei usually begins with slight and isolated symptoms, such as pains in the head or neck, slight embarrassment

of speech or deglutition, a feeling of stiffness in the lips and, more rarely, paroxysms of dyspnœa or apoplectiform attacks. These symptoms do not furnish sufficient grounds to permit a diagnosis of the grave disease which is developing. The steady progress, and especially the succession of disturbances of the tongue, lips, and deglutition, reveal to the physician all the dangers which threaten his patient.

We must carefully consider all the symptoms, and not make a hasty diagnosis of labio-glosso-pharyngeal paralysis, since disorders of deglutition, of movements of the tongue and of speech, with marked salivation, may exist in hysteria, insanity, disseminated sclerosis, and in other localized lesions (from alteration of the nuclei and roots of the nerves), and since these forms of disease may be recovered from. The case must be watched with great care and for a long period. The final appearance of motor and sensory disturbances characteristic of hysteria, of insanity, of cerebral and spinal paralyses, of disorders of the senses, etc., enables us to recognize the disease with which we have to deal.

Symptoms of labio-glosso-pharyneal paralysis may occur incidentally in irritation of the cervical cord. Thus, I observed a case of commencing ataxia, with neuralgic pains in the neck and arms, intermittent sciatic, girdling pains, and rapid fatigue. Inability to swallow solid food (probably of spasmodic origin) was also present in the beginning, with embarrassment of speech, paresis of the lower facial muscles on the left side, and abundant salivation on certain days. This condition lasted about six months, being undoubtedly due to persistent hyperæmia of the medulla oblongata, and completely disappeared under electrical and hydropathic treatment directed against the spinal affection. The latter persisted, however, its symptoms becoming aggravated, and it continues to the present day.

Embolism of one of the vertebral arteries may also give rise to symptoms of labio-glosso-pharyngeal paralysis, from obliteration of the anterior spinal artery and of its median branches which supply the nuclei of the spinal accessory, hypoglossal, and the inferior nucleus of the facial nerve (as Duret has shown). In a case of this kind published by Proust (Comptes rendus de la Soc. de Biol., 1869), the autopsy revealed obliteration of the upper half of the vertebral artery and of the inferior cerebellar artery, with softening of the corresponding cerebellar lobe. In an analogous case published by Leyden (Congress of German Naturalists, Innsbruck, 1869), a hemorrhage, with softening of the pons and medulla, was found upon autopsy. The vessels were seriously diseased, and the atrophied nervous elements were replaced by compact fibrous tissue. Joffroy's patient (Gaz. méd., 1872) recovered. The differential diagnosis in all these cases depended upon the sudden (apoplectiform) appearance of the symptoms of labio-glosso-pharyngeal paralysis, upon the presence of hemiplegia, anæsthesia, variations in the paralyses, and the coexisting disorders of sight and hearing. Tumors and circumscribed inflammatory processes at the base of the brain may also during life assume the appearances of labio-glosso-pharyngeal paralysis, on account of the compression of the roots of several bulbar nerves. But in tumors, which we shall discuss further on, we find, in the beginning, neuralgia followed by anæsthesia of the trigeminal nerve, clonic convulsions of the muscles of the face and tongue, disorders of smell and audition, the voice remaining intact—all of which symptoms are not found in the classical history of labio-glosso-pharyngeal paralysis properly speaking. The characteristic signs in multiple paralyses of the cranial nerves, due to circumscribed meningitis or

periostitis at the base (as in Graefe's and Virchow's cases), consist of chronic cephalalgia, vertigo, ptosis, various ocular paralyses, and final disturbances of deglutition and respiration.

The chief symptoms of the diseases of the posterior cerebral fossæ consist, in addition to disorders of deglutition and of the articulation of sounds, of enfeebled hearing, nystagmus, paralysis of the external motor oculi, staggering gait, and paresis involving all the limbs. In the varieties which are due to syphilis (Cheadle's and Silver's case), the previous history, the hemiplegic disorders appearing from time to time, and the rapid cessation of all the symptoms upon the employment of iodide of potassium, will suffice to enable us to recognize the real character of the affection. Finally, bulbar facial diplegia is readily distinguished by the double peripheral paralysis of the face. In bulbar facial diplegia, when limited to the lower, or more rarely to the upper muscles of the face, the characteristic signs consist of the gradual appearance of the paralysis, dissociation of the movements of the face, the coexistence of paralyses of the tongue, lips, pharynx, and jaw, profuse salivation, aphonia, symptoms of paralysis of the pneumogastric, and persistent difficulty in deglutition, the nose being closed (on account of the weakness of the tongue). On the other hand, double peripheral prosoplegia (facial paralysis) is less serious, and is usually characterized by complete paralysis from the outset (with frequent abolition of faradic and increase of galvanic contractility), by the immobility and stiffness of the features, by the trivial disturbance in the pronunciation of labials, and by the preservation of the movements of the tongue, the integrity of the timbre of the voice, and finally by the diminution of the difficulty of deglutition when the nose is closed (by the action of the air enclosed in the nasal fossæ).

With regard to prognosis, nearly all observers agree in regarding the central lesions of labio-glosso-pharyngeal paralysis as incapable of being arrested in their progress and as inevitably fatal. I have observed twelve cases, some simple, the others complicated by progressive muscular atrophy. Sometimes, in the less advanced forms, a period of arrest and even of improvement is observed, lasting several weeks or months. But the final deterioration and fatal termination will occur, despite persevering treatment by electricity and hydrotherapeutics. But Coppette and Tommasi have seen some cases, and Benedikt has also observed a considerable number of cases of improvement and even of recovery. Kussmaul (l. c.) has submitted observations of this kind to very rigorous criticism. It is impossible for us, at present, to explain how the catalytic and antiphlogistic action, which is attributed to the galvanic current, can occur in these cases, and how it reacts upon lesions occurring in the delicate framework of the nerve elements, either when it is applied to the sympathetic (Charcot and Maier have found this intact, and it does not, therefore, act as a cause in the disease in question), or when applied to the mastoid processes. In the present state of our knowledge, we must restrict ourselves to deploring the inexorable gravity of the facts, without yielding to a deceptive optimism.

Treatment.

In the first weeks or months after the appearance of suspicious symptoms, the measures indicated in robust individuals consist of local bleedings from the neck or mastoid processes and the prolonged application of ice-bags. Then we may employ strong galvanic currents along the

cervical vertebræ and along the course of the hypoglossal nerves (continued daily for weeks and months), producing thereby frequent movements of deglutition. Benedikt recommends galvanization of the sympathetic nerve in the cervical region and through the mastoid processes and, when paralysis of the diaphragm occurs, stimulation of the phrenic nerve. The hypersecretion of saliva may be arrested by hypodermic injections of atropine which produce, according to Heidenhain, irritation of the secretory cells.

Internal medication (nitrate of silver, iron, strychnine, phosphorus) has not produced any notable results. I have obtained in the beginning more lasting and positive effects from cold effusions to the cervical portion of the spine while the patient is in a warm half-bath, and from douches to the anterior and posterior parts of the neck. With regard to the selection of food, we must be careful to administer only soft substances and in small mouthfuls. When the dysphagia is very marked, our only resource consists in artificial alimentation by means of the œsophageal sound. In Blumenthal's case, tracheotomy was performed on account of the intense dyspnœa (of a laryngeal nature). The introduction and continued use of the canula caused the cessation of the attacks of suffocation; the paralysis of deglutition and speech continued its progress.

c. *Tumors of the Medulla Oblongata.*

The medulla oblongata may be compressed or pushed to one side, and is sometimes the seat of hæmorrhages or foci of softening in consequence of the presence of tumors, which may either develop within its substance or from the base of the brain or cerebellum, especially from the middle lobe. The cases collected by Ladame (l. c.) present no characteristic symptoms. It is only within the last few years that some examples of very sharply defined tumors have been observed, which were manifested by symptoms sufficiently clear to make a diagnosis possible and thus furnished important data for the symptomatology of diseases of the medulla oblongata.

In Erichsen's observation (Peters. Zeitschr., 2 H., 1870), the disease was characterized by headache, dilatation of the pupils, nausea, vomiting of food, and obstinate hiccough. At a later period, temporary contractures and insensibility developed in the right arm, with persistent anæsthesia of the right half of the face, aphonia (from paralysis of the vocal cords), paresis of the right half of the velum palati, and, finally, paralysis of the bladder. On account of the simultaneous and distinct affection of the pneumogastric, spinal accessory, trigeminal and phrenic nerves, a diagnosis was made of tumor of the right half of the medulla, and was confirmed upon autopsy. A tubercle, larger than an almond, was found occupying a half of the medulla oblongata, penetrating freely into the fourth ventricle, and extending from the substantia ferruginea to the lower limit of the calamus scriptorius.

Edwards' patient (Brit. Med. Journ., Feb., 1870) suffered from converging strabismus, weakness of the legs, disturbance of deglutition; later, the patient could only drag himself along, his speech was unintelligible, and during the night he gave utterance to groans and moans. Finally, vomiting, hiccough, dysphagia, alalia, abdominal respiration, incontinence of fæces, paralysis of the legs and then of the arms, supervened. At the autopsy, a tumor was discovered occupying the centre of the medulla oblongata. In Voisin's case (Annal med-psych., Jan., 1871), the labio-glosso-pharyngeal paralysis appeared suddenly with salivation, dyspnœa, and loss of taste and hearing. Upon autopsy, two epitheliomata were found upon the anterior and posterior portions of the cerebral hemispheres; the one on the left side, which was as large as a hazel-nut, penetrated between the medulla oblongata and cerebellum and compressed the acoustic, facial, hypoglossal, spinal accessory and glosso-pharyngeal nerves, which

were markedly atrophied. Baelz's patient (Arch. f. Heilk., Bd. XIII., 1872), presented the symptoms of labio-glosso-pharyngeal paralysis, without dyspnœa or aphonia, but with diminished sensibility in the integument of the face and head, all these symptoms being preceded by trigeminal neuralgia and spasms, followed by paralysis of the tongue and labial commissures. At the autopsy, an enchondroma was found at the base of the skull, with compression of and hæmorrhage in the medulla oblongata, which was pushed to the right side. The larger number of the bulbar nerves were in a condition of fatty degeneration, but the ganglion-cells of the nuclei of origin were intact.

Independently of the bulbar symptoms, which we have mentioned, and of labio-glosso-pharyngeal paralysis, which is recognized, in these cases, by its unusual progress and by the coexistence of neuralgia, muscular spasms, anæsthesia, and disorders of the special senses, compression of the medulla oblongata by tumors may also give rise to the Cheyne-Stokes' respiratory phenomenon, which we have investigated above, and to modifications of the urine.

The latter vary, according as the lesion affects this or that centre of vascular innervation in the cord. Perroud has published a case (Lyon. Méd., 23, 1869) of syphiloma of the tonsillar lobe of the cerebellum, with compression of the upper part of the fourth ventricle, in which polyuria had occurred during life. Not infrequently, albuminuria occurs in tumors compressing the medulla oblongata. Diabetes is a characteristic symptom of tumors of the fourth ventricle, which are manifested by signs of compression of the adjacent parts (tubercula quadrigemina, cerebellum, medulla oblongata).

Sugar appeared in the urine in Recklinghausen's case (Virch. Arch., XXX. Bd.) of fibrous tumor of the choroid plexus of the fourth ventricle. Mosler has since published another case (Virch. Arch., XLIII. Bd.) of vertigo, periodical headache, especially in the occiput, vomiting persisting an entire year, and diabetes insipidus. At the autopsy, a hard tumor, as large as a walnut, was found in the fourth ventricle, extending anteriorly to the opening of the aqueduct of Sylvius, and filling up the ventricle posteriorly. The cerebellum and medulla oblongata were indented at this point. A microscopical examination of the tumor by Virchow showed its structure to be that of a large cell glio-sarcoma. It was, in fact, a glioma of the upper part of the ependyma.

We know from the experiments of Claude Bernard that the urine increases in quantity and contains sugar, after lesion of the floor of the fourth ventricle between the origins of the pneumo-gastric and acoustic nerves. If the lesion is produced higher up, the urine is more apt to contain albumen. As Schiff demonstrated, at a later period, the lesion produced by Claude Bernard involved the centre of the vascular nerves of the liver. The latter pass through the upper part of the anterior columns and the communicating branches into the sympathetic, the splanchnic branches and the nerves of the hepatic plexus. A lesion, produced in any part of this course, will give rise to diabetes. According to recent researches by Cyon and Aladoff (Bulletin de l'Acad. des Scienc. de Saint-Petersb., 1871, T. XVI.), the nerve fibres, which preside over the production of diabetes, also contain fibres which cause contraction of the hepatic arteries. The artificial diabetes is, therefore, explained by paralysis of these nerves and increase of the current of blood in the dilated hepatic vessels. Simple hydruria indicates lesion of the medullary centre of vascular innervation of the kidneys.

CLASS III.

DISEASES OF THE MENINGES AND PARENCHYMA OF THE CORD.

GENERAL CHARACTERISTICS OF SPINAL DISEASES.

Contemporary medicine has applied all its energies to dissipating the profound obscurity which has hitherto enveloped the diseases of the spinal cord. The progress of histology and experimentation and careful clinical investigations have equally contributed towards this end. In proportion as their light is thrown upon them, the morbid forms and types hitherto unrecognized, grow more clear, take their place in the growing list of our observations, and permit us to hope that other phenomena, which are still obscure, will follow in their course. Spinal diseases, for a long time misconstrued and passed by in silence, have been the subject of profound study during our own times, which have especially contributed towards perfecting the diagnosis and treatment of this class of diseases.

Spinal lesions and their situation are manifested by an ensemble of functional disorders which occur in the principal paths of the transmission of motion and sensation, and also in the domain of the trophic nerves, the nerves of vegetative life and the sympathetic system. In the majority of cases, symptoms of irritation predominate in the beginning and are only followed, at a later period, by signs of depression.

The initial irritation is manifested, on the part of the sensory nerves, by neuralgic pains, hyperæsthesia and formication. Neuralgic pains of various kinds occur in certain affections of the meninges, spinal cord, and vertebræ. The pain appears in the head and face, if the irritation of the nerve trunks originates in the spinal cord or medulla oblongata. Rachialgia develops in the cervical, dorsal or lumbar regions (with or without pain on pressure over certain vertebræ), when the irritation exists in the posterior branches of the spinal roots. Neuralgic pains appear in the upper or lower extremities in vascular or inflammatory irritation of the posterior columns or roots, or of their prolongations in the gray substance. These pains, according to their intensity and character, are dull, shooting, lancinating, or boring. They affect the skin, muscles or bones, and depend upon the situation and nature of the spinal lesion. The girdling pain is a neuralgic pain starting from the spinal column and encircling the trunk. Hyperæsthesia of the skin or muscles may be accompanied, in spinal affections, by neuralgic pains or it may appear independently of the latter. Dysæsthesia (Charcot), that is to say, distressing sensations of

vibration following slight irritation, is only a variety of hyperæsthesia Finally, formications consist of slight irritations, usually due to compression of the nerve roots, propagated from one fibre to another and undergoing instantaneous modifications.

The symptoms of depression on the part of the sensory nerves, occurring in spinal affections, consist of general dulness of sensibility, numbness, and even complete abolition of sensibility or anæsthesia. The latter, as I have demonstrated, corresponds to the distribution of the cutaneous nerves, discovered by Voigt. According to recent researches, we may, in addition, observe objective differences in the abolition of certain varieties of sensation (such as paralysis limited to tactile sensibility).

In order to recognize, objectively, the sensory disturbances of spinal origin, we must carefully examine the various qualities of sensibility by scientific and rigorous means. Before entering into the discussion of this question, we may state that, from a practical point of view, we can obtain useful data concerning the changes in the various forms of sensibility, without having recourse to prolonged or difficult investigations. Tactile sensibility may be estimated by examining the skin with the finger under cloth, or directly by means of a piece of cloth or linen or with a pin. The sensibility to pain may be judged by pricking or pinching the skin or by pulling the hairs in different localities; the sensibility to temperature by means of a spoon or thermometer plunged into hot or cold water and applied immediately to the skin.

More delicate methods of exploration enable us to detect partial disturbances of sensation. Tactile sensibility and the notion of space separating two impressions are measurable by Weber's compass or Sieveking's æsthesiometer. To determine the sensibility to pressure, we may employ various weights (E. H. Weber) or apply an india-rubber sac filled with water upon that portion of the skin which we wish to examine, and ascertain the weakest impulse conveyed to the fluid which can be perceived by the patient (Goltz). We may also employ Eulenburg's æsthesiometer. This is composed of a spiral spring, which presses more or less strongly, according to its tension, upon a plate of caoutchouc. This compression is then transmitted, by means of a rod and cog-wheel, to an index connected with a graduated dial. The sensibility to temperature is determined by plunging a finger alternately into two vessels filled with water at different temperatures (E. H. Weber) or by placing, upon the skin, cylinders of copper filled with water, surrounded by a poor conductor and carrying a thermometer in the lid (Nothnagel).

The sensibility of the skin to pain may be measured by means of faradization, after removing the secondary coil. The electro-muscular sensibility may be measured, in intelligent patients, by the feeling of contraction experienced in the muscles when they are stimulated by the primary current. In order to recognize the condition of the muscular sense, it is necessary, according to Weber, that the patient lift different weights (notion of muscular energy).

The sensation of the position of the limbs (Leyden), which is preserved in purely motor, but not in sensory paralyses, and which resides in the sensory nerves of the skin, muscles, and joints, can be examined by subjecting different portions of the limbs to passive and faradic movements. Reflex excitability is determined by means of mechanical irritation and still better by electricity.

The different varieties of sensibility are unequally affected in spinal lesions. They constitute partial paralyses of sensibility of a variable dura-

tion and are rarely permanent. Thus, in the majority of cases, the sensibility to tickling disappears first, then the sensibility to contact and pressure, then to temperature, and finally to pain.

Schiff, in accordance with his experimental investigations, considers loss of tactile sensibility with preservation of sensibility to pain as anæsthesia properly speaking. He connects it with a lesion limited to the posterior columns, and attributes the abolition of the sensibility to pain (analgesia or anodynia) to a lesion of the gray substance. But Brown-Séquard considers the gray substance (and especially its central portion) as the organ of transmission of both varieties of sensibility. In the majority of spinal affections, the disorders of sensibility which we have studied will not enable us to determine whether the lesion is limited to the posterior columns or whether the gray substance is also affected at the same time. The retardation of sensory perceptions depends (according to Leyden) upon a resistance to transmission in the gray substance, and the retardation of motor impulses (with manifest slowness of movements) to a disorder of co-ordination.

The following are the symptoms of irritation on the part of the motor nerves: spasms, tremors, muscular stiffness, contractures, tetanic and epileptiform attacks. The muscular spasms may be limited to certain fibres and give rise to fibrillary contractions (as in injuries to the cord and progressive muscular atrophy); or they may consist of more intense clonic convulsions involving the limbs and trunk, being, most frequently, reflex spasms due to irritation of the cord, especially of the gray matter. Tremor consists of muscular contractions with oscillations. It can be experimentally produced in decapitated animals, by stimulating the spinal cord by means of induced currents with slow intermissions. Tremor also occurs, under the influence of irregular stimuli transmitted through weakened motor fibres, in ataxia, sclerosis, myelitis, etc., especially after motor stimulation.

Muscular stiffness is characterized by a peculiar rigidity and stiffness of voluntary movements, which can only be accomplished with great effort and after which the contracted muscles only gradually come to rest. Contractures with the peculiar attitudes which they cause the limbs to assume are either of an irritative, spasmodic nature (they then yield during sleep or under the influence of narcotics) or of paralytic origin (paralysis of the antagonists) or, finally, they result from circumscribed spinal lesions, as in sclerosis of the lateral columns. Tetanic symptoms are observed in spinal meningitis and injuries of the cord, in poisoning and in hysteria. They usually result from a morbid exaggeration of the reflex power of the gray substance of the cord, which Schroeder v. d. Kolk has found strongly congested and strewn with small hæmorrhages in the tetanus induced by strychnine.

Finally, the epileptiform attacks (spinal epilepsy) which occur in spinal affections, such as caries of the vertebræ and traumatism, are directly under the influence of the cord or, by reflex means, under the influence of certain peripheral nerves. In the latter case, the irritation is transmitted to the centre of general convulsions (pons and medulla). Experimental lesions of the cord and sciatic nerves (Brown-Séquard) bring into play, in the same way, the function of the cord as a conductor and terminate in epilepsy.

The most characteristic symptoms of depression are represented by the extremely varied forms of spinal paralysis. The paraplegic type, known even to the ancients, chiefly involves the lower limbs with frequent

extension of the paralytic symptoms to the sensory nerves and to the sphincters of the bladder and anus. Paraplegia dolorosa is observed in compressions of slow progress, as in cancer of the vertebræ (Charcot). In circumscribed lesions of the cervical cord, the upper limbs may be alone paralyzed (cervical paraplegia). Unilateral spinal paralyses are rare; as examples of hemiplegic spinal paralyses, we may mention those which occur in circumscribed medullary lesions of the lateral column on the opposite side, and sometimes also in ataxia. We observe more often that the other half of the body has not been completely spared by the disease. In the true spinal hemiplegia of Brown-Séquard, we find in man, as he also observed in his experiments upon animals, motor and partial vaso-motor paralysis, with hyperæsthesia, on the side corresponding to the lesion, and paralysis of sensation with integrity of motion on the other half of the body. Spinal hemiparaplegia, or paralysis of one leg with crossed anæsthesia, is found in unilateral lesions of the cervical and lumbar cord.

Alternate spinal paralysis, affecting the upper limb of one side and the lower limb on the opposite side, is produced in localized lesions of the pyramids, when the lesion involves the nerve fibres of the upper limbs before, and those of the lower limbs after, decussation. Spinal monoplegia, or isolated paralysis of certain limbs or of certain groups of muscles, is found in caries of the vertebræ, infantile spinal paralysis, and in the beginning of progressive muscular atrophy. In these cases, other pathognomonic signs indicate the spinal origin of the isolated paralyses. We may finally mention general spinal paralysis, affecting all the limbs, though in different degrees. It usually results from progressive changes of the medullary parenchyma which, starting from the anterior half of the cord, as in multiple fractures of the vertebræ and tuberculosis of the gray matter, successively involves the gray horns and posterior columns, producing paralysis of the upper and lower limbs.

Trophic disturbances may occur in the course of spinal paralyses, chiefly affecting the muscles of the paralyzed limbs. In the majority of spinal diseases, autopsy reveals, in the muscles of the limbs which have been rendered immovable, pallor and thinning of the fibres, advancing even to disappearance of the transverse striæ and to fatty degeneration. The muscular atrophy is much more pronounced in those forms which invade the trophic centres situated in the anterior horns (progressive muscular atrophy, infantile spinal paralysis, etc.), or when these centres have ceased to be in communication with the peripheral nerves (compression of the anterior roots, paralytic meningitis and neuritis). The emaciation of the muscles is then rapid and considerable, the atrophy and paralysis often, though not always, pursuing a parallel course. Hypertrophy of the skin, muscles, and bones, arthropathies, herpetic eruptions, and acute forms of bed-sore, also occur as trophic disorders in various spinal diseases.

The electrical reactions of the paralyzed nerves and muscles are extremely variable in the different forms of spinal affections. Circumscribed processes occupying the upper parts, with simple wasting of the muscles, do not give rise to noteworthy changes in the electrical reactions. In those forms of myelitis occupying the entire transverse diameter of the spinal axis, the galvanic excitability of the nerves and farado-muscular contractility progressively diminish and may even entirely disappear. In diseases which are characterized by a lesion of the trophic centres of the muscles, the disorders of the nutrition and activity of the muscles are frequently accompanied by abolition of their electrical excitability. But

the two phenomena do not necessarily go hand in hand, and sometimes the atrophy, sometimes the alteration of the electro-muscular excitability predominates. Galvano-muscular reaction usually persists much longer than the farado-muscular reaction. In the irritative forms of tabes and in the stage of ataxia, we meet with abnormal increase of the galvanic excitability of the nerves to weak currents, and increase of the excursion of the limbs from applications of short duration or when the current is reversed.

In addition to the paralytic forms which we have referred to, we must also mention the disorders of co-ordination, the ataxias, peculiar to certain spinal diseases. Voluntary motion is then preserved in the individual muscles, but there is more or less complete abolition of the complex movements which are produced by the synergic action of different muscular groups. According to recent researches, the lesions, in these cases, occur in the long paths of conduction, which maintain a constant relation between the centres of co-ordination situated in the mesocephalon and cerebellum (pons, tubercula quadrigemina, and cerebellum) and the system of ganglion-cells and paths of transmission in the posterior and anterior portions of the cord. We shall enter into this subject more in detail in the chapter upon ataxia.

The various morbid conditions of the spinal cord may also produce considerable and serious disturbances in the nervous system of vegetative life and in the grand sympathetic. Not only the intensity of the spinal lesion, but also the height at which it is situated, exert a decisive influence in this direction. In affections of the cervical cord and medulla oblongata, respiration, cardiac activity, and the motor nerves of the iris become involved. The centres of vascular innervation, which extend from the medulla oblongata to the spinal cord, according to the recent researches of Goltz, Vulpian, etc., and the vaso-motor nerves, which pass out in the anterior roots, are more or less involved in the various spinal diseases. We thus observe considerable elevation of temperature in traumatic lesions of the cervical cord, in hysterical convulsions, and in spinal paraplegias and hemiplegias of acute origin. Reflex contraction of the vessels rarely occurs in the paralyzed limbs; but, on the other hand, we usually find chronic vascular dilatations and venous stases with cyanosis, cool skin, and œdema.

Disorders of digestion with constipation, meteorism, and, more rarely, rebellious diarrhœa, are observed in diseases of the cervical and lumbar portions of the cord. Goltz has seen diarrhœa occur in dogs, after destruction of the lumbar cord. Irritation of the cervical and upper portion of the dorsal cord produces morbid increase of sexual desire and of the virile functions (as in the beginning of ataxia and general paralysis). Concussion and lesions of the cord (as in fractures of the vertebræ) may give rise to painful erections and priapism. Diminution of sexual desire and impotence may occur in chronic spinal diseases. According to Goltz's recent researches (Pflueger's Arch., VIII. Bd., 1874), the reflex centre of erection is situated in the lumbar portion of the cord.

The lumbar portion of the cord also exerts a manifest influence upon the sensory and motor functions of the rectum and bladder. In meningitis and ataxia, the patients sometimes experience severe neuralgic pains and a feeling of constriction and intense tenesmus in the rectum or bladder. At a later period, sensibility becomes involved, causing loss of reflex excitability. Paralysis of the bladder may be produced, according to the latest experiments of Budge (Pflueger's Arch., Bd. II.), by section of the anterior

roots of the third, fourth, and fifth pairs of sacral nerves, which contain the motor fibres passing to the muscles of the bladder and urethra, and also by section of the posterior roots of the same nerves, which preside, by reflex action, over the tonus of these muscles. Even when the lumbar cord and reflex paths are intact, lesions of the cervical and dorsal regions may produce disturbance of the vesical functions by interrupting the action of the cerebral motor centres (contained in the peduncle) and their connections with the anterior columns and sacral nerves. Retention of urine then occurs, with decomposition and consequent cystitis and pyelitis, which may hasten and still further aggravate the progress of the spinal affection.

Participation of the brain in diseases of the spinal cord sometimes occurs from morbid processes, extending from the cord to the brain, from the cervical cord to the nuclei of the nerves at the base of the brain, or vice-vêrsa. Cephalic symptoms of grave import are also produced as a final complication in severe diseases of the cord. The extension of morbid processes, in an ascending direction from the cord to the brain, is observed in inflammations of the spinal meninges, in myelitis and ataxia complicated with disease of the cranial nerves, in psychical disorders following spinal lesions of long standing, and in diffuse central affections of a sclerotic or syphilitic nature. Secondary degenerations of the cord from above downwards are produced in cerebral apoplexy and softening, and others are superadded to the cerebral symptoms of progressive general paralysis.

Cerebro-spinal meningitis, tubercular forms of basilar meningitis and various kinds of extravasations and tumors, are also propagated from the brain to the cord.

The cerebral symptoms of depression in diseases of the medulla oblongata may be complicated by convulsions and disturbances of respiration and deglutition, or they may follow inflammatory processes extending from below upwards (as in meningitis, caries of the vertebræ, etc.). Sometimes they are of a pyæmic nature (in bed-sores, and purulent cystitis), and, at other times, they appear as "final phenomena" with strong fever, small and hurried pulse, difficult respiration, and dysphagia, when the bulbar and vaso-motor centres are invaded by medullary processes of a rapid course. These conditions quickly terminate in fatal collapse, with delirium and coma.

Finally, the nerves of the organs of special sense may become implicated by the extension of the spinal affection. The phenomenon of most frequent occurrence is the atrophy of the optic nerve, after which the ocular muscles and the pupils are the first affected. The acoustic, trigeminal and facial nerves become involved in rare cases, when the degenerations are propagated to the nuclei and roots of these nerves in the medulla. Disorders on the part of the pneumogastric and spinal accessory nerves are exceptional. The motor nerves of the tongue may be affected in diffuse sclerosis of the nervous centres, in bulbar paralysis, in general paralysis, and in ataxia.

The diagnosis of spinal affections is made according to their symptomatology, with the extent of which we are better acquainted at the present time, and according to the anatomical course of certain disorders. The situation and extent of spinal lesions are usually determined with much more readiness than their anatomical characteristics. We have made considerable progress in this direction, but many points are still involved in doubt and obscurity.

I.—DISEASES OF THE SPINAL MENINGES.

The participation of the meninges in spinal affections is as frequent and important as in diseases of the brain. The primary morbid changes in these membranes present considerable analogy within the skull and spinal canal. They only vary on account of the different conditions of the venous circulation and of the stases and lowering of the circulation. The secondary lesions of the meninges, which supervene in the majority of spinal affections, are, to some extent, accessory and of minor clinical importance. The following remarks refer especially to those morbid forms which are peculiar to the spinal meninges and which are capable of being distinguished clinically.

CHAPTER XVI.

HYPERÆMIA AND APOPLEXY OF THE SPINAL MENINGES.

a. *Hyperæmia of the Meninges and Cord.*

The older physicians and notably Ollivier regarded congestions of the cord (plethora spinalis) as very frequent; but this view was based less on positive facts than on theoretical considerations, and even Abercrombie had opposed this notion. At the present day pathological anatomy is much more reserved with regard to the increase of the quantity of blood contained in the meninges and parenchyma of the spinal cord. We are not justified in admitting the evidence of spinal congestion unless we observe marked fulness of the vessels of the dura mater and pia mater, and a red, dark color of the tissue of the spinal cord, especially in the gray matter. The ordinary position of the cadaver in dorsal decubitus, the stasis of blood in the venous plexus of the lower part of the cord, the imbibition of the tissues with the coloring matter of the blood under the influence of putrefaction—all these causes may produce cadaveric congestion, though this does not prove that the vascular repletion existed during life. On the other hand, very active but temporary congestions of the spinal system frequently occur during life, and leave no traces in the cadaver.

In addition to marked fulness of the vessels, especially those of the pia mater, and the redness of sections of the cord, recognizable with a lens, we may also observe hæmorrhagic specks and a dark color of the gray substance (which is richer in capillaries). This justifies us in regarding the process as an active congestion, and sometimes even as the commencement of inflammation of the spinal cord. These appearances are presented in certain cases of cerebro-spinal meningitis, typhoid fever, puerperal diseases, acute exanthemata, etc. In the course of convulsions and of choreic and tetanic symptoms, we may observe venous hyperæmia, which must be considered as terminal stases, and not as the causes of death.

If, in order to appreciate the symptoms of congestion of the cord, we recur to cerebral hyperæmia, whose signs surpass the former in frequency and clearness; if we oppose the cephalalgia, dulness of sensibility, diffuse pains in the limbs, the weakness of the tongue, of an arm or leg, which

are observed in cerebral hyperæmia, to the lumbar pains, the numbness, formication and painful twitches in the legs, the sensation of heaviness in the lower limbs, which accompany spinal congestion, we will find a complete analogy between these two morbid conditions, an analogy which is even more marked if we take into consideration the similarity of their causes, and the prompt recovery in which they both frequently terminate.

There are, on the other hand, certain objective signs of cerebral hyperæmia, such as redness of the face and conjunctivæ, dilatation of the jugular veins, throbbing of the carotids, nausea, vomiting, and mental excitement, which are observed with less distinctness in spinal hyperæmia. We may also add that, in the latter, the respiration is often difficult, short, and incomplete.

Among the causes of spinal hyperæmia, we may mention, as most prominent, general irritation of the nervous system, which causes unusual fulness of the vessels in the brain and cord.

The communications of the basilar and vertebral arteries, and of the anterior and posterior spinal arteries to which they give rise, favor the distribution of the hyperæmia throughout corresponding territories of the brain and cord. In respiratory and circulatory disorders (organic diseases of the lungs and heart), especially in diseases and degenerations of the abdominal viscera and in stases of the portal system, the congestion especially occurs in the lumbar venous plexuses. Their varicose dilatation and the consequent swelling and softening of the tissue of the spinal cord may accompany chronic hyperæmia.

Hyperæmia of the spinal cord is frequently due to sexual excesses and onanism, which keep up long-continued symptoms of spinal irritation, and may terminate in tissue changes, when their influence is prolonged and exaggerated.

The symptoms of spinal congestion also appear after violent and prolonged efforts and after traumatic concussion of the vertebral column. A severe chilling, and the cessation of the menses or perspiration, which frequently occurs in consequence, may exercise a depressing action upon the vaso-motor centres, by a reflex influence of the vast nervous meshwork of the skin, and may give rise to the development of spinal hyperæmia and even of tissue changes, as Feinberg has observed in experiments upon animals coated with varnish.

In these experiments, injection of the spinal meninges and gray matter was found, with dilatation of the capillaries and microscopic hæmorrhages in considerable number. In some cases, proliferation of the neuroglia also occurred. We may finally add that Magnan (l. c) has noticed a diffuse rose color and vascular injection in thin sections of the cord in animals poisoned with absinthe.

The lumbar pains, the painful sensations and weakness of the legs, which temporarily occur in the course of severe febrile diseases, typhoid fever, acute exanthemata, puerpera' diseases, etc., should also be attributed to the hyperæmia of the cord, although this has been only directly demonstrated in a small number of cases. In morbid processes characterized by spinal convulsions, such as tetanus, eclampsia, etc., intense hyperæmia occurs in the gray matter of the cord. The experiments of Feinberg have shown that this is sufficient in itself to produce convulsions; but it is usually accompanied by capillary hemorrhages, which alter the tissue of the cord in numerous points. We shall, at a later period, recur, in detail, to the relations of spinal irritation to hyperæmia of the cord. The symptoms of spinal hyperæmia are generally irritative in character.

The rachialgia, which is situated in the lumbar or sacral regions, is a dull pain, increased by violent emotions, exertion, and sometimes even by dorsal decubitus. The pains radiate into the lower limbs, more rarely into the upper, and are attended by numbness or formication. Very often spasms are temporarily produced in various muscles. Reflex excitability is usually increased; I have often seen marked increase of the galvanic excitability of the nerves.

As symptoms of depression, we often find dulled sensibility, heaviness and stiffness in the legs, and sometimes also in the arms. True paralyses are rare, but Hesse (Krankh. d. Nervenapparates, 1855) has, nevertheless, observed paralysis of the lower limbs after exposure, and, more recently, Steiner (Arch. d. Heilk., 1870) saw a case of ascending paralysis in consequence of physical fatigue. Both cases rapidly improved under the influence of rest, warmth, and appropriate antiphlogistic remedies, and terminated in complete recovery. The paralyses not unfrequently assume an intermittent or remittent character.

The diagnosis of congestion of the cord and its meninges is obtained from the symptoms which we have given, and from its causation. The promptly favorable course of the disease renders the diagnosis of spinal congestion, which is at first somewhat arbitrary, very probable. The duration and termination vary according to the etiology of the congestion. The acute cases, whose causes we have mentioned, generally terminate in rapid recovery. The chronic varieties, due to venous stases, run a more prolonged course, present more marked changes in their symptoms, and yield more slowly. Sometimes they recover very quickly under the influence of a spontaneous hemorrhage from the veins of the uterus or rectum, which anastomose with those of the cord. Death rarely occurs as a termination of simple hyperæmia of the cord. In these unfavorable cases an intra-medullary hæmorrhage, a serous infiltration, or softening of the parenchyma generally coexists. Symptoms of asphyxia are very frequently present, indicating an affection of the respiratory centres. Prolonged congestion may give rise to secondary degeneration and hyperplasia in the cord as well as in the brain.

The treatment should depend upon the causation of the spinal congestion. In acute hyperæmia, antiphlogistic measures are indicated, together with rest and absolute diet. Wet cups or leeches to the vertebral column, and leeches applied to the anus or vulva, give good results. Benefit is also derived from cool effusions to the vertebral column while the patient is in a warm half-bath, from applications of cold compresses to the back, or of Chapman's rubber-bag (this should be filled with water which is gradually made colder and colder, and not immediately with pieces of ice).

I have noticed that frictions with cold water and dorsal douches are not well tolerated in acute cases. Stimulation by cold may increase the congestion by reflex means. For the same reason, we should be cautious in the employment of galvanic or faradic currents, and abandon them as soon as they produce disagreeable sensations. In the chronic forms, associated with vascular stasis, good effects are seen from moist frictions, lumbar douches, electrization of the dorsal region, cold washings, milk diet, and the cautious employment of gymnastic exercises.

b. *Apoplexy of the Spinal Meninges.*

Under this term we include hæmorrhages occurring between the vertebræ and dura mater, or between the meninges themselves. Hayem has

recently made an exhaustive study of this subject from an anatomical and clinical standpoint (Des hemorrhagies intra-rachidiennes. Paris, 1872). The extravasated blood is black and coagulated, more rarely fluid and mixed with clots, and is found in the loose connective tissue upon the external surface of the dura mater, in the cavity of the arachnoid, or in the tissue of the pia mater. Sometimes it occupies several of these positions at the same time. The most frequent form is that of the extra-meningeal hæmorrhages, between the vertebræ and dura mater. In the chapter on diseases of the medulla we recognized arteriitis of the basilar and vertebral arteries. Since the latter supply the arteries of the dura mater and the spinal arteries which are distributed to the pia mater by numerous ramifications, it is probable that, upon closer examination, degenerations of the vascular walls with rupture and consequent hæmorrhages will also be found in them. In certain cases the collections of blood outside of the dura mater are more extensive, on account of the abundant venous plexuses of this region, than hæmorrhages into the arachnoid cavity, in which the veins are less numerous.

Hæmorrhages of the dura mater, bounded externally by the connective tissue, are characterized by the presence of clots surrounding the roots of the nerves and by imbibition of their tissues. In rare cases the dura mater is separated by abundant collections of blood. Upon the inner surface of the dura mater, and in the meshes of the pia mater, we find either small, circumscribed hæmorrhages, or foyers, extending into the cavity of the arachnoid, filling up a large part of the spinal canal, and sometimes associated with a cerebral hæmorrhage. The cerebro-spinal fluid is stained with blood.

The causes of meningeal apoplexy are very diverse. According to Hayem, the affection was only primary twice in thirty-eight cases. The causes of the secondary varieties consist of traumatic lesions of the dura mater or vertebræ, of tetanus, eclampsia, epilepsy, chorea, and circulatory disorders, especially in the new-born. Meningeal apoplexy also occurs in toxic convulsions, meningitis, and more rarely and to a less severe extent in the affections connected with vascular stasis, which we have previously mentioned.

We will here mention some very rare examples of hæmorrhages following lesions of the dura mater and arachnoid. Ollivier (l. c.) has published a case of syphilitic caries opening into the spinal canal, with perforation of the dura mater. In Laennec's case of aneurism of the thoracic aorta, sudden, incomplete hemiplegia occurred, resulting in death at the end of several hours. At the autopsy, the aneurismal sac was found opening into and communicating with the spinal canal and with the left pleura. The cord was compressed by a clot of blood. An analogous case has been since published by Chandler.

The extravasation may occur between the layers of the arachnoid or inside of this membrane. Only one case of primary intra-arachnoid hæmorrhage has been observed. The extravasation has almost always been propagated from the cranial to the spinal cavity. Among eight cases of subarachnoid hæmorrhage collected by Hayem (l. c.), two were spontaneous and primary. It was secondary in the other cases, being either consecutive to hemorrhage into other portions of the nerve centres, or occurring in the course of various diseases, such as alcoholism, senile dementia, spinal meningitis, hæmorrhagic variola, scorbutus and its different forms.

The symptoms of meningeal hæmorrhage are sometimes of an exclu-

sively medullary nature, when the brain is unaffected, and the origin of the process then appears more clearly. At other times, it is complicated with syncope, loss of consciousness, disturbances of speech and the special senses, indicating the coexistence of severe congestion or of an extravasation within the cranial cavity. When death occurs rapidly, it is hardly possible to comprehend the exact part played by the two sets of lesions. Hæmorrhage is sometimes preceded by the hyperæmia which we have described above. But in the majority of cases, meningeal hæmorrhages, whether traumatic or spontaneous, have a sudden and apoplectiform invasion. The patient is seized with violent pain, and falls down, giving utterance to cries; he is paralyzed in the limbs and often also in the organs of special sense. When the violent symptoms of this first stage have passed we are better able to distinguish the characteristic phenomena of the disease.

Intense rachialgia develops in the beginning, situated either in the neck or lower down or along the whole length of the spinal column, and we can thus draw deductions as to the situation of the hæmorrhage. The most important symptom, in addition to this pain, is the tetanic stiffness of a segment of the vertebral column. This tonic spasm chiefly involves the muscles which receive their nerves from the affected region, and the limbs are usually contractured. From time to time, clonic spasms or tremor also occur. All movements increase these phenomena, as when the patient wishes to raise himself, to stoop down, or to turn around. Very frequently the skin of the dorsal region and the spinous processes do not present an abnormal sensibility. When the extravasation compresses the nerve roots, peripheral irradiations occur into the upper or lower limbs, under the form of scalding sensations, formications, numbness, etc.

Sensibility is also affected in its turn. Cutaneous or even muscular hyperæsthesia on pressure occurs in the beginning, especially in the lower limbs. After this first modification, or even much later, anæsthesia occurs in the dorsal or lumbar regions, and extends downwards to the buttocks, perineum, genital organs, and thighs. Motion is also more or less affected. Voluntary motion may be impeded by the muscular stiffness occurring at the onset, or even abolished by paralyses, which are readily recognized.

The height of the hæmorrhagic lesion in the spinal cord influences the entire symptomatology. In apoplexy of the cervical cord or of the upper part of the dorsal region (usually of traumatic origin), we observe, in addition to temporary loss of consciousness due to cerebral hyperæmia, pains and stiffness in the shoulders, dilatation of the pupils, spasms in the neck, pain, contractures, and sometimes paralysis of both arms, with partial increase of sensibility. In many cases we also notice disorders of deglutition and respiration, and, finally, irregularity of the heart.

Lesions of the lower parts of the dorsal region, or of the lumbar region (from a fall upon the loins, buttocks, or feet), are recognized by local pain and muscular stiffness, by the abolition of motion and sensation in the lower limbs, paralysis of the sphincters (at first with retention of urine and constipation), and by priapism, which is sometimes observed at the beginning. The course of apoplexies of the spinal meninges is determined by the abundance and extent of the hæmorrhagic extravasation. Small circumscribed hæmorrhages may terminate in recovery (as Ollivier's observations serve to demonstrate). The meninges are found strongly pigmented, with adhesions of the dura mater and pia mater (remains of small apoplexies), after vertebral lesions, which have finally proved fatal from

their complications. Large extravasations, affecting the cervical region and medulla oblongata, may rapidly prove fatal, from disorders of respiration or circulation, or from complications involving the brain.

In favorable cases the violence of the symptoms soon diminishes, but at the end of forty-eight hours the condition is again aggravated under the influence of the inflammatory reaction, which varies in its intensity and duration and usually does not disappear for two or three weeks. The extravasation is then absorbed, all the symptoms gradually mend, the functions become re-established, unless complications on the part of the meninges, or medullary substance, cystitis or complete paralysis of the sphincters, give rise to new dangers.

The diagnosis is based upon the apoplectiform onset of the symptoms of meningeal irritation, and upon the etiological factors. Sudden and violent rachialgia, tetanic stiffness of the trunk and neck, contracture of the limbs, partial convulsions, weakness of the limbs and absence of cerebral symptoms, are the symptoms indicating the spinal origin of the meningeal hæmorrhage. In order to localize the situation of the lesion, we must bear in mind the symptoms above mentioned. The prompt appearance of hemiplegia of motion and sensation and of paralysis of the sphincters, and the abolition of electro-muscular excitability, prove that the substance of the cord is involved. Rapid improvement on the part of the brain indicates that the irritation resulting from shock has soon subsided. The prognosis of apoplexy of the spinal meninges loses its gravity when the threatening symptoms of the first few days have subsided. It improves still further about the fourteenth day, after the inflammatory reaction has ceased. At a later period improvement is characterized by the return of sensibility, and then of motion and of the action of the sphincters. Complications with cystitis and bed-sores render the prognosis more grave, and the rapid disappearance of the vesical disturbances generally favors the return of the vital energies. Weeks and even months elapse, however, before all the disorders of motion and sensation subside.

Venesection is indicated in the treatment of cases complicated with cerebral symptoms and in individuals who are sufficiently robust. We must then have recourse to local treatment, such as bleeding along the vertebral column by means of wet-cups or leeches and ice-bags over the affected portion. The patient should be kept absolutely quiet in bed, upon strict diet, and, at a later period, we may employ mercurial or iodine ointment. Injections of morphine may be used to combat intense pain. The vesical disturbances and the constipation demand special attention. Persistent paralyses require electrical, hydropathic, or thermal treatment.

CHAPTER XVII.

INFLAMMATIONS OF THE SPINAL MENINGES.

The inflammatory processes of the spinal meninges exert considerable pathogenic influence upon the cord itself, and upon the nerve roots which they surround. Despite the great analogy in the symptomatology of the various forms of spinal meningitis, we nevertheless recognize marked distinctions in the manner of invasion and development. These are useful in studying the diagnosis and clinical history of these diseases. We shall, therefore, consider separately the diseases of the spinal dura mater, arachnoid, and pia mater.

A.—Diseases of the Spinal Dura Mater.

Among the older observers we only find one case of purulent perimeningeal infiltration mentioned by Ollivier (l. c., T. II., p. 272). The inflammatory processes of the cellular tissue surrounding the dura mater and the inflammation of the dura mater itself have been more carefully investigated in our own times. The morbid processes present considerable differences, according as the inflammation chiefly affects the external or internal surface of the dura mater.

a. *Perimeningitis and External Spinal Pachymeningitis.*

Spinal peri-pachymeningitis (Traube, Mannkopf, Mueller) consists of inflammation and suppuration in the cellular tissue surrounding the dura mater, and may be diffused or circumscribed. When the pus collects in considerable quantity, especially in the posterior part, it may compress the dura mater, push it against the spinal canal, and then compress the cord. Several foci may be present without extension of the inflammatory process to the brain.

The inflammation of the perimeningeal cellular tissue is usually secondary.

True spinal pachymeningitis, such as has been recently described in caries of the vertebræ, by E. Wagner (Arch. d. Heilk., 4. H., 1870), and by Michaud (Sur la méningite et la myélite dans le mal vertébral, 1871), consists of inflammation of the anterior and external layers of the spinal dura mater. Upon microscopical sections, we can distinguish, from within outwards, in addition to the normal connective tissue, a fibrillary middle layer, strewn with nuclei, fusiform cells, and new-formed capillaries, and a superficial granulo-fatty layer, deprived of blood-vessels and in a condition of cheesy degeneration. This is, therefore, an external pachymeningitis, characterized by the presence of inflammatory products upon the external

surface of the dura mater, the formation of abscesses in the false membranes, and retrogressive changes in the peripheral layers. The process is usually confined to the external surface of the spinal dura mater, but, more rarely, it extends also to its internal surface. The lesions of this variety of pachymeningitis may finally terminate in compression and atrophy of the nerve roots, and of the superficial layers of the cord.

The causes of perimeningeal and intrameningeal inflammations are usually found either in suppurations extending from the exterior or in local irritations. The peri-pachymeningitis in Traube's (Gesamm. Beitr., II. Bd.) and Mannkopf's (Berl. klin. Wschr., 1864) cases resulted from phlegmons of the psoas muscle and cervical region respectively, and in H. Mueller's case (Peripachymen. spin. Diss., Koenigsberg, 1868), from inflammation of the subpleural cellular tissue. In all these cases the suppuration was propagated through the intervertebral foramina to the perimeningeal cellular tissue. External pachymeningitis may follow traumatic lesions of the vertebræ and, especially, caries of these bones. In the latter disease, according to Michaud (l. c.), a cheesy purulent mass is found at the level of the diseased vertebræ, and comes in contact with the dura mater after ulceration of the posterior vertebral ligament, thus giving rise to inflammation and proliferation by the direct action of the pus upon the external surface of the dura mater. Thickening and gummy products in the dura mater and other meninges may also develop under the influence of syphilis (Lancereaux, Winge). The existence of an idiopathic, rheumatic pachymeningitis has not been demonstrated.

The symptoms of perimeningitis and pachymeningitis are, with a few slight exceptions, similar to those of spinal meningitis. The most striking symptoms are the same in both, such as pains in the shoulders or lumbar region, stiffness, especially in the middle or lower segments of the vertebral column, cutaneous and muscular hyperæsthesia, especially in the lower limbs, periodical painful irradiations, more or less complete paralysis of the legs or bladder, and more or less intense, irregular fever. The diagnosis of external pachymeningitis is only possible if we are able to determine the existence of foci of suppuration in the neighborhood of the vertebral column, and if the symptoms of an insidious spinal meningitis then develop, usually in the cervical region.

In traumatic lesions, caries of the vertebræ, deep bed-sores over the sacrum, abscesses developing around the spinal column in the pelvis or between the cervical vertebræ and the pharynx (Angina Ludovici) which may penetrate into the spinal canal,—in all these cases, when fever and inflammatory symptoms on the part of the meninges occur, the spinal dura mater has become involved.

The course of the disease has been unfavorable in the majority of the cases hitherto observed. Death ensues from the extent of the suppuration, from pyæmia, or from compression of the cord. But in many cases of abscess which terminate favorably, and in caries of the vertebræ, which we shall discuss hereafter, we may so much the more hope for recovery, as this sometimes occurs even in spinal meningitis.

With regard to treatment, we should direct all our attention to the primary origin of the suppuration. If this is susceptible of cure, we are usually also successful in the treatment of the secondary pachymeningitis. We refer for further details to the remarks that have already been made on the treatment of spinal meningitis.

f. *Internal Spinal Pachymeningitis.*

Meningitis originating upon the inner surface of the spinal dura mater is divided, from an anatomical and clinical standpoint, into two varieties, viz.: hypertrophic cervical pachymeningitis, and internal hæmorrhagic pachymeningitis.

Hypertrophic pachymeningitis, recently demonstrated by Charcot (Soc. de Biol. 1869), consists in a considerable thickening of the spinal dura mater in the cervical region, resulting from proliferation of its internal layers, and of the adjacent arachnoid and pia mater. When the circular thickening of the meninges extends over a large part of the spinal axis it produces constriction of the cord and roots of the nerves, with consecutive neuritis.

This disease is distinguished from external pachymeningitis (after caries of the vertebræ) by its situation in the cervical region, by its point of departure from the inner surface of the dura mater, and by the circular compression which it exercises upon the spinal cord, causing it to become smaller in all its dimensions; the site of external pachymeningitis depends upon that of the vertebral affection, it starts from the external layers of the dura mater, and on account of its slight extent only affects a limited portion of the cord.

Hypertrophic cervical pachymeningitis is manifested by clinical signs which render it accessible to diagnosis, according to its degree of inten-

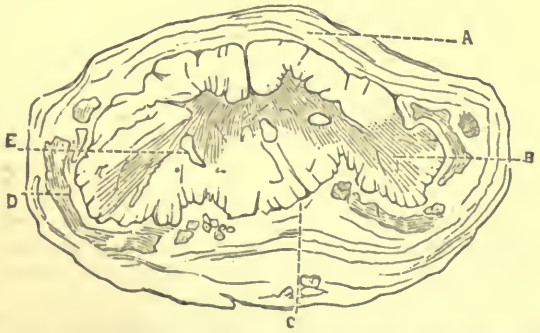

FIG. 11. Hypertrophic cervical pachymeningitis. *A*, hypertrophied dura mater; *B*, nerve roots passing through the thickened meninges; *C*, pia mater adherent to dura mater; *D*, lesions of chronic myelitis; *E*, section of two newly-formed canals in the gray substance.

sity and its anatomical lesions. During the stage of irritation, which lasts two or three months, violent neuralgic pains occur in the neck and upper limbs (often upon one side alone), with a girdling pain around the upper part of the thorax. Then the stage of paralysis appears, characterized by more or less complete paralysis and contracture of the upper limbs (superior or cervical paraplegia), with very marked muscular atrophy and diminution of electro-muscular contractility (as in progressive muscular atrophy), in consequence of atrophy of the anterior and posterior roots of the nerves. At a later period the lower limbs become affected in their turn, but usually to a less extent. This affection, after a prolonged stationary period, may go on to recovery and terminate in complete resto-

ration of the power of motion. This spontaneous retrogressive change has been several times observed by Charcot, and runs counter to the pretensions raised by therapeutics.

Internal hæmorrhagic pachymeningitis corresponds to the process of the same name and character which occurs upon the cerebral dura mater. The spinal dura mater is covered upon its inner surface by several layers of false membrane of a yellowish-red or blood-red color, very rich in vessels, strewn with smaller or larger extravasations, and readily detached. The false membrane at first occupies a very small extent of the interior surface of the dura mater. In very rare cases it is spread over a large surface, and extends to the cranial dura mater. Sometimes the spinal pia mater is tinged with blood, but it hardly ever adheres closely to the dura mater. The cerebro-spinal fluid is tinged with blood in varying degrees.

The *clinical signs* of this disease, for which the materials at our command are insufficient, are less understood than its anatomical characters. The most marked forms of internal hæmorrhagic pachymeningitis have been observed in insanity, associated with analogous cerebral lesions. Observations of this character have been published by A. Meyer (De pachymening. cerebro-spinale. Diss. Bonn., 1871), Th. Simon (Griesing., Arch., Bd. I. and II.), Huss, in his work on Alcoolisme chronique avec démence, and more recently by Magnan and Bouchereau (Union méd., 1869). In the first cases the disease was manifested by neuralgias and feebleness of the lower limbs of long duration, and by apoplectic and convulsive symptoms with continually increasing dementia. In the varieties due to chronic alcoholism, inflammatory thickening of the cranial meninges was found, with rupture of cerebral and retinal vessels, which were affected by aneurismal dilatations.

Leyden has very recently published (Klin. d. Rueckenmarkskrank., 1874) a case of traumatic hæmorrhagic pachymeningitis, characterized by sopor and symptoms of cerebro-spinal meningitis (hyperæsthesia, stiffness of the muscles and neck); after an apparent improvement, death occurred at the end of a month, from gangrene of the lungs. At the autopsy the skull was found fissured; the inner surface of the spinal dura mater was covered with a slightly adherent false membrane, of a brownish-red color, and containing numerous extravasations. The cord, especially in its posterior part, was softened and strewn with a large number of bloody specks. Gangrenous foci, surrounded by pneumonia, were found in the lungs.

The *pathogeny* of hæmorrhagic spinal pachymeningitis is scarcely better known than its clinical characters. In the cerebral forms, in which alcoholism plays an important etiological part, the inflammation must be considered primary, according to the doctrines of Virchow (l. c.), and the hæmorrhages as consecutive to rupture of the delicate vascular meshes in the false membranes. In the spinal form of hæmorrhagic pachymeningitis the inflammatory reaction may give rise, in the same manner, to the formation of very vascular layers of connective tissue, and to frequent ruptures in their capillary meshes. Further investigations and observations are necessary in order to clear up the obscure phenomena of this disease.

B.—Diseases of the Spinal Arachnoid and Pia Mater—Spinal Meningitis.

Pathological Anatomy.

The inflammatory exudations of the spinal meninges are derived, in their most common form, from the pia mater, the arachnoid, or even from the inner layers of the dura mater. The exudation, which is either fibrinous or purulent, is thick and yellowish, occupies the loose tissue of the pia mater and arachnoid, and is often deposited upon the inner surface of the dura mater. According to the duration of the exudation the deeper coverings of the meninges are strongly injected, red and even infiltrated with small hæmorrhages. In chronic cases they are opaque, of a firm consistence, adherent and covered by false membranes. Osseous plates and masses of pigment are found in the arachnoid, but they have no pathological significance, on account of their frequency in old age.

The exudation is rarely limited and circumscribed, but is usually of considerable extent, and may occupy a large portion, if not the whole length of the cord. The same lesions may also involve the cranial meninges or, inversely, they may descend from the brain to the cord. The medulla oblongata is usually only slightly affected. The cord often appears pale and anæmic, but presents no tissue changes. In other cases it is very much congested, œdematous and softened, or atrophied and sclerotic. Similar changes may also occur in the posterior roots.

Etiology.

Inflammation of the spinal meninges may be *primary* and follow the action of intense cold, or severe injuries to the spinal column, even without fracture of the vertebræ. *Secondary* spinal meningitis occurs in caries, fractures or dislocations of the vertebræ, more rarely after bedsores, when the ulceration has extended into the sacral canal, or after extravasations of pus into the spinal canal (opening of a pulmonary cavity into the spinal canal, Cruveilhier, Gaz. hebdom., 1856), or from changes in the meninges caused by spina bifida. Cerebro-spinal meningitis, whose sporadic forms and epidemic manifestations have been described in the first chapter of this work, must also find a place here.

Spinal meningitis, and its combinations with cerebral forms, are observed most frequently in youth and old age. The male sex is more often affected than the female. When meningitis is epidemic, it often occurs, especially in children, as a complication of febrile diseases and acute exanthemata. After an operation for spina bifida, a spinal meningitis of sudden origin and rapid course may endanger the life of the patient.

Symptomatology.

On account of the frequent participation of the cranial meninges in inflammations of the coverings of the cord, the symptomatology of spinal meningitis is often very complex, and it is rare that the spinal symptoms are predominant. They are most distinct in the acute rheumatic forms

which are free from cerebral complications. The development of spinal meningitis is generally accompanied by febrile symptoms, acceleration of the pulse, elevation of the temperature of the body and delirium. Loss of consciousness and coma belong to cerebral complications. One of the first and most frequent signs is intense rachialgia which sometimes only occupies a few points and, at others, extends along the whole length of the dorsal region, is remittent or intermittent in character, and is increased by the slightest attempt at movement. In recent cases, cutaneous and muscular hyperæsthesia are present at the same time, and extend more or less over the trunk or lower limbs. In addition, clonic spasms occur in the muscles of the neck and back, with rotation of the head backwards, stiffness of the trunk, opisthotonos or orthotonos. The spasm in the neck denotes irritation of the cervical cord and of the spinal accessory nerves, whose fibres originate in the lateral columns of this region as low down as the sixth cervical vertebræ, and from the lower part of the medulla oblongata. When the painful muscular stiffness, which extends over a large portion of the trunk, is moderate in degree, it presents remissions and may even permit the performance of some movements. When this stiffness is persistent, the patient is rendered almost absolutely motionless. The muscles of the bladder and rectum also participate in these spasmodic phenomena, giving rise to the initial retention of urine and constipation. Painful irradiations, generally periodical, also occur, especially in the lower limbs, and appear to be of a neuralgic character. Dyspnœa develops in spinal meningitis of the cervical region, the muscles of inspiration being in a condition of painful rigidity. In cases which terminate in death, it is not rare to find pulmonary congestion and even hæmorrhages. The activity of the heart is increased as in fever. General elevation of temperature and abundant perspiration belong to the subacute forms. Gastric disturbances also may occur; vomiting and tympanites indicate that the brain is affected.

When the acute period has passed, more or less pronounced atrophy and contractures make their appearance, with paralysis of different groups of muscles, especially of the extensors. The columns of the cord or the nerve roots, which were compressed by the exudation, are not always altered to the same extent. As I have previously shown (in my Treatise on Electro-therapeutics, 1 Edit., 1865, p. 142–145), the severe forms of spinal meningitis present paralyses like those of progressive muscular atrophy (atrophy and paralysis of the thenar and hypothenar eminences, of the interossei, the muscles of the shoulder, and of the extensors of the arm and leg).

The motor and sensory excitability of the nerves is diminished to both currents on account of the separation of the nerves from their centres. The motor elements in the large (mixed) nerve trunks are affected more often and more seriously than the sensory elements.

The electrical current, although it produces no contraction, still acts upon the peripheral sensibility. All the muscles are not equally affected, since some of them contract under the influence of the will or of the electrical current, while others (most frequently the extensors) do not respond to these stimuli. This is due to the unequal action of the lesions in the central terminations of the nerve tracts. Finally, the power of motion may progressively improve and be re-established, while the electric excitability remains abolished, that is to say, the nervous paths have become permeable to the centrifugal stimulus of the will, but are still unresponsive to the inverse action of the electric current. The obstruction is

probably due to abolition of the excitability of the intra-muscular nerve fibres.

The extent and gravity of all these symptoms vary according to the intensity of the lesions. The recovery of the disease depends upon the same causes, and generally requires several months for its completion. Unfavorable cases, or those which are aggravated by complications, terminate in death at the end of several hours or two or three weeks. The character of the meningitis, its influence upon the cord and the centres contained in it, and upon the brain, play a decisive part in the termination of the disease.

Chronic spinal meningitis results from the prolongation of the inflammation and its sequences, and is characterized anatomically by thickening and pigmentation of the meninges, arachnoid, pia mater, and internal surface of the dura mater, which are adherent to each other and to the cord. This fibrous degeneration may cause atrophy of the nerve roots and, when the posterior roots are affected, may produce degeneration of the posterior columns. In other cases chronic parenchymatous myelitis develops, after a time, from the compression of the cord.

The *clinical signs* of chronic spinal meningitis are less clearly marked. The latent, insidious, and apyretic course of the disease, the difficulty and pain in movements of the vertebral column, the slowly developing paralyses of the limbs, and the irradiating pains with hyperæsthesia of the skin and muscles, remind us rather of a chronic medullary process.

We have already studied the epidemic form of cerebro-spinal meningitis in detail (p. 37–47) and refer to those remarks in order to avoid unnecessary repetition.

Diagnosis and Prognosis.

The diagnostic value of the symptoms is not readily mistaken in spinal meningitis due to a violent cold, to injuries of the vertebral column or to ulcerative lesions of the spinal canal. Epidemic cerebro-spinal meningitis is also characterized by its extension and by the number of cases. The *tubercular* form of cerebro-spinal meningitis (*vide* the observation by Magnan and Liouville, p. 30) can only be diagnosed with any degree of probability when symptoms of inflammation of the spinal meninges appear in individuals in whom inherited phthisis, scrofula, or tuberculosis of other organs or of the choroid (*vide* p. 145) can be demonstrated. The *differential diagnosis* from *typhoid fever* depends upon several factors. In the latter disease the tongue presents a characteristic appearance; the temperature is remittent and irregular in spinal meningitis; finally the termination of the disease dispels all doubt. Spinal meningitis is distinguished from *tetanus* by its initial fever, its frequent complication with cerebral symptoms, the rarity and slight intensity of trismus, and the tonic muscular spasms which are usually increased more by movements than under the influence of irritation. The differential diagnosis between *myelitis* and *chronic spinal meningitis* is furnished by the widely distributed muscular atrophy and the electrical phenomena which I have regarded as indicative of circumscribed compression of the cord and its nerves.

The *prognosis*, especially in young people of previous good health, is not always as unfavorable as Ollivier has concluded from his observations. The rheumatic forms are often susceptible of complete recovery, but this is rarer in cases of traumatic origin. In the *secondary forms* the termination depends on the primary affection, but it is rarely favorable. Chronic

spinal meningitis may terminate in more or less complete recovery after the lapse of a considerable time. As proof of this statement, we not infrequently find, upon autopsy, the remains of inflammation of the spinal meninges.

Cases which are severe from the beginning, those which begin with cerebral symptoms, the tubercular forms, and those in which respiration and deglutition become more and more embarrassed, generally terminate in death, which occurs most frequently during the first or second week of the disease. Finally, life may be endangered by inflammation of the bronchi, lungs, urinary passages, etc.; and, in unfavorable cases of chronic spinal meningitis, by secondary atrophy of the cord, and by the continued increase of paralysis.

Treatment.

In the beginning of the inflammatory period, local bleedings are indicated upon each side of the vertebral column, and even, in certain cases, from the mastoid processes. Venesection is only justifiable in robust individuals and in intense cerebral congestion. Benefit is derived, in traumatic cases, from cold local applications by means of an india-rubber bag filled with pieces of ice or ice-water, or by frequently renewed cold compresses.

At a later period the temperature of the cold applications to the back should be regulated according to the sensations of the patient. It will even be necessary to retain the compresses for a long time without renewing them. The distressing muscular spasms and the pains in the back and limbs are best combated by subcutaneous injections of morphine.

When all the symptoms of inflammation and irritation have disappeared, we should endeavor to accelerate the absorption of the exudations. Frictions to the back, with mercurial ointment, and the internal employment of iodide of potassium, are highly praised by a large number of physicians.

Warm baths are also useful and agreeable to the patients, as well as moist packings (until the return of an agreeable warmth), followed by warm half-baths and dorsal effusions. In cases of a slow course we should prescribe tonic regimen and country air, followed, at a later period, by the use of mineral waters and moor-baths. Electricity is useful in muscular atrophy and paralysis, galvanization of the nerves being alternately employed with faradization of the muscles.

II.—DISEASES OF THE PARENCHYMA OF THE CORD.

Thanks to the modern achievements of experimentation and histology, our knowledge of the diseases of the spinal cord has been greatly enlarged of late years. Clinical observations have rapidly increased in number, and, at the same time, we are better able to comprehend their symptomatology; though these diseases are presented to us under the most varied forms, we have better means of diagnosis and treatment at our command. We are, furthermore, continually progressing in this direction. All these reasons compel us to undertake an exhaustive study of these conditions in order to give the actual state of our knowledge on the subject.

Commencing with the circulatory disturbances of the cord, we shall then discuss the inflammatory processes and hyperplasiæ of the different portions of the spinal axis, and the typical morbid phenomena caused by these lesions. We shall then pass to the discussion of parasitic productions, tumors, and diathetic diseases of the cord. The anomalies in the development of the cord have more interest for the anatomist than for the clinical observer.

CHAPTER XVIII.

a. *Anæmia and Hyperæmia.*

In spinal anæmia, according to the facts at present at our command, the pallor is especially noticeable in the gray matter, which is richer in capillaries than the white substance. Only the large vessels of the pia mater still contain blood, although there may be congestion of the superficial venous plexuses. The consistence of the cord is increased in the majority of cases.

Tenner and Kussmaul (Moleschott's Unters., II. Bd., 1857) have produced anæmia of the cord by ligature of both subclavian arteries at their origin, after compression of the arch of the aorta. In the rabbits thus experimented upon, paralysis was noticed, after a short time, commencing in the posterior limbs and extending gradually to the anterior parts, so that finally the animals died from arrest of respiration. In 1867 Stenson made a series of experiments upon animals in whom he had tied the abdominal aorta, below the origin of the renal arteries and thus produced paraplegia with anæsthesia. According to Schiff (Centralbl., 1869), the cause of this paralysis was the spinal anæmia which was produced by the simultaneous obliteration of the spinal branches of the lumbar arteries. The excitability diminished from the centre to the periphery, together with the electro-muscular contractility, which persists for a much longer time. Paraplegias of a similar nature, but of a slower course, have been observed in man by Barth (Arch. génér., 1835) and by Gull (Dub. Quart. Journ., 1856) in consequence of progressive obliteration of the abdominal aorta.

Among the causes of spinal anæmia we may mention general debility, occurring after hæmorrhages and in patients reduced by suppuration, in chronic diseases and diatheses. The paraplegia experimentally produced by Panum (l. c), in consequence of embolism of the spinal arteries, with hæmorrhages and softening of the cord, has not been hitherto observed in

man. But, in discussing the diseases of the medulla oblongata, we have recognized thrombosis of the vertebral artery and the spinal arteries originating from it, and we must, therefore, admit the possibility of embolismic paraplegia. In most cases the general condition and the cerebral anæmia mask the symptoms of the spinal anæmia. But in certain cases the affection of the medulla and cord is shown by special symptoms, such as weakness and neuralgic pains in the legs (as in advanced tuberculosis and chlorosis), abnormal increase of reflex excitability, slow development of the power of co-ordination (in weak, rachitic children), dyspnœa, and palpitation of the heart, which are induced, in anæmic patients, on the slightest effort.

These disturbances usually disappear in great part under the influence of tonic and sustaining measures, and the cerebral and spinal functions are gradually restored, proving that the symptoms in question are not caused by organic lesions, but merely by disturbances of nutrition. We may employ these indications in prescribing a rational treatment.

Spinal hyperæmia has been previously considered in the article on hyperæmia of the spinal meninges, from which it is inseparable (*vide* pp. 177–179).

b. *Apoplexy of the Spinal Cord* (*Hematomyelie.*)

In addition to the extravasations into the spinal meninges which we have described above, hæmorrhages also occur into the tissue of the cord, and have been carefully studied in recent researches. Levier has devoted a monograph to the subject (Beitr. z. Path. d. Rueckenmarksapoplexie, Diss., Bonn., 1864), since which time other publications have appeared, among which we may especially mention the investigations of Hayem (Des hæmorrhagies intra-rachidiennes, Paris, 1872), who has thrown new light upon the clinical history and pathological anatomy of spontaneous hæmorrhage into the spinal cord.

From an anatomical point of view, spinal apoplexy is characterized by the extravasation of blood into the central axis of the cord.

According to Hayem, among thirty-one cases the foci were circumscribed in fifteen, and in the others the blood was infiltrated into the tissues. The hæmorrhagic foci, formed of the débris of nerve tissue, amyloid corpuscles, and fatty and pigmentary granules, are found in the gray matter, which is affected in certain portions of the gray horns or throughout its whole thickness. The extravasation may extend upwards to the cervical region and downwards to the terminal portions of the cord. More rarely the blood penetrates between the fibres of the cord.

According to the most recent researches, hæmorrhage only occurs into the tissue of the cord when it has been previously inflamed. Thus, according to Hayem, hematomyelie would be more correctly termed hematomyelitis. The few microscopical researches at our command also bring spinal apoplexy and myelitis into closer relationship. Charcot has observed considerable swelling of the nerve cells and axis cylinders, which he refers to a parenchymatous inflammation (W. Mueller has observed the same phenomena in acute traumatic softening). Liouville has found ampullary dilatations of the large vessels, with thickening of the walls and nuclear proliferation, in hæmorrhagic softening of the gray substance. In the completely paralyzed muscles, the striæ have disappeared, and granules have accumulated between the fibrillæ. The other muscles present a normal structure. The causes of hematomyelie are hyperæmia and vascular ruptures in the spinal system. They are most frequently caused by cold, vio-

lent efforts, sexual excesses and injuries. The cessation of the menses (Levier), in consequence of previous irritative causes, only gives rise to spinal congestion in an indirect manner. The miliary aneurisms of the capillaries, which play such an important part in the etiology of cerebral apoplexy, have not been hitherto observed upon the spinal vessels in hematomyelie. But the previously mentioned researches of Liouville serve to show that analogous vascular dilatations upon the larger vessels also exist in the cord. It remains for further investigations to demonstrate the relationship which exists between medullary apoplexy and the degenerations of the large arteries or arterioles of the cord.

On account of the frequency of the accidental causes which give rise to spinal congestion, the latter would be more frequently followed by hematomyelie, were it not for the fact that an alteration in the walls of the vessels must coexist in order to cause them to rupture. According to Levier and Hayem, men are much more subject to spinal apoplexy than women (undoubtedly on account of the differences in the habits of life). Cases are most frequent from the twentieth to the fortieth years. The invasion of the disease is almost always preceded by symptoms of spinal hyperæmia, under the form of circumscribed pains in the back, numbness, and formication in the fingers and toes, prostration, a feeling of fatigue, and stiffness in the movements. After these prodromata (which very readily pass unnoticed) have lasted for a longer or shorter interval, the symptoms of hematomyelie appear suddenly, with rapid development of paralysis of the lower or even of the upper limbs.

Consciousness, speech, and the functions of the special senses are frequently unaffected. It is only in abundant hæmorrhages of a rapid course, and especially in those of the cervical region (with whose vascular relations to the brain we are already acquainted), that momentary loss of consciousness occurs.

The paraplegic limbs most frequently, also, lose their sensibility. The anæsthesia usually extends upwards to the upper half of the back and of the abdominal region. Reflex excitability is preserved or even increased at the beginning, but it rapidly disappears when destruction of the cord occurs in a transverse direction, and especially in the gray matter. The paralysis also involves the sphincters; retention of urine and constipation occur in the beginning, but are soon followed by involuntary evacuations. The urine is usually alkaline.

The muscles which are insensible to mechanical stimulation and to the will, have also lost their electrical contractility. Colin has observed this abolition of muscular excitability upon the very day of the attack. Durian found it nine days, and Levier fifteen days, after the appearance of the characteristic symptoms. This peculiar phenomenon is explained by the disorganization of the muscular tissue, to which we have referred above The atrophy and degeneration of the muscles would undoubtedly assume still greater proportions if life were prolonged.

Other interesting phenomena also occur, such as constant elevation of temperature in the paralyzed limbs, which Schiff and Brown-Séquard had long observed in experimental lesions of the spinal cord. Abolition of cutaneous perspiration also appears in the paralyzed limb. The trophic disturbances consist of rapid atrophy of the paralyzed muscles, and, later, in the appearance of bed-sores and erythema.

The symptomatology varies greatly, according to the height at which the lesion is situated and to the extent of the hæmorrhage. When the extravasation occupies the cervical regions, the upper limbs are affected,

and stiffness of the trunk and contractions of the thoracic muscles occur If the hemorrhage approaches the medulla oblongata or the origin of the phrenic nerves, respiratory disturbances, such as a sense of oppression, dyspnœa, and embarrassment of diaphragmatic breathing, and dysphagia, develop. When the lesion involves the lower portions of the cord, paraplegia of motion and sensation occurs, with paralysis of the sphincters, cystitis, moderate priapism, etc. If the apoplectic foyer is limited to a lateral half of the cord (as in Monod's and Ore's cases), motor paralysis, elevation of temperature, and hyperæsthesia appear upon the side of the lesion, and preservation of voluntary motion and abolition of sensation on the opposite side (as in the spinal hemiplegia obtained experimentally by Brown-Séquard). The course of hematomyelie depends upon the situation of the extravasation and its extent. Abundant intra-medullary hæmorrhages are generally followed by death in a short period. At the end of several days, inflammatory reaction develops around the hæmorrhagic foyer (with symptoms of motor and sensory irritation), and thus renews all the danger. Less abundant hæmorrhages and those occurring in the lower segments of the cord run a more protracted course. The duration of the disease may, consequently, be counted by hours (thirteen hours in Moynier's case), by days, weeks, or even by months (six months in Trier's case).

When the disease rapidly terminates in death, this almost always occurs in the midst of disturbances of respiration and deglutition. When the fatal termination is delayed, it is caused by progressive extension of the paralyses, by abolition of the vegetative functions, by bed-sores, cystitis, pyelitis, and the final febrile exacerbations which accompany these complications. The course of chronic hematomyelie corresponds to that of chronic myelitis. In Hasse's case, the hemorrhagic foyer was surrounded by thickened connective tissue and partly filled with serum (like the apoplectic cyst of the brain).

The diagnosis of intra-medullary hemorrhage is based on the sudden appearance of motor and sensory paraplegia, elevation of temperature in the paralyzed parts, the absence of febrile and convulsive symptoms at the onset, and the rapid abolition of the reflex and electrical excitability of the muscles. The absence of fever and motor irritation, the prompt and general suppression of electro-muscular contractility, and the rapid atrophic degeneration of the paralyzed muscles, enable us to distinguish hæmorrhages from inflammatory lesions of the spinal meninges. In aortic embolism, which is an extremely rare cause of sudden paraplegia, the characteristic signs are the absence of pulsation in the femoral arteries, and the severe circulatory disturbances which soon appear in the lower limbs. The situation of the medullary lesion may be approximately determined from the symptoms to which we have above referred. In apoplexy of one half of the cord, motor hemiplegia and vaso-motor disturbances occur upon one side, and anæsthesia and preservation of motion upon the other.

The prognosis is extremely unfavorable in those forms of hematomyelie which are severe at the onset. Abundant hemorrhages in the neighborhood of the medulla oblongata or in the cervical cord are the most dangerous. The hæmorrhages in the lower portions are proportionately less grave. When the patient has recovered from the first shock, and the affection takes a chronic course, the danger continually diminishes. But, as in other forms of myelitis, complications may again endanger the favorable termination. With regard to treatment, we must refer to what has been already said on this subject under the head of apoplexy of the spinal meninges.

CHAPTER XIX.

MYELITIS AND ITS PRINCIPAL FORMS.

Modern research has made great progress in the pathology of the spinal cord, and we are to-day much better acquainted with its more minute anatomical lesions and with the multiplicity of its morbid forms. We also recognize more clearly their clinical manifestations and diagnostic signs. The inflammatory processes of the cord present a certain number of symptomatic types, according as they develop more or less rapidly, or as the degenerations are diffused or circumscribed. Although a perfectly scientific classification of spinal paralyses is not yet possible, we must, nevertheless, state that, thanks to the modern advancements of histology and clinical observation, the study of these affections is now based upon a positive anatomical and clinical foundation, in place of the obscure conceptions hitherto accepted. Myelitis assumes very different forms, according as its destructive action is acute or chronic. It is divided, in addition, into two principal groups, one of which consists of *interstitial myelitis*, including the primary affections originating in the neuroglia (myelomeningitis), and the peri-ependymal forms (Hallopeau); the second group is represented by parenchymatous myelitis, viz.: *the degenerations of the tissue of the cord* (that of the posterior or lateral columns, as in tabes and secondary ascending or descending degenerations), *the inflammatory changes of the gray columns*, and the *trophic disorders* (as in progressive muscular atrophy, spinal infantile paralysis, and other spinal affections). Parenchymatous myelitis may, in addition, affect (in the diffuse variety) the entire diameter of the cord, or it may attack, by preference, the anterior, posterior, or lateral portions of the cord.

A.—Acute Parenchymatous Myelitis.

The acute form of myelitis, which was known to the older observers, has very recently been the subject of profound anatomical and clinical studies. We may cite the works of Mannkopf (Congress of German Naturalists, Hanover, 1865), Engelken (Beitr. z. Path. d. acut. myelitis, Zuerich, 1867), Frommann (Unters. ueber norm. u. path. Anat. d. Rueckenm. Jena, 1867), and Dujardin-Beaumetz (De la myélite aiguë, Paris, 1872). The results of these investigations have received an important confirmation from the experiments of Hayem and Liouville, and have been increased by recent observations on acute myelitis occupying circumscribed portions of the gray substance.

The *pathological anatomy* of myelitis is especially indebted to the researches of Frommann, Mannkopf, Charcot, etc. In the beginning the medullary tissue is found swollen, with considerable increase in the volume of the vessels and cells of the neuroglia, hyperplasia of the reticulum,

development of granulations (causing irritation of the nerve-fibres during life), ampullary dilatations of the axis cylinders, and very marked hypertrophy of the cells of the anterior horns. At a more advanced stage exudative softening of the basement substance occurs, with formation of pus globules and granular cells at the expense of the neuroglia, granular degeneration of the nervous and interstitial tissue (granular disintegration of L. Clarke), and fatty and pigmentary atrophy of the nerve cells. In the extremely rare cases of recovery, retrogressive metamorphoses and absorption of the exudations may take place, with formation of cicatrices or cysts.

Rapid destruction of the medullary centres is very promptly followed by degeneration of the muscles, which assume a reddish, tawny color (like that of rare meat), and are softened and friable, or even dry and brittle (Rokitansky). Mannkopf and Engelken have observed nuclear proliferations in the sarcolemma. Fatty degeneration of the primitive fibrillæ and slight changes in the muscular nerves have been seen in rare cases. If the patient survives for a long time, the muscles may become subject to considerable atrophy. Hayem and Liouville have experimentally produced acute myelitis in animals, by placing iodine and glycerine upon the cord after opening the spinal canal. Paraplegia developed in consequence, with elevation of temperature in the paralyzed limbs, eschars and alopecia. According to Dujardin (l. c.) we may, in dogs, produce myelitis from traumatism.

In some very recent experiments, Leyden (Klinik. d. Rueckensmarkskr., II. Bd., 1875) has produced myelitis in dogs by the injection of a few drops of Fowler's solution, and has found disseminated foci of central peri-ependymal or endo-ependymal softening (suppuration and inflammation around or in the interior of the central canal). In some cases these foci were especially found at the periphery of the cord like a true peri-myelitis with all its histological characteristics. Acute myelitis may be due to violent chills, traumatisms (especially blows upon the back), and to exaggerated bodily exertion.

The influence of venereal excesses and of onanism is less demonstrable. External causes may sometimes be added to spinal lesions of slow growth, and tumors occupying the central portion of the cord may give rise to acute inflammation. Young and adult patients are most frequently subject to acute myelitis, but sex has no appreciable etiological influence. The symptomatology of acute myelitis permits of various descriptions, according as the inflammation originates in the central axis of the cord and then extends to the periphery, or whether it forms circumscribed foci, or especially involves the nerve cells of the anterior gray columns. The height of the lesion and its complications also modify the symptoms. I shall here narrate two personal observations, the first of which has already appeared in the first edition of my "Treatise on Electro-therapeutics" (1864).

1. A man, twenty-eight years old, fell down a staircase, striking upon the back, and immediately experienced a sharp pain in the dorsal spine, and soon afterwards in the lower limbs. After high fever and violent muscular pains during movements of flexion the power of motion and farado-muscular contractility progressively diminished during the course of the first month. At the commencement of the eighth week the paraplegia was complete, and the bladder was paralyzed. The electro-muscular contractility had only disappeared completely at the end of the second month of the disease. The patient died two days later, and the autopsy revealed complete softening of the cord in the upper part of the dorsal region, with impossibility of recognizing its structure (under the microscope, débris of nerve fibres, drops of myeline, and masses of

granulations were found). A short time afterwards an analogous case occurred in Frerichs' clinic, and was published by Mannkopf (Berl. Wschr., 1864).

2. In another more recent case, a young man, after exposure to severe cold, was seized with paraplegia, and soon afterwards with incomplete paralysis of the left inferior half of the face. The patient died at the end of five weeks. At the autopsy softening was found in the upper dorsal portion and in the posterior columns of the cervical cord, with spots of softening in the pons and in the posterior and external walls of the lateral ventricle.

Acute myelitis, developing in the gray axis of the cord, begins with fever, severe rachialgia with girdling pains, intense pain in the muscles at every movement, and rapid abolition of motion and electro-muscular contractility, soon terminating in complete paralysis. As a rule, the temperature of the paralyzed limbs is at first elevated. This phenomenon continues a short time, and is followed by disappearance of sensibility and reflex excitability. The initial retention of urine and fæcal matters soon gives place to incontinence.

Trophic disorders occur at a more advanced period, in consequence of lesions of the gray columns (especially the anterior, Charcot). They are characterized by muscular atrophy, acute bed-sores over the sacrum (in the course of the first or second week, according to Duckworth, Engelken, and Raymond), some disorder of the urinary excretion (alkaline and bloody urine), œdema of the paralyzed limbs, and effusions into the joints. Death may occur at the end of a few days, or in one or two months, from the ascending progress of the disease, with respiratory paralysis and asphyxia. At a later period death is due to cystitis, marasmus, bed-sores (with symptoms of pyæmia, or of meningitis, when the lesion advances into the spinal canal), hypostatic pneumonia, etc.

When acute myelitis is more circumscribed the symptoms are less severe and develop less rapidly. The fever is moderate, the symptoms of motor and sensory irritation are more prolonged, reflex sensibility is preserved (often even increased), and electro-muscular contractility is only partially diminished (especially in the extensors). The subsequent depression-symptoms of motion and sensation are also less extended and pronounced. The different varieties of sensibility are unequally affected, the paralyses are less complete and general, the trophic disorders are very rare and nearly always limited to the first period. After the acute stage the affection reveals the characteristics of chronic myelitis.

The symptoms also vary according to the height of the lesion. When the cervical cord is involved the chief symptoms are pains in the neck, inequality of the pupils, diminution of motion and sensation in the upper limbs, temporary dyspnœa, symptoms of angina and dysphagia. When the lesions predominate in the dorsal and lumbar regions, motor and sensory paralysis occurs in the lower limbs, in the abdominal muscles, the bladder and rectum, with impotence, and often with acute bed-sores (according to Ashhurst).

We shall discuss the symptoms of acute anterior myelitis, occupying the region of the anterior horns, at a later period, under the head of infantile spinal paralysis and its cognate forms.

The diagnosis of acute myelitis rarely presents great difficulties. Landry has described, under the name of *acute ascending paralysis* (Gaz. hebdom., July, Aug., 1859), a form of paralysis which rapidly extends from the lower to the upper limbs and to the medulla oblongata, and usually proves fatal from asphyxia. It sometimes disappears with a rapidity equal to that of its development. It is often attributed to an acute ascending my-

elitis. In other cases, however, no morbid change has been discovered, even on careful microscopical examination. If we may draw any deductions from the ill-defined data which we at present possess concerning Landry's paralysis, it may be distinguished from acute myelitis by the slight disturbances of sensation, the preservation of the electrical excitability of the muscles, the absence of motor irritation and of trophic disturbances, and the final embarrassment of speech. *Spinal meningitis* is characterized by the initial stiffness of the vertebral column, the cutaneous and muscular hyperæsthesia, the tardy appearance of paralyses, and the peculiar electrical reactions which we have previously discussed. The differentiation from *hematomyelie* is more difficult, and often impossible, for we have learned that the hæmorrhage occurs in a tissue previously softened by myelitis. Acute *hysterical paraplegia* is recognized by the preservation of faradic and galvanic excitability, with abolition of electro-cutaneous and electro-muscular sensibility, the frequent co-existence of anæsthesia of the mucous membrane and special senses, and by the presence of other hysterical phenomena.

The *prognosis* of acute central myelitis is very unfavorable, death usually occurring in the first or second months of the disease, with the symptoms to which we have above referred. The forms of acute myelitis characterized by circumscribed foci are less grave, the paralyses and sensory disturbances yield in part, and the patient passes into a condition of chronic myelitis. The least unfavorable prognosis belongs to the circumscribed traumatic inflammations of the cord. Flourens and Brown-Séquard have observed complete restoration of all the spinal functions, even after complete section of the cord, and the latter observer, in some microscopical examinations undertaken in conjunction with Robin, has found that the union occurred by first intention.

Masius and Vanlair have recently observed (Mem. de l'Acad. Roy. de Belgique, Bruxelles, 1870), in frogs a new formation of nerve elements, and restoration of motion and sensation to the normal condition, after excision of a portion of the cord, two millimetres in length. Dentan has obtained similar results (Sur la régénér. fonc. et anat. de la moelle épinière, Berne, 1873) in his experiments on young dogs. I have, also, often divided the lower part of the cervical cord in rabbits, by entering laterally through a small opening in the intervertebral space, and have seen the paraplegia, thus produced, disappear at the end of several days. In one of these animals, in whom the right anterior limb had also been affected, this leg remained in extension and abduction during walking for several weeks, although all signs of paraplegia had disappeared.

But, in man, the cord does not appear to be endowed with a similar power of restoration. We know that slight wounds or compression of the cord suffice to produce myelitis, which usually does not disappear completely, as we shall show further on in the section on traumatic lesions of the cord. In one of Ollivier's cases (l. c., T. I., p. 373), the paraplegia and paralysis of the sphincters, following a gunshot wound of the cervical cord, had disappeared, although paralysis of the left arm persisted six months longer.

In the *treatment* of acute myelitis, as in inflammation of the spinal meninges, we must rely chiefly on antiphlogistics and derivatives (intestinal). Cutaneous derivatives, moxæ, and setons must be avoided, on account of the frequent complication of the disease with cutaneous anæsthesia, and the tendency to eschars. Electricity, in its different forms, should not be employed, so long as inflammatory symptoms are still present.

Belladonna and ergot are highly praised by Brown-Séquard (Lect. on

the diagn. and treat. of paral. of the lower extremities, London, 1861). From the results of his experiments on dogs, in whom large doses of these two substances produced contraction of the vessels of the spinal pia mater, with diminution of the reflex excitability, Brown-Séquard recommends these drugs in the cases of spinal irritation in which the indication is to diminish the afflux of blood to the cord and to stimulate the nerve elements. This includes spinal hyperæmia, meningitis, and recent myelitis. The vascular contraction, which is probably due to irritation of the vaso-motor centres, does not appear to be very marked in extent or very lasting, since, after suboutaneous injections of concentrated solutions of ergotine in dogs, I have seen the tension of the blood barely rise above 40 millimetres.

Chronic myelitis, which develops after the symptoms of irritation and inflammation have disappeared, must be treated according to the principles which will be laid down in the following chapter.

B.—Chronic Parenchymatous Myelitis.

The large group of chronic inflammations of the cord is divided for the sake of convenience, into two categories, viz.: the primary and the secondary forms (diffuse sclerosis).

In *primary myelitis* the inflammation proceeds from the tissue of the cord itself. Prolonged or frequently repeated congestions may give rise to a primary hyperplasia, starting from the interstitial tissue of the cord. Continued stimulation of the nerve fibres and cells may also cause profound changes in them. The inflammation and its sequences chiefly involve either the columns of the cord or the gray matter, or both combined, and extend over a variable distance.

In *secondary myelitis* the lesions are due to external destructive processes (diseases of the vertebræ, perimeningeal and intermeningeal inflammations or hyperplasiæ, and intra-medullary neoplasms). Sometimes circumscribed foci form in the cord, but in other cases they occupy a large part of its transverse diameter.

Chronic Primary Myelitis.

Pathological Anatomy.

Intra-medullary inflammatory processes present the greatest analogy with the changes which characterize encephalitis. These processes, which are scarcely appreciable in the beginning, are not visible to the naked eye by any peculiar appearances, and the cord seems normal in volume and consistence. But, upon microscopical examination of the fresh cord, by teasing or in thin sections, we can observe a fibrillary network with nuclei penetrating between the nerve fibres, nuclear proliferation in the walls of the vessels and granular bodies in the ganglion cells.

After the cord has been hardened in alcohol and concentrated solutions of bichromate of potash, the diseased parts are, at first, colored more strongly, and become still more marked after the preparation has imbibed carmine or aniline blue; the section is finally cleared up by means of turpentine, poppy-oil, creosote, benzine, carbolic acid, etc. We may then recognize the proliferation of the neuroglia by the multiplication of the

nuclei and by the enlargement of the spaces between the transverse sections of the nerve-fibres. The vessels which are most diseased present thickening of the tunica adventitia and nuclear proliferation, and are very often obstructed by thrombi. At a more advanced stage of the degeneration the nerve tubes which are at first swollen, become shrivelled, the myeline is disintegrated and transformed into fat, and finally, the axis cylinder also disappears by atrophy. The gray horns are often unsymmetrical, and their nerves cells are opaque, of an œdematous pallor or opaline, with dark borders, deprived of their prolongations and containing abundance of fat or pigment. Lymphoid elements develop in the basement tissue, especially around the vessels; in rare cases we find purulent elements distributed over a large surface. These anatomical changes may extend over considerable segments of the gray substance of the cord, combined with swelling of the adjacent tissues. At a later period, after absorption of the exudation and nervous detritus, the gray substance is transformed into a red tissue (central softening of the cord, Albers) and is replaced by a fine vascular meshwork or by an oblong cavity formed of a laminated tissue (Rokitansky).

In other cases the degeneration is not limited to the central gray axis. It leads to the spontaneous formation of foci (sometimes multiple) which invade the transverse diameter of the cord to a greater or less depth. The cord then projects above the cut section and is softened. According to the duration of the lesion it assumes a reddish or grayish-yellow tint, and, under the microscope, shows débris of tissue, granular cells in abundance, amyloid corpuscles, pigment masses and destruction of the vessels. The softening of the tissue, caused by exudation, may also terminate in *granular disintegration* (Lockhart-Clarke). The tissue surrounding the vessels is, at first, transparent, and softened, with disintegration and segmentation of the nerve fibres. It then increases in fluidity and transparency, until finally the nervous and interstitial tissues become transformed into a finely granular matter or into an opaque and granular fluid. According to Michaud and others, this process is produced by an exudation from the neighboring vessels. Finally, fissures and canals are produced in the cord throughout the white and gray substance. Myelitis often terminates in sclerosis of the tissues. Upon cut section, the cord is found traversed to a variable extent by gray or yellow striæ (in an ascending direction) of a gelatinous or firm consistence. The network of the reticulum, formed of the elements of the neuroglia, has been transformed into large stellate corpuscles and the nervous elements have undergone fibrous metamorphosis. The hyperplasia of the ependyma of the central canal may also terminate in the formation of fibrous columns, in atrophy of the white and gray substances, and in the development of cavities in the cord (peri-ependymal sclerosis of Hallopeau).

The inflammatory processes may also involve the membranes of the cord, and the nerve roots. In chronic myelo-meningitis hypertrophy of the cells and proliferation of the nuclei occur, according to Frommann (l. c.), in the arachnoid and pia mater, around the vessels, in the neuroglia and in the cortical layers. The nuclei receive fresh protoplasm and form new cells, whose anastomotic meshes become more and more filled with nuclei and, by their increase, cause compression of the nerve fibres, the transformation of the myeline into a finely granular substance, transparency and pallor of the axis cylinders, and, finally, the appearance of large, stellate, varicose bodies. The nerve roots are often affected with neuritis and granulo-fatty change of the nerve fibres.

Etiology.

Among the etiological factors we must mention, in the first rank, debilitating social conditions, excessive exertion and exposure. The sudden cessation of the menses and of the perspiration of the feet should not be considered as causes, but rather as effects of a central vascular irritation (especially induced by exposure), with consequent disorders of the influence of the cord upon the secretions and excretions. Feinberg has demonstrated experimentally that the application of intense cold to the cord may produce myelitis.

Among the causes are also included traumatic lesions and concussion of the cord, especially described by Erichsen—the railway-spine of the English (with vertigo, irritation, weakness, paralysis, anæsthesia, etc.). In a case of this kind observed by Gore, Lockhart-Clarke found atrophy of the cord, especially upon its posterior circumference, and, furthermore, chronic inflammation of the cerebral and spinal meninges, with inflammatory lesions in the cerebral cortex. According to Ollivier, Hine, Leyden, etc., mental shocks, especially from fright, may also give rise to medullary paralyses. Certain forms of medullary affections often appear after severe diseases, such as typhoid fever, puerperal diseases, acute exanthemata, and diatheses. Neuritis may also extend to the cord and there produce profound changes, as Tiesler and Feinberg demonstrated experimentally a short time ago. They found spots of softening disseminated throughout the cord, after peripheral inflammations followed by ascending neuritis.

The male sex, which is more exposed to external noxious influences, generally furnishes a larger quota of medullary affections than the female sex. Among forty-four cases of simple myelitis, or of myelo-meningitis, collected by Brown-Séquard and Ramskill (loc. cit.), there were thirty-five males and nine females. But in the working classes in which women are not less exposed than men to the vicissitudes of life, the predisposition of the male sex to myelitis diminishes very much, as I have been able to convince myself by fifteen years' observation in the Vienna hospitals. The largest number of the victims of myelitis are found in middle age.

Symptomatology.

In the majority of cases myelitis develops in a slow and insidious manner. Slight, wandering rheumatic pains, an abnormal sensation of heat or cold, tingling or numbness of the limbs, and temporary sensitiveness, from time to time in the back—these are generally the first scarcely appreciable peripheral manifestations of central irritation, denoting hyperæmia and irritation of the gray matter. The latter, on account of its abundant vascular supply, is more predisposed to inflammation than the white substance. The first signs of sensory irritation then become more and more apparent. The dull transient pains of the neck or loins become more severe and persistent, and are often combined with a sensation of numbness in the thorax or abdomen. From time to time neuralgic pains appear and radiate into the limbs. The skin and muscles (especially over the vertebræ and intercostal spaces) present hyperæsthesia and exaggerated sensibility to pressure (*vide* General Characteristics, p. 171).

At a later period, when the degeneration of the cord extends in a transverse direction, the symptoms of sensory depression become more

and more prominent. The patient at first complains of numbness of the fingers (especially in the territory of the ulnar nerve) or toes and, later, he experiences in walking a sensation as if sand or wool were placed under the soles of the feet. At a more advanced stage of the disease, partial paralyses of sensation appear. Generally, as we have mentioned above, the sensibility to tickling disappears first, then the sensibility to contact and pressure, to temperature and finally to pain. Even when the sensibility appears to be intact, we may soon recognize, with the aid of the æsthesiometer, phenomena which differ markedly from the normal condition; or, if we touch the skin with a pen dipped in ink and direct the patient to indicate the points which have been touched, we will be able to determine errors of several centimetres.

The regions involved in the anæsthesia and analgesia extend from the inner surface, but more frequently from the posterior surface of the thighs to the hips and loins, and from the lower part of the trunk towards the lateral portions of the abdomen. Normal sensibility is again observed, after passing a narrow zone, in which sensation is merely dulled. Certain regions supplied by the sacral nerves (the inferior gluteal region, the perineum, and the genital organs), and certain portions of the limbs and neck sometimes escape the abolition of sensation. When the sensibility to pain is changed, cold water or a current of cold air may give rise to a sensation of scalding, or all the various modes of irritation may be converted into a feeling of tickling or numbness. In other cases retardation of perceptions occurs, or irradiation of sensations in an upward or downward direction, or even to the opposite side. Some of these peculiar aberrations of sensation are explained by the resistance to transmission, and by partial changes and obstruction in the gray substance; and others, by a concussion which is communicated by degrees to the central origins of the different nerves, whence they radiate directly to the periphery, or, by decussation, to the other side of the body.

In many cases of myelitis analgesia occurs without tactile anæsthesia. The exclusive abolition of sensibility to pain is due, according to Schiff's experiments, to a lesion of the gray substance; the loss of tactile sensibility with preservation of the sensibility to pain, to a lesion limited to the posterior columns. On the other hand, Brown-Séquard and Vulpian admit the existence of only a single system of conductors in the gray columns for the different varieties of sensibility. But as physiology has need of new explanations of these fundamental questions to which histology has hitherto furnished no solid basis, and as the anatomical lesions (*vide* a case of this kind under caries of the vertebræ) are not limited to the columns of the cord, or to the gray matter, but almost always involve the nerve roots as well, clinical observation has not been in a position hitherto to sanction either one or the other of the theories advanced. The motor disorders appear in the beginning as symptoms of peripheral irritation. They consist of muscular spasms, partial contractures, or choreiform tremor of the limbs, these movements being excited by exertion. Generally the symptoms of irritation soon give place to evident signs of depression, rapid fatigue in walking or standing, stiffness, and heaviness of movement, especially immediately after rest. At a later period one leg and then the other refuse to perform their functions, and both limbs are finally rendered incapable of supporting the weight of the body.

When the degeneration is propagated to successive layers of the parenchyma of the cord, the lesions, at the same time, involve the fibres of

the anterior roots, which (according to Gerlach) take a transverse course in order to enter the cells of the anterior horns, and also affect the longitudinal ascending fibres which pass into the antero-lateral columns to terminate in the motor ganglia of the brain. If the medullary degeneration is circumscribed, it may only attack a portion of the transverse fibres of the anterior roots and the gray cells which give origin to the root fibres of the upper and lower limbs. In other cases the lesion chiefly involves the ascending longitudinal fibres, and the communication between the gray substance of the brain and the anterior gray substance of the cord is interrupted. Pathological anatomy has also demonstrated that medullary paralysis may arise from transverse lesions of the gray matter, or of the white substance exclusively. But in almost all the important varieties of parenchymatous transverse myelitis, the degeneration also extends in a diffuse form to the paths of conduction which connect the brain and spinal cord.

The loss of motion extends from below upwards, more rarely in the inverse direction, according to the extent of the lesion and to the fibres which arise from the diseased part. Certain symptoms depend, in the first place, upon the situation of the medullary lesion. When it involves the *cervical* cord, motor and sensory paralysis occur in the upper limb and upper portion of the trunk, with inequality of the pupils, dyspnœa, disorders of deglutition and speech, palpitation of the heart, and irregularity of the pulse. Affections of the *dorsal* cord are characterized by increase of reflex excitability, and by spasm followed by paralysis of the sphincters and of the lower limbs. Lesions of the *lumbar* region are accompanied by painful sensations in the loins and along the sciatic or crural nerves, with genital excitement, followed by impotence, and symptoms of paralysis in the legs. In the majority of cases the degeneration of the dorso-lumbar portions of the cord is more marked, and the symptoms appear in accordance with the seat of the lesion.

According to the severity of the chronic parenchymatous myelitis, and its extent in a horizontal direction, the muscles become separated from their trophic centre, and undergo atrophy, their electrical contractility and sensibility diminish more and more (especially in the extensors of the thigh and foot), together with the galvanic excitability of the nerves. The reaction at the positive pole disappears more quickly than that at the negative. At the same time the reflex excitability, which was preserved at the beginning, also becomes involved, and is sometimes even increased (with movements in the antagonists, or even in remote groups of muscles). The deterioration of the electrical reaction, and the considerable diminution in active motion which almost always accompanies it, indicate that the myelitis is increasing in extent and intensity.

The genital functions are usually excited in the beginning, and frequent and complete erections occur. We very often observe, at an early period, a disproportion between sexual desire and its satisfaction. The ejaculation is then difficult or precipitate, frequent pollutions with exacerbation of the spinal pains, and gradual impotence occur. Cases of prolonged priapism are rarer. In the article on fractures of the vertebræ I shall report a curious case of priapism continuing thirty-six hours after death, from a lesion of the cervical cord. The functions of the bladder are generally disturbed from the beginning. There is frequent desire to urinate, especially after exertion, excitement, or irritation of the cord. At a later period there is frequent emission of a few drops of urine, the stream loses its force and continues to escape in drops after micturition. The patient is

then obliged to strain in order to urinate, or micturition even becomes impossible unless he is seated or lies upon the side in certain positions. Sometimes, after painful spasm of the bladder, the retention of urine becomes complete and recourse must be had to catheterism.

According to the latest experiments of Budge (Zeitschr. f. rat. Heilk., XXI., and Pflueger's Arch., II. Bd.) the emission of urine is produced by all the muscular fibres of the bladder and not by a sphincter proper. The muscles which prevent the flow of urine, viz.: the urethral muscle (also called constrictor of the urethra, or internal sphincter) and the bulbo-cavernosus belong to the urethral canal. The motor nerves of the bladder and muscles of the urethra are derived from their centres and pass through the cerebral peduncle, the restiform bodies, and the anterior columns, and escape from the cord through the third and fifth pairs of sacral nerves. The sensory nerves of the bladder are contained in the posterior roots of the third, fourth, and fifth pairs of sacral nerves. By reflex action these nerves maintain the tonus of the muscles of the urethra, whose contraction is also subject to the action of the will.

Experimental section and pathological lesions of the cord as high as the fifth sacral nerves, produce ischuria, from reflex increase of the tonus of the urethral muscles. After some time the fulness of the bladder and the pressure exercised by it upon the contracted urethral muscles give rise secondarily to incontinence. If, on the other hand, the lesions of the cord are situated lower in a position in which the conditions of the reflex act do not exist, a true paralysis of the muscles which close the bladder occurs, the urine escapes drop by drop, the action of the vesical muscles meets no further obstruction, and the incontinence is then primary.

I have several times seen febrile movements develop in the course of chronic myelitis, with an elevation of temperature appreciable to the thermometer, loss of appetite and heavily coated tongue. This febrile condition is usually due to exposure or over-exertion, and I have seen it considered as a walking form of typhoid fever. It is merely an exacerbation of the inflammatory process, increased by external influences. It lasts a short time, but is usually followed by a more rapid deterioration of the motor and sensory faculties.

The disease only passes into the last stage after the lapse of several years. Motion and sensation are then paralyzed, the malnutrition of the muscles advances more and more towards the upper parts, and the paralysis of the sphincters causes involuntary evacuation of fæcal matters and of urine. The latter accumulates and decomposes in the bladder, purulent cystitis develops, with dilatation of the bladder and pelves of the kidneys, sometimes accompanied by chills and uræmic symptoms (vomiting, diarrhœa, cerebral symptoms). Patients suffering from myelitis usually perish at the end of several years, with bed-sores over projecting bony parts which are most exposed to pressure, and in a condition of marasmus and continually increasing paralysis, or they are carried off by acute inflammatory complications.

Diagnosis and Prognosis.

In order to recognize the initial symptoms of myelitis we must pay strict attention to the first peripheral symptoms of medullary irritation. These are the vague (neuralgic) pains, which are often unrecognized or wrongly interpreted, circumscribed sensations of cold or numbness in the limbs, circumscribed anæsthesia or analgesia and disseminated hyper-

æsthesia. We must pay equal attention to the abnormal excitement of the genital organs and the seminal losses followed by exacerbation of the spinal pains.

At a later period the nature of the disease becomes more evident, the disorders of sensation become more widely distributed and occupy the cutaneous territories of the spinal nerves, and the paraplegic character of the paralysis becomes more marked. If we carefully consider the mode of development of these symptoms, we will not readily mistake the disease under consideration for other paraplegias.

Cerebral paraplegia is often produced by two distinct hemiplegias, and is complicated by paralyses of the cranial nerves, especially in lesions of the median portions of the pons, or of the base of the brain (*vide* p. 51). *Paraplegia from spinal apoplexy* is recognized by its sudden onset, by the rapid loss of voluntary and reflex motion, and of electro-muscular contractility. *Paraplegia from spinal meningitis* is characterized by its febrile onset, the painful stiffness of the muscles, and by the tonic spasms and the peculiar electrical reactions to which we have previously called attention. We shall reserve for a future period the differentiation from *paraplegia due to compression of the cord*, and the so-called *reflex paraplegia*. Finally, we can readily distinguish *hysterical paraplegia* by the coexisting anæsthesia of the skin, mucous membranes and deeper organs, by the abolition of electro-cutaneous and electro-muscular sensibility, by the preservation of farado-galvanic excitability, and by the existence of other hysterical phenomena.

Primary chronic myelitis almost always advances to a fatal termination, and cases of complete and permanent recovery demand rigid scrutiny. The term chronic myelitis is often employed to include reflex paralyses caused by slight exudations into the spinal meninges, by stasis of the venous plexus in the pelvis, by inflammations propagated to the intra-pelvic cellular tissue or by diseases of the abdominal organs, and these cases are then complacently reported as examples of recovery. But in the majority of cases of myelitis, we must remain satisfied with more or less improvement in the disorders of motion, sensation, and nutrition.

We may state, in general, that the lower the site of the myelitis in the cord, and the more the symptoms indicate that the lesion is extending slowly across the transverse diameter of the cord, the longer will be the duration of the disease. In certain severe forms all therapeutic measures are unavailing. These cases last two or three years, while the more benign forms may continue six or eight years and even longer. The patients usually die, as we have stated above, from marasmus, from the effects of bed-sores, and from cystitis with pyelitis, which sometimes gives rise to diphtheritic or gangrenous softening of the mucous membrane and to hæmaturia. The death of the patients may also ensue from pneumonia, tuberculosis, etc.

Treatment.

Energetic counter-irritation (wet cups, setons, moxas, etc.), which is highly praised in recent forms, is ineffectual against deeper inflammatory changes in the cord. The only results obtained thereby are numerous unpleasant cicatrices. Iodide of potassium may have a favorable influence on the absorption of meningeal exudation, but it is acknowledged that it is ineffectual in the treatment of inflammatory and hyperplastic processes in the cord itself.

Brown-Séquard recommends the employment of belladonna and ergotine, on account of the contraction of the vessels which they produce in hyperæmia of the cord (*vide* p. 199). I have often employed these drugs for a long period in the congestive forms of myelitis, without having been able to observe any appreciable diminution in the irritative symptoms. Strychnine is contraindicated in these cases, on account of the hyperæmia to which it gives rise. It is more serviceable as a stimulating agent in symptoms of depression. But as the strychnine has no effect upon voluntary movements and only excites reflex action the patients can derive little benefit from its exclusive employment.

At present we employ more commonly and with more success the thermo-hydropathic and electrical measures of treatment. The warm mineral waters (Wildbad, Gastein, Teplitz, Ragaz, etc.) are especially useful against the neuralgic pains, the spasmodic phenomena, and those forms of the disease which are accompanied by stiffness of muscles and unpleasant sensations of cold. Cool baths have a more tonic and invigorating action. Their temperature should be gradually lowered to 22° or 18° C., and frequent affusions should be made to the back. The stimulating action of the temperature upon the numerous sensitive nerves of the skin is transmitted to the brain, and to the medullary and vaso-motor centres, and reinforces the reflex acts presiding over the circulation, respiration, and the functions of the abdominal viscera.

The diameter and rapidity of the vascular currents traversing the skin differ very much, according to the actual temperature of the integument, on account of its action upon the vascular muscles. According as the bath dilates or contracts the vascular meshes in the skin, the mean pressure of the blood, and the number of blood globules which carry oxygen to the other organs, will diminish or increase, and these phenomena exert a great influence upon the nutrition of the tissues, and upon the elimination of excrementitious matters. On the other hand, the more the temperature of the bath is lowered the more the abstraction of heat by the water will act upon the development of heat by the current of blood. The stimulating action of the temperature upon the nerve fibres will be felt so much the longer, the more the temperature of the nerves deviates from that of the surrounding medium. According to Osborne's experiments (Dubl. Quart. Journ. of Med. Science, 1862), water possesses a refrigerating power twelve times greater than the air; it is, therefore, twelve times colder for the body, all things being equal, than air at the same temperature. The stimulating action of cool baths upon the nervous system will be so much the more intense, the weaker and more impressionable the latter becomes (and this is especially so in myelitis).

Hydropathic treatment with cool half-baths may be often preceded by the following measures, viz.: wet-packs (especially in neuralgic and spasmodic symptoms), or frictions with wet cloths (as a weaker stimulant). We thus produce dilatation of the vascular canals of the skin, which soothes and calms the irritation of the deeper organs.

With regard to electrical treatment, we may recommend the cautious application of ascending galvanic currents along the vertebral column, and from here to the nerve trunks (3–5 minutes). Currents which produce strong and painful contractions and the tension current of the induction coil, must be avoided in conditions of great irritability of the centres. In the treatment of paralysis of the bladder I have found the greatest advantage from galvanization by introducing one electrode into the rectum, and by applying the other to the symphysis pubis or perineum. Direct electrization by means of the vesical electrode is usually too irritating. Benefit is also derived from combining moist frictions, ascending douches to the perineum and douches to the lumbar region.

Symptoms of Secondary Myelitis (from compression).

We here refer to those forms of spinal paralysis from compression, in which neoplasms which originate externally, or which occupy the osseous canal, the meninges, or even the cord itself, exercise upon the latter a continually increasing pressure, and thus give rise to and maintain a chronic degeneration of its parenchyma. The cause of this compression has less influence upon the symptomatic manifestations than the situation and mode of propagation of the consecutive central changes.

The anatomical changes which secondarily extend their destructive action to the cord, affect both the site of compression and also certain bundles of fibres which, starting from a morbid focus limited to the cord or the nerve roots, extend along the whole length of the spinal axis. At the point of compression the cord is atrophied, sclerosed, and the nerve fibres are more or less completely destroyed. Starting from this primary focus the degeneration follows the different columns of the cord in an ascending or descending direction, and diminishes in extent and intensity from its origin to the extremities of the cord.

These secondary changes were first thoroughly studied by Tuerck (Sitzb. d. Wien. Akad. d. Wiss., Bd. XI., XVI., u. XXI.) in cases of compression of the cord from lesions of the vertebræ. In those portions of the cord situated above the lesion the anterior columns present their normal appearance, or are only involved at the point of disease. The posterior columns are affected by an ascending degeneration, either in their totality or for a considerable distance, or only in their internal segment. On the other hand, in that portion of the cord situated below the lesion, the posterior columns are intact, and the degeneration only affects the anterior and lateral columns, and sometimes only the posterior portion of the latter. Cornil and Lange have observed ascending degeneration of the posterior columns in cases of tumors compressing the posterior roots.

Westphal has obtained these secondary degenerations experimentally in dogs (Arch. f. Psych., II. Bd.), by perforating the cord without opening the spinal canal. In the lesions of this nature, which chiefly affected the posterior columns, the adjacent gray substance, or the internal portions of the antero-lateral columns, the posterior columns were also affected by ascending degeneration, and the antero-lateral columns by descending degeneration. Westphal (l. c.) has observed another peculiarity in man in cases of tumor compressing the cord. He has seen upon transverse section of the degenerated portions, and especially of the posterior columns, ring-shaped or oval figures, which were rendered more distinct by staining with bichromate of potash, and often enclosed another central point of degeneration. The clinical history of prolonged compressions of the cord may vary very much, according to the situation and violence of the primary lesion, and also according to the number and intensity of the symptoms. Most frequently the first morbid signs which make their appearance are disturbances of sensation. They consist of tingling, pruritus, a feeling of constriction, numbness, etc., which denote irritation of the nerve roots, or of their prolongations into the gray substance. By careful examination (by means of the æsthesiometer) we are soon able to recognize anomalies of cutaneous sensibility, and of its different varieties. I shall describe, in detail, under the head of vertebral caries, a personal observation of analgesia without tactile anæsthesia, with the results of

the autopsy. As we have already stated, retardation of perception or irradiation of sensibility may occur in other cases. Complete abolition of sensibility in the limbs is only observed in transverse degenerations of the gray substance, and in lesions of the gray horns. The anæsthesia, as we shall show by several examples, occupies certain definite regions, corresponding to the distribution of the cutaneous nerves. When anæsthesia dolorosa (painful anæsthesia) occurs, the peripheral terminations of the nerves may be degenerated, although the central portions still receive stimuli and transmit them to the gray substance.

Among the symptoms of sensory irritation we may mention hyperæsthesia (cutaneous and muscular), the peculiar anomaly described by Charcot under the name of dysæsthesia (very painful delayed sensations, like the tingling experienced after pinching, and the local applications of cold), and different varieties of neuralgia. When the degeneration occupies the upper portions of the cord the pain may assume the appearances of a cervico-occipital, cervico-brachial, or intercostal neuralgia. When the lesion is situated further down, crural, sciatic, or abdominal neuralgias will result. In cases of extra-medullary compression (exceptionally in intra-medullary lesions) severe, sharp, pseudo-neuralgias, described by Cruveilhier and more recently by Charcot (neuritis of the nerve roots or peripheral branches often) occur, and are characterized by the absence of tender spots on pressure, and by their combination with trophic disorders. Finally, these authors have observed, especially in cancer of the lumbar vertebræ, a paraplegia dolorosa (periodical pains of extreme intensity, increased by active or passive motions, and accompanied by cutaneous hyperæsthesia.

The first symptom on the part of the motor nerves consists of paresis, which soon changes to paralysis. We also observe spasms, muscular rigidity, permanent contracture of the paralyzed limbs (from sclerosis of the lateral column, according to Charcot), and epileptiform attacks. The latter consist of convulsions of the lower limbs, produced especially by passive movements (forced extension of the toes), rarely spontaneously, and may be explained by exaggerated stimulation of the gray substance, with diminution of the inhibitory action of the brain. These symptoms, termed spinal epilepsy by Brown-Séquard, are better described as reflex spinal spasm, reserving the term spinal epilepsy for those cases (sometimes seen in caries or wounds of the vertebræ) of general convulsions with disorders of consciousness. Brown-Séquard has experimentally produced these phenomena, the centres of which are found in the medulla oblongata, and in which the cord only acts as a conductor. The paralysis may be at first confined to one of the upper or lower limbs, on account of a unilateral compression of the nerves of the brachial plexus, or of the cauda equina. Spinal hemiplegia follows compression of the antero-lateral column of the cord on the same side, or of the medulla oblongata on the opposite side. Crossed hemiplegia with hemianæsthesia is a very rare form, observed by Charcot and Gombault in cases of tumors occupying one side of the cord, as we shall illustrate by examples.

Paraplegia is the most frequent form of spinal paralysis from compression. It usually occupies the lower limbs and is, for a long time, combined with a slight alteration of sensibility and increased reflex excitability. Cervical paraplegia is much rarer and is due to various causes of medullary compression. It has been especially investigated by Gull (Guy's Hosp. Reports, IV., 1858), and more recently by Charcot (Leçons sur les maladies du syst. nerveux, 2e fasc. 1873). As this double paraly

sis of the arms possesses great scientific and diagnostic interest, it will not be unprofitable to analyze the experimental and clinical data at our command. Some of them have come under my own observation, others belong to different observers.

If we extirpate the first vertebræ in frogs, at the height of the upper portion of the cervical enlargement, and remove a portion of the cord towards its anterior surface and extending from one side to the other, the animal will present a deflection of the head and paralysis of the two anterior limbs. The latter are directed inwards during the act of jumping, and their sensibility to mechanical and electrical irritations is markedly diminished. At the end of eight to ten days the motor power of the fingers begins to improve. Microscopical examination of the hardened cord shows a hyaline exudation deposited between the nerve fibres, and pus globules scattered in large number throughout the gray substance, and grouped in places around the central canal. We may also reach the anterior cervical cord in rabbits by the same procedure, and dyspnœa, hyperæsthesia and paralysis of the two anterior limbs, or even of the posterior extremities are thus produced. In rabbits we may act upon the anterior portion of the cervical cord through either the anterior or posterior surface of the vertebral column. In incision or excision of the cord from the anterior surface, great difficulty is experienced in the extirpation of the corresponding bodies of the vertebræ, and the operation is also more difficult and dangerous on account of free hæmorrhage. If the cord is only incised upon one side, hemiplegia will occur upon the same side of the body. If the incision of the cord passes the median line and involves the two lateral columns, paralysis will occur in the two anterior limbs, often with paresis of one of the posterior extremities.

The paralysis of the anterior limbs is usually incomplete, and chiefly involves the extensors, the flexors being less affected. In most cases the animal is immediately seized with intense dyspnœa, soon followed by partial tetanic spasms of the extremities and, finally, by epileptiform convulsions. Death occurs at the end of twenty-four to thirty-six hours. Upon autopsy a considerable intrameningeal, often also intramedullary hæmorrhage, is found at a point corresponding to the lesion.

The operation is less difficult and serious when performed through the posterior surface of the vertebral column. The spinous processes of the upper cervical vertebræ are raised, and a curved india-rubber needle is directed laterally upon the cord, avoiding the lateral venous plexuses. The cord is then cautiously drawn out, and incision or excision is performed with a fine knife, which should be curved on the flat. This operation is followed, as we have stated above, by incomplete paralysis of the anterior limbs, or even of one of the posterior extremities. Disturbance of reflex action also often occurs. If one of the sciatic nerves is exposed and stimulated with electricity, contractions will be produced in both posterior limbs, while the anterior ones remain quiet. If the median nerve is stimulated, the corresponding extremity will alone contract. At the autopsy a hæmorrhage is found in the cervical region, starting from the transverse section of the cord, and leaving only a few thin lamellæ of tissue intact. In the portions below this it is confined to the gray substance and further back, merely a few hæmorrhagic specks are visible (Med. Jahrb., T. IV., 1876).

Clinical observation agrees with experimental investigation, and has shown that in circumscribed myelitis of the cervical region paralysis first occurs in the upper limbs, and later in the lower limbs, with disturbances of sensation and reflex excitability. Acute myelitis of the cervical portion, from fracture or dislocation of the first vertebræ, or from the presence of tubercles in the gray substance of that region (Budd), may begin with isolated paralysis in the upper limbs. In cases of compression from caries of the first dorsal vertebræ, degeneration may occur in the two lateral columns extending upwards to the cervico-brachial enlargement, and may produce, according to Charcot and Michaud, paralysis and contracture of the upper limbs. Finally, atrophy of the cells in the anterior gray horns of the cervical region, which serve as the origin of the root fibres of the upper limbs, may be followed by paralysis of both arms.

Cellular atrophy and the consequent paralysis run either an acute or chronic course, and their clinical manifestations vary accordingly. Thus

Raymond (Progrès Medical, 17, 1875) has published a case of central myelitis, followed by death at the end of twelve days, in which complete paralysis of the left arm (with diminution of faradic excitability) and incomplete paralysis of the right arm had occurred during life. At the autopsy the cervical enlargement presented sclerotic lesions, the cells of the anterior horns were wanting in places, and the interstitial tissue showed a moderate degree of nuclear proliferation. Infantile spinal paralysis results, according to the investigations of Prévost, Charcot, Joffroy, Recklinghausen, Roth, and Leyden, from an acute degeneration of the cells of the anterior gray horns. In a case of this disease (which will be reported hereafter) I have observed paralysis and atrophy of the right arm, and less pronounced symptoms in the left arm. A similar disease has been observed in adults, during the last few years, by Duchenne, Frey, Bernhardt, Erb, etc. According to Gombault, Cornil, and Lepine, it is due to fatty or pigmentary degeneration of the cells of the anterior horns, and is sometimes manifested by paralysis of both upper limbs, an illustration of which I shall describe at a later period. There is still another form of cervical paraplegia, due to atrophy of the cells of the anterior gray horns. This includes the double paralysis of the arms which occurs in the beginning of amyotrophic lateral sclerosis and in certain forms of progressive muscular atrophy. The cases of paralysis belonging to this latter category may be considered as amyotrophic forms of spinal cervical paraplegia.

A third form, due to diseases of the peripheral nervous system, remains to be described. In these cases the diplegia brachialis may be caused by an extravasation of blood around the upper nerve roots (Ollivier, Schuetzenberger), or by caries of the cervical vertebræ with consequent external pachymeningitis, when the morbid products compress the nerves of origin of the brachial plexus on both sides. Finally, it may be due to internal spinal pachymeningitis (hypertrophic, of Charcot), which exercises circular compression of the cord. Side by side with these peripheral forms is a central variety which belongs to the preceding categories, according to Joffroy (Sur la pachyméningitis cervicale hypertrophique, Paris, 1873). He has found myelitic changes, especially in the cervical enlargement, and almost always with the formation of lacunæ in the medullary tissue.

We must make a careful analysis of all the symptoms in order to make a differential diagnosis between the various forms of cervical paraplegia. Peripheral cervical paraplegia from compression presents the characteristics of a neuritic paralysis. At the onset, pains and hyperalgesia occur in certain nerve trunks, followed by anæsthesia, abolition of reflex excitability and paralysis with muscular atrophy. In the beginning the electrical excitability is increased and afterwards abolished, the farado-muscular contractility is diminished, the galvano-muscular contractility is increased. In cervical paraplegiac aused by compression of the cord, the paralyzed limbs remain for a long time without any noteworthy change in volume, electrical reactions, and sensibility. The reflex excitability is usually increased; when it is less marked, and after it has entirely disappeared, we may observe from time to time painful spontaneous contractions (spinal reflex spasms, from irritation of the gray matter, with diminution in the inhibitory action of the brain).

The ordinary forms of amyotrophic cervical paraplegia are easily recognized. In the anterior myelitis of adults, the diagnosis depends upon the following symptoms: febrile attack, rapid paralysis of the legs, then of the arms, early atrophy of the muscles with the electrical reactions previously mentioned, absence of disturbances of sensation of the geni-

tal functions and of the sphincters, with progressive improvement of motion.

When the spinal paralyses from compression increase in extent and intensity, the farado-muscular contractility and sensibility diminish more and more, together with the galvanic irritability of the muscles and nerves, at first upon making and breaking the current at the anode, then upon making it at the cathode, and finally contraction only occurs with an inverse direction of the current from the anode to the cathode (with the aid of a strong current). The compressions of the cervical cord or of the adjacent portion of the dorsal region also present other signs, such as oculo-pupillary disorders, and disturbances of the cardiac and gastric fibres of the pneumogastric nerves. The *oculo-pupillary disorders* consist most frequently of a paralytic *myosis* (contraction from interruption of conduction, followed by dilatation), and of a spasmodic *mydriasis* (dilatation from medullary irritation of the dilator of the pupils). These symptoms may alternate, and are sometimes observed in both eyes, sometimes only on one side. Pathological observations of this character have been reported by Ogle, Eulenburg, Rendu and myself. According to Budge's and Cl. Bernard's experiments, the centres of innervation of the iris extend in the cord as low as the second dorsal vertebra. According to Salkowski, their upper limit, which is less clearly defined, is above the atlas.

The persistent slowing of the pulse, from irritation of the cardiac fibres of the pneumogastric, forms a very interesting, but not well understood phenomenon of cervical lesions. I have described one case (Zeitschr. f. pract. Heilk., 46, 1866) which has all the positive value of a vivisection. A boy, 15 years old, who had received a blow with a knife at the level of the sixth cervical vertebra, had temporary disturbance of consciousness and right hemiparesis, which disappeared at the end of twenty-four hours. Dilatation of both pupils, but especially of the left, was then observed and, for four weeks, the pulse varied from 56–48 beats per minute. Three months later the recovery was complete.

Almost at the same time, Landois (Centralbl. 1865) demonstrated that compression of the superior vena cava produced venous congestion of the medulla oblongata and brain, and considerable retardation of the pulse. When the cerebral congestion is more marked, the slowing of the pulse may even advance to complete arrest of the heart's action, and may be complicated with epileptiform attacks. Halberton's patient (Med. Chir. Trans. 24, 1841) experienced, in consequence of a fall, pain and difficulty in the movements of the head. During the following year he presented persistent slowness of the pulse (33–15 beats per minute), especially during epileptiform and syncopal attacks. At the autopsy the medulla oblongata was found flattened, indurated, and compressed by a narrowing in the upper part of the spinal canal. The articulations of the atlas and occipital bones were anchylosed, and the dura mater thickened.

We have already spoken of the dyspnœal symptoms following compression of the upper portions of the cord. They are explained by lesions of the phrenic, intercostal and cervical nerves, which are contained in the cervical and dorsal regions of the cord. When the abdominal respiration is entirely preserved it is evident that the compression has not involved the diaphragmatic nerve, the principal trunk of which passes through the fourth cervical nerves. Hiccough, disturbances of deglutition and gastric disorders (pains and frequent vomiting) are also observed in compression of the upper parts of the cord. Epileptiform attacks occur very rarely in man from compression of the cervical cord (Duménil, Leudet, Bouchard, etc.)

When the compression involves the lower parts of the cord, paralysis

of the bladder and rectum (which we have previously discussed) is added to the paraplegic disorders. In these cases, also, we may observe absence of erections, or the paralysis of the vascular muscles and the venous distention of the corpora cavernosa and glans may give rise to the production of priapism. In the following chapter, under the head of fractures of the vertebræ, I shall refer to a case of priapism which persisted thirty-six hours after death.

Finally, spinal paralyses from compression are sometimes accompanied by trophic disorders, such as the production of herpes along the distribution of certain nerves, in compression or inflammation of the nerve roots and in degeneration of the cells of the spinal ganglia (Baerensprung, E. Wagner), the acute bed-sores which we have mentioned so frequently (Samuel., Charcot), acute articular inflammations (Mitchell), hydrarthroses (Michaud), and the trophic changes in the muscles which we have previously investigated.

The diagnosis of paralysis from prolonged compression of the cord depends upon the manner of development of the symptoms, and upon disturbances which indicate a circumscribed anatomical lesion. A clinical analysis of the phenomena will more readily enable us to determine the situation and extent of the compression than the character of the primary affection, the symptoms being identical in most cases. We shall study the symptoms of paralysis from compression more in detail in the chapters on the individual varieties.

In order to pass in review the different varieties of secondary myelitis (from compression), we must consider the multiple morbid processes which may develop in the organs surrounding the cord or in the cord itself, and following the anatomical order, we shall proceed from without inwards. Commencing with affections of the vertebræ, we shall take up the consideration of perimeningeal processes, then the intra-meningeal inflammatory products, tumors and parasitic growths, and, finally, intramedullary neoplasms and diathetic spinal diseases.

CHAPTER XX.

COMPRESSION-MYELITIS, FOLLOWING DISEASES OF THE VERTEBRÆ.

In the following remarks we shall only discuss those vertebral diseases which give rise to secondary lesions of the cord or spinal nerves. The most frequent and important morbid processes are caries, fractures, and dislocations of the vertebræ; then follow spondylitis deformans, atrophy from compression (in consequence of aneurisms and hydatid cysts), and cancer of the vertebræ.

A.—Caries of the Vertebræ (Spondylarthrocace).

In the latter part of the last century, Pott had given a clear description of this disease, which may terminate in paraplegia and in softening and inflammatory destruction of the vertebræ. The meninges and parenchyma of the cord are, also, often attacked by the diseased process.

In young subjects, the osseous tissue of the vertebral bones undergo inflammatory softening and necrosis, in which the periosteum and joint surfaces likewise participate. This results in relaxation and mechanical compression during movements which keep up the inflammation and terminate in giving way and displacement of the vertebræ. The pus is thick and cheesy, and appears in foyers or collects towards the dependent portions. In older subjects the affection begins in the intervertebral articulations or in the transverse apophyses. Chronic synovitis occurs, and the inflammation then attacks the ligaments, the periosteum, and even the bones, terminating most frequently in destructive caries of the cartilages and bones, the vertebræ being hollowed out and dislocated. The inflammatory softening not only affects the joint surfaces, but also the vertebral bodies, which become transformed into a cheesy mass containing debris of osseous tissue.

This carious destruction and displacement produces, at a later period, kyphosis with an acute or obtuse angle. Despite permanent deformity, recovery may occur from arrest of the inflammation and the return to a more favorable condition of nutrition. The softened osseous tissue regains its normal rigidity and firmness, the vertebral column recovers its original solidity and again becomes capable of supporting the weight of the body.

One of the most important phenomena in this affection is the participation of the spinal meninges, the spinal nerves, and the cord itself in the carious process. It is rare that the meninges, and especially the dura mater (as we have shown above) remain completely intact. According to Michaud (Sur la méningite et la myélite dans le mal vertébral, Paris, 1871), these inflammatory phenomena are due to the cheesy suppuration of the carious vertebræ, which, by destroying the posterior ligament, causes the pus to come in contact with the external surface of the dura mater. Inter-

stitial abscesses and considerable thickenings (external pachymeningitis) may also develop in the same manner. In rare cases, the dura mater is detached or its infiltrated layers project towards the inner surface. The nerve roots present the lesions of neuritis with granulo-fatty degeneration. The spinal ganglia are bathed in pus and are in a condition of marked hypertrophy, with atrophy of the cells and advanced fatty degeneration (E. Wagner). Finally, the cord itself, in caries of the vertebræ, is attacked by the inflammatory process.

That simple compression of the cord, without spinal lesions, may produce paralysis, is proven by the prompt recovery of certain sudden paraplegias, after straightening the vertebral column (observation by Brown-Séquard). E. Rollett also reports a case, in which dislocation of the axis posteriorly had produced paralysis of all the limbs, and in which reduction of the vertebral column forwards was followed by its gradual disappearance. On the other hand, we know that considerable incurvations of the

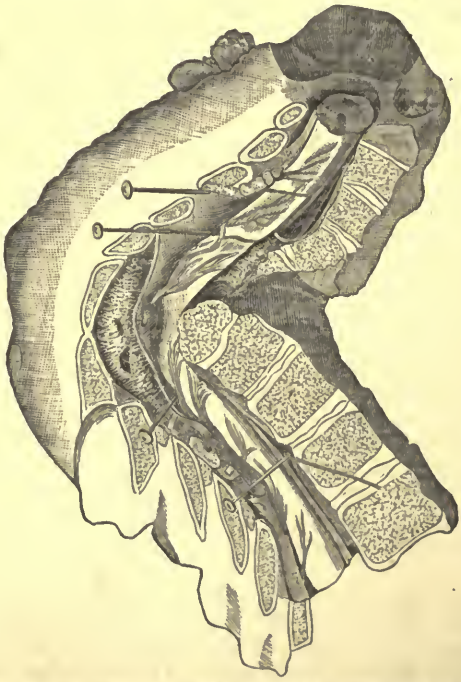

FIG. 12.—Caries of the middle dorsal vertebræ with kyphosis presenting an acute angle. The cord is markedly compressed at the point of curvature. A collection of pus is situated between the dura mater and the vertebral arches of the diseased vertebræ.

vertebral column may exist without lesion of the cord. In Berend's case (extreme scoliosis from caries of the vertebræ, without paralytic symptoms), Virchow found simple atrophy of the cord.

But, in most cases, the compression soon produces inflammatory changes in the parenchyma of the cord. In a case cited by Michaud (l. c., Obs. I.), a child had been affected with spinal curvature, for a short time, without real paralysis. The cord, which appeared to be normal, presented, under the microscope, a fibrillary network, containing nuclei, scattered be-

tween the nerve fibres; these lesions were diffused throughout the cord. By the final development of myelitic changes, foci of softening develop around the compressed portions and serve as points of departure for the secondary degeneration (Tuerck, Charcot, Bouchard, etc.), which has been already referred to under the general characteristics of spinal diseases, and which develops upwards in the postero-internal columns, and, downwards, in the postero-lateral columns.

The sclerosis is sometimes more pronounced upon one side than the other. When the pressure is exercised at the level of the upper dorsal vertebræ, the sclerosis sometimes, though rarely, extends to the cervical region and produces diplegia brachialis, as we have explained above; or ascending lateral sclerosis may develop above the lesion, without posterior degeneration (Michaud); or the sclerosis may also extend outwards to the posterior root fibres which are adjacent to the posterior horns. According to Charcot and Pierret, the degeneration of the latter fibres produces disorders of co-ordination. The gray columns, enclosed within these degenerations of the cord, do not remain intact, and the gray horns undergo serious changes (deformity and partial atrophy, Michaud).

From an etiological point of view, we may state that large cities, both in private and hospital practice, present numerous cases of inflammatory affections and deformities of the vertebral column, combined with general scrofulosis, tuberculosis, and rachitis. Spinal curvature and its sequences are frequently observed in Vienna, especially among the working classes. Among the better classes, children predisposed to scrofula are the objects of more care and attention, and vertebral diseases, whether spontaneous or traumatic, are better observed and more readily treated. On the other hand, the poorer classes are unable to give their sickly, scrofulous children the necessary care and attention. The children have barely passed the twelfth year, when they are apprenticed, often without any discretion on the part of the parents or guardians, and labor is demanded of them disproportionate to their incomplete physical development. The germs of scrofula of the bones readily develop in those trades in which the children are compelled to assume a sitting or bent position, and to carry heavy loads. Under the influence of unfavorable conditions, the osseus inflammation, which was at first latent, increases more and more, and the vertebral column, which is of very delicate structure, finally becomes curved.

The symptoms of vertebral caries vary according to the height of the primary disease and according to the disorders to which it gives rise. The clearest and simplest manner of understanding these symptoms is to discuss separately the diseases of the upper, middle, and lower segments of the vertebral column. The differentiation and more careful study of these varieties possess no less importance for diagnosis than for prognosis and treatment.

Caries of the Cervical Vertebræ.—This form, which is the most dangerous to life, is manifested, at first, by occipital and cervical neuralgias, which are generally regarded as rheumatic. Movements of the head are retarded, and the pains increase on pressure or movement of the upper cervical vertebræ. These symptoms are followed by complete stiffness of the neck, and sometimes by torticollis. When the head is straightened and, in all changes of position, the patient supports the occiput with the hand (Rust, Abhandlungen, I. Bd., p. 176 et seq). According to Leyden (Klin. d. Rueckenmarkskr., Bd I., p. 249), we must regard the affection as confined to the articulation of the odontoid process with the atlas, when

rotatory movements of the head are obstructed and the power of flexion is preserved.

At a more advanced stage the head inclines laterally, deglutition and respiration are embarrassed, speech is abolished, and partial spasms and subsultus occur, with paralysis, chiefly affecting the upper limbs. Sometimes, towards the end of the disease, the rotatory movements of the head are accompanied by creaking, which is appreciable to the hand and ear. The patient succumbs gradually to marasmus or dies suddenly from fracture of the odontoid process, from hæmorrhage from the ulcerated vertebral arteries, or from the extravasation of pus into the thoracic cavity. Purulent collections, originating in the cervical vertebræ, sometimes travel along the lateral portion of the neck, and are directed upwards towards the clavicle and more rarely towards the axilla. The pus more frequently reaches the posterior wall of the pharnyx, and gives rise to a retro-pharyngeal abscess, attended with dysphagia, embarrassment of speech, and dyspnœa. An exploration of the cervical region will then reveal the presence of a purulent collection.

In two of my cases (Prakt. Heilk., 50, 1866), caries of the odontoid process of the axis occurred. Inflammation of the second cervical vertebræ, followed by caries of the odontoid process, is a very rare event. The symptomatology offers in these cases, upon careful examination, several data for diagnosis.

Of the two cases belonging to this category, the first was characterized by caries of the odontoid process of the axis, destruction of its ligaments, and projection of the axis into the spinal canal, the cord being compressed and softened. In the second case, the autopsy revealed caries of the odontoid process and of the lumbar vertebræ, a very large abscess in the left iliac fossa and another smaller one behind Poupart's ligament.

In these two cases the disease began with formication and neuralgic pains in the neck and occiput, the head being turned to one side, and the pains increasing in severity when the attempt was made to correct this vicious position. In one of these patients deglutition was embarrassed, the voice nasal, the pupils strongly contracted, and the iris immovable. In both cases the formication and anæsthesia were soon followed by motor paralysis in the limbs. In the first case, the tingling was the precursor of the paralysis, and symptoms of motor irritation had also occurred. Temporary diminutions of the compression gave rise to variations in the paralysis. Signs of paralysis of the diaphragm occurred in both, with hurried, interrupted, and incomplete costal respiration, with dyspnœa and asthma, but without contractions of the diaphragm. Considerable acceleration of the pulse and retention of urine occurred in the beginning. Disorders of consciousness were absent in both cases. Finally, multiple tuberculization or other carious processes were present. In Leyden's observation (l. c., p. 251–256), the compression of the cord was at first more marked on one side, and the reflex movements were propagated from one side to the other (by the action of the medulla oblongata, according to Pflueger's law).

In caries of the cervical column, compression of the nerve roots, which constitute the brachial plexus, may occur during their passage through the dura mater or intervertebral foramina; neuritis of the brachial plexus is thus produced, and may terminate, like progressive muscular atrophy, in marked atrophy of the muscles of the arm, with abolition of electrical excitability.

In a case which I published some years ago (Oestr. prakt. Heilk., 48, 1866), a man, twenty-eight years old, was seized, after a blow upon the head, with cutting pains in the arm and shoulder of the right side. At the end of a year and a half, paralysis of the arm occurred, with deviation of the head and flattening and lowering of the shoulder. The arm and forearm were flaccid, immobile, and markedly atrophied, and the thenar and hypothenar eminences were equally atrophied, with abolition of the elec-

trical contractility of the muscles. The right leg also became weaker, and deglutition was embarrassed. An anterior curvature of the cervical column could be distinctly seen and felt upon the posterior wall of the pharynx. The posterior cervical region presented a marked concavity, and was painful on pressure.

In caries of the lower cervical vertebræ we often observe mydriasis, and, more rarely, myosis. In the cases of caries and tuberculosis of the first vertebræ, published by Leudet, Eulenburg, and Schüchard, unilateral pupillary dilatation was observed. The same phenomenon was also noticed in two of my patients (l. c.), one of whom suffered from caries with displacement inwards of the fifth cervical vertebræ, and the other from cancerous degeneration of the bodies of the vertebræ, from the last cervical to the third dorsal. In these latter cases the application of a small square of Allen's Calabar gelatine to the conjunctiva, or the introduction of Calabar glycerine, produced contraction of the pupils, which were reduced to the size of a pin's head. But this action was only temporary, as we also observe in other spinal paralyses.

Caries of the Dorsal Spine.—Caries of the dorsal vertebræ is the most frequent form of the disease, both in children and adults. In the latent and less severe forms, the cervico-brachial or intercostal neuralgias which are present, may at first put us on the wrong scent with regard to the real situation of the disease. We then find the dorsal vertebræ sensitive to pressure and electrical exploration. The functional disorders also become more and more marked. The gait is stiff and uncertain, fatigue is soon experienced, and the pains increase in severity in any occupation which necessitates flexion of the trunk. Deviation of the dorsal column also soon occurs, and we are better able to observe the curvature and subluxation (angular projection of Bampfield) caused by absorption of the bony tissue and the intervertebral cartilages.

As the spinal lesion develops, the inflammatory irritation or compression will act upon the sensory roots and upon the prolongations which they send into the gray horns. This produces pain and reflex movements; but if the irritation predominates in the anterior roots and their prolongations into the substance of the cord, intermittent or continuous spasms will result. The pains and spasms usually precede the abolition of motion and sensation. It sometimes occurs that, in the midst of extended paralyses, certain circumscribed parts retain their sensibility, especially when the lesion of the cord leaves a part of the nerve roots and their prolongations into the gray substance, intact. According to the severity of the medullary lesions and to the intensity of the compression, and according as the latter is caused by degeneration of the vertebræ, by an exudation or by a new-growth, the tonic spasms will result in movements of extension or flexion. Moderate compression results especially in contracture of the flexors of the thigh and leg, while a more severe compression, acting upon a larger segment of the cord, will chiefly produce tonic contraction in the extensors of the forearm and hand, of the leg and foot. Schiff has seen flexion of the limbs followed by extension develop in his experiments upon animals, by producing, at first, weak, and then stronger and stronger irritation of the spinal cord. These results agree with my own observations upon vertebral caries (l. c., No. 51). When the caries involves the cervico-dorsal region of the vertebral column, the cervical enlargement is affected, as we can determine from the neuritis and paralyses of the arms, the pupillary disturbances, etc. If the lesion is situated lower, the chief symptom is furnished by the paraplegia from compression, together with

all the signs which we have previously described. Partial disorders of sensation sometimes occur in these cases. The following observation, with the microscopical examination, furnishes an instructive example.

A peasant woman, forty-four years old, entered Dr. Scholz's service. For two years past she had suffered from weakness of the legs, with intermittent pains in the legs and back. Examination showed angular curvature of the vertebral column from the sixth to the ninth dorsal vertebræ. There is considerable weakness of voluntary motion in the lower limbs, and the patient can only walk a few paces and with great effort. The electro-muscular contractility is normal, the electro-muscular and electro-cutaneous sensibility is abolished. These facts induced me to examine sensation more carefully. I then found that tactile sensibility is everywhere preserved, but the sensibility to pain (pricking, pinching, electric brush) and to temperature (a piece of ice gives a sensation of contact or weight) is abolished over a large surface. This analgesia occupies the anterior surface of both lower limbs, extends upwards to the fifth rib, and passes backwards over the thighs as far as the third lumbar vertebræ. Sensibility is preserved in the lower portion of the gluteal region, in the anus, perineum, mons veneris, and labia majora, but at the end of about a month these regions became equally analgesic. At a later period, the insensibility extended from above downwards to the posterior surface of the thighs, but the upper limit of the insensibility in the thoracic region remained the same. The upper limbs were not affected, either as regards motion or sensation. At the end of three months the patient was unable to leave her bed; periodical spasms in the paralyzed lower limbs and paralysis of the sphincters occurred. The patient died in the fourth month.

At the autopsy, a serous infiltration was found in the brain, and the cord was compressed and softened between the sixth and eighth pairs of dorsal nerves. Microscopical examination of sections of the cord showed that the nerve cells of the gray substance were opaque, shining, opaline, deprived of their prolongations, and sclerosed. Meynert made a very careful examination, and found that the trabeculæ of the reticulum, in the medullary tissue surrounding the gray substance, and especially around the posterior horns, and in the anterior portion of the posterior column, were thickened and transformed into large granular fibroid bands. Around many of the axis cylinders the section of the cord appears swollen, and of a dull, grayish-pearl color, and the axis cylinders have disappeared in many places. The appearances, therefore, were those of a foyer of myelitis, above and below which, from the cervical cord to the conus medullaris, the gray horns contained a large number of sclerosed nerve cells, especially below the foyer in the dorsal region. Some of the nerve cells, in this situation, were distinctly opalescent, with dark borders, containing large nuclei, and in which the sclerosis had been preceded by fatty or pigmentary degeneration. In the cervical enlargement, the posterior horn contained numerous masses of small opaque nuclei, which are undoubtedly derived in part from fission of the fusiform elements of the gelatinous substance. Diffuse swelling of the reticulum, with large granular fibroid bands, was observed along the whole length of the cord.

Caries of the Lower Dorsal and Upper Lumbar Vertebræ.—This form is of less frequent occurrence and more difficult to recognize. Pain on pressure over the vertebræ is a deceptive symptom, as Behrend had shown several years ago. The curvature of the spine is very slight, and appears very late, even in severe affections of the lumbar column. The large surfaces by which the lumbar vertebræ are in contact, and the slight flexibility of this segment of the spine, cause ulcerations and curvatures of this region to run a slow course. Percussion of the diseased vertebræ, and exploration with a sponge, according to Copland's method, furnish few important diagnostic signs, even in old cases.

Several years ago, Behrend called attention, from a diagnostic point of view, to the stiff, peculiar position, to the tottering, uncertain gait of these patients, and to the apparent shortening of the corresponding lower limb, with preservation of the rotatory movements of the thigh. Behrend has also noticed that pain is produced by flexion of the trunk and, even more, by concussion. At a later period, Adams (Lancet, May 13th, 1865) laid stress upon three principal symptoms, which may furnish important data

for early diagnosis of caries of the lumbar vertebræ, and which depend upon the relations of the psoas muscle to the lower segment of the vertebral column. These symptoms are as follows: 1. When the patient passes from the horizontal to a sitting or erect position, he does so with great caution, helping himself with his arms and hands; 2, when he wishes to pass from the abdominal to dorsal decubitus, or vicè-versa, the patient endeavors to effect the rotation of the pelvis very carefully by crossing the thighs and supporting himself upon the elbows; 3, in walking up a flight of stairs, the patient tries to place both legs very quickly upon each step, in order to avoid as much as possible, any sudden movement of the pelvis.

Shortly afterwards, I published a series of observations which have aided in clearing up the symptoms of vertebral diseases and the nervous disorders consequent thereon (Wien. med. Presse, 42–45, 1865, and Zeitschr. f. prakt. Heilk., 46–51, 1866). These observations prove that the electrical current constitutes a good means of exploration, when the situation of the lesion is doubtful, in order to determine the secondary irritation in the ramifications of the sensory roots. If the electrodes of a galvanic battery are placed in close proximity to one another over the transverse processes of the vertebræ, no especial sensitiveness will be noted in the healthy portions, but, over the situation of the vertebral lesion, the burning or pricking caused by the negative pole becomes so much the more insupportable, the nearer the electrode is placed to the real seat of the disease. The sensibility sometimes passes beyond the ribs and radiates forwards into the thoracic region, while a current of the same intensity is feebly felt upon the corresponding portion of the opposite side, and in the upper and lower portions of the same side. We may also mention other characteristic symptoms of the latent forms of caries of the lumbar vertebræ. From the beginning of the disease, a prolonged sitting position causes pain, though the gait is still normal. Spasms and neuralgic pains appear upon the anterior and posterior parts of the lower limbs, as a peripheral expression of the spinal irritation. The weakness of the legs then progresses, the patient soon becomes tired, and the rachialgia increases. The patient assumes an oblique, lateral, and markedly stiff position. The lower part of the vertebral column begins to be immovable, and even passive rotatory movements of the pelvis are painful. From the aggregate of these pathognomonic signs, we are justified in diagnosing an affection of the lower part of the vertebral column, consisting of a latent chronic inflammation of the vertebræ and often terminating in caries.

The abscesses of the lower segment of the vertebral column descend along the psoas muscle and Poupart's ligament into the thigh, and, in rare cases, they open into a loop of the intestines, into the bladder or vagina. Abscesses originating in the dorsal region, unless they discharge upon the back, open into the thoracic or abdominal cavity, into the trachea or œsophagus (observation of Lambl, with expectoration of debris of carious bone, and subcutaneous emphysema of the dorsal region); they are rarely converted into psoas abscesses.

The pains in the crural or sciatic nerves, which develop in caries of the lower segment of the vertebral column, are often combined with painful tenesmus of the rectum and bladder, and with a sensation of weight in the abdomen or perineum. The paraplegia then becomes more and more complete; the diminution of sensibility is slower and less complete; reflex actions disappear quickly, especially when the lesion is situated in the lower portions. The deplorable complication of paralysis of the sphincters is almost always superadded in cases of this kind.

The early diagnosis of vertebral caries sometimes meets with great difficulties.

The violent dorsal pains, the curvature of the vertebral column, the paralyses from compression and contractures, also belong to various other affections. Intense rachialgia and extreme sensibility in movements of the spinal column are present in neuroses of the vertebral articulations. (Esmarch). But, in addition to this symptom of spinal irritation, cutaneous hyperæsthesia and excessive tenderness of the vertebræ to slight pressure also occur, while stronger pressure is unnoticed when the attention of the patient is drawn away. Menstrual disorders frequently coexist, together with aggravation of the symptoms from moral causes; the existence of other signs of hysteria clears up the real nature of the rachialgia. In the *rheumatic pains*, which frequently develop under the influence of cold, in people who work in the open air during inclement weather, we observe tenderness of the vertebral column upon pressure, a deviation towards the affected side, marked sensitiveness to pressure in the lumbar muscles and in the longitudinal muscles of the back or neck. Electrical explorations in these cases do not give as significant results as in vertebral caries.

In *cancer of the vertebræ*, deflection of the spinal column and paraplegia also occur, and sometimes, as we shall prove by several examples, we cannot distinguish it from vertebral caries. Nevertheless, in some instances, the paraplegia dolorosa, the arterial thromboses, the evident cachexia, the degeneration of the glands, and the advanced age of the patients will enable us to make a differential diagnosis. The paraplegias from compression and the contractures caused by tumors of the meninges are distinguished from vertebral caries by the absence of deflection, by the rapid development of the symptoms, and by the prompt abolition of the electrical reactions, and often also of reflex excitability. Aneurisms and hydatid tumors, leading to atrophy of the vertebræ from compression, are rarely recognized during life.

Finally, the diagnosis of vertebral caries may be cleared up, not alone by the abnormal curvature, but also by the youthful age of the patient, by the existence of cold abscesses, and of scrofula or tuberculosis. The theory of syphilitic caries is not admissible, unless we have determined the existence of a previous infection and have obtained positive results from specific treatment.

Caries of the vertebræ must be regarded as one of the most serious diseases of youth and middle age. It is true that quite a large number of the patients may be improved and gradually cured. But the number of invalids remains very large, the duration of the disease is long, kyphosis is very frequent, the favorable termination is, for a long time, uncertain, and the, often, diathetic nature of the disease and its complications involve sufficient danger to render the prognosis very grave. In general, caries which is due to the above-mentioned causes runs a more favorable course in the lower than in the upper portions of the vertebral column.

Recovery from paraplegia and contractures in vertebral caries has been affirmed especially by Bouvier and Leudet (Soc. de Biol., T. IV., 1862–'63); it has been more recently confirmed and demonstrated histologically by Charcot (loc. cit.). Michaud has published in detail (loc. cit., Obs. 3) the case of a woman who had recovered for five years, from a paraplegia with contracture, after treatment with repeated moxas, and who finally died of coxalgia. The cord, at the point of compression, was reduced to the thickness of a goose-quill, and was very firm and of a gray color. The

parenchyma was in a condition of secondary degeneration, and the gray substance had been reduced to one of the anterior horns. Under the microscope a considerable number of nerve tubes with their axis cylinders were found in the midst of the sclerosed tissue. In my opinion there was no regeneration of nerve tubes or even of the myeline sheath in this case. The meningitic and myelitic processes had merely retrogressed and had left unaffected the conductibility in the intact portions of the white and gray substance, and this sufficed to maintain motion and sensation. In certain rare cases of spondylarthrocace, a spontaneous retrogression of the paralytic symptoms was observed.

Therapeutics may sometimes aid in bringing about this favorable termination. Cauterization with the hot iron appears to give good results in some cases. After repeated cauterization, the pains, paralyses, and contractures of the limbs may disappear. This treatment appears to produce its best effects in patients who are otherwise in good health and whose muscles are well preserved. But a considerable number of cases withstand all treatment. Mechanical and orthopedic treatment is of the greatest importance when begun at an early period. The patient is confined to bed for several months, in dorsal or abdominal decubitis, the latter being more disagreeable. Then we must begin the methodical application of extension apparatus, such as Volkmann has recently invented, in order to relieve the vertebral articulations, and it is necessary to wear for a long time some apparatus for the fixation of the vertebral column.

Among the internal measures, we may recommend mild ferruginous preparations, cod-liver oil, and small doses of iodide of potassium; among the balneotherapeutic measures may be mentioned iodine-waters, salt-baths, and sea-baths. In the first rank must be placed the invigorating air of the country and a generous diet. The galvanic current is less useful in paralyses from compression than in treating the accompanying symptoms of nervous irritation. On the other hand, the induced current is more useful for the purpose of invigorating the muscles which surround the vertebral column.

B.—Fractures of the Vertebræ.

Fractures of the vertebræ, and the paralyses of motion and sensation dependent thereon, were known to the most ancient physicians of Greece and Rome; they are of frequent occurrence in large surgical services and in hospitals. The large amount of material accumulated in medical literature has placed our knowledge of this disease upon a firm foundation. In the following remarks we shall pay especial attention to the effects of fracture of the vertebræ upon the cord.

Leaving aside incomplete fractures and fissures of the vertebræ, we shall examine strictures of the spinal canal caused by the compression of the vertebræ or the simultaneous displacement of fragments of bone (with or without luxation), and by traumatic lesions of the cord. In violent strains sustained by the vertebral column (fall or bruise), the cord is compressed; in fractures, it is subjected to deep solutions of continuity. Fracture involves either the arches of the vertebræ, as in lesions of the cervical portion, or of the vertebral bodies, as in lesions of the lower dorsal or lumbar vertebræ. The fractured vertebral arches may cause a lesion of the cord by their lateral or anterior displacement; the vertebral bodies, when fractured obliquely or transversely, involve the cord by flexure and displacement of the fragments; in the latter varieties the meninges

may be torn by the splinters of bone. A traumatic hemorrhage of the superficial venous plexuses very frequently occurs between the vertebræ and dura mater, or a hemorrhage from the deeper meshes of the pia mater penetrates into the parenchyma of the cord. We sometimes find, in addition, flexion, flattening, and wounds of the cord, which may be completely crushed. In less severe accidents small collections of blood appear between the fibres, or hæmorrhagic softening develops around the vertebral fracture, and can be followed under the microscope for a certain distance above and below this point. In lesions of long duration myelitic processes develop with the ascending and descending degenerations, which we have described in detail in the preceding chapters. In fractures which are complicated with a gunshot wound opening into the spinal canal, spinal or even cerebral meningitis, wounds, ruptures, and hemorrhages of the cord may ensue.

The symptoms of vertebral fractures present numerous variations, according to the situation and extent of the lesion. Among the initial symptoms we frequently find loss of consciousness (with or without vomiting) from cerebral concussion and shock, caused by acute anæmia of the nerve centres. These severe symptoms may be merely temporary, or they may increase to such an extent as to endanger life. Severe pains develop at the site of the fracture and radiate towards the limbs, attended by temporary muscular spasms. A palpable deformity may exist from the lesion of the vertebral arches or the spinous processes, and from displacement of the fragments, but it may also be absent when no displacement occurs or when the fragments have been pushed forwards.

The functional disorders of spinal origin, in fractures of the upper cervical vertebræ, consist of pain and stiffness in the neck and difficulty in performing rotation of the head, except in the cases of sudden death from fracture of the posterior arch of the atlas, and of the odontoid process and rupture of their ligaments. A sudden movement has been known to cause a fatal shock to the cord. The phrenic nerves and brachial plexus may be affected in fractures of the lower cervical vertebræ. Respiration is then involved, the paralysis predominates in the upper limbs, partial muscular spasms occur, sensibility is dulled, deglutition embarrassed, and the temperature is very high. We can often notice an abnormal arching or concavity of the neck, or a projection in the pharynx. In fractures of the dorsal and lumbar vertebræ, paralysis occurs in the lower limbs, with hyperæsthesia limited to the same region and followed by descending anæsthesia, and, later, by paralysis of the sphincters. The lumbar vertebræ are much more rarely fractured on account of their size and firmness. When fracture in this region does occur we find, in addition to the preceding symptoms, pains in the distribution of the sciatic or crural nerves, abolition of reflex and electrical excitability in the paralyzed and atrophied muscles and in the nerve trunks.

Fractures of the cervical and adjacent dorsal vertebræ may be accompanied by temporary spermatorrhœa followed by persistent erections. I have recently observed, in the Vienna General Hospital, a very interesting case of fracture of the lower cervical vertebræ, with priapism continuing seven days during life and thirty-six hours after death.

A brewer, forty-three years old, fell into a cellar, May 27, 1875, while carrying a keg of beer, and was carried in an unconscious condition to a police station (he was thought to be intoxicated), and, twenty-four hours afterwards, to the surgical service of Prof. Salzer. The patient was then found in the following condition: consciousness had returned, the fourth, fifth, and sixth cervical vertebræ were fractured and

the lower limbs and trunk were paralysed and insensible. There was retention of urine with constipation and priapism, the latter symptom lasting until June 2d; the patient died on this day, consciousness being retained, with symptoms of acute pulmonary œdema.

The autopsy was made thirty-six hours after death. Upon external examination an ecchymosis, about two centimetres long, was found over the lower part of the occipital bone. The penis was erect and only relaxed after incision of the tunica albuginea and corpora cavernosa, and after the venous hæmorrhage resulting therefrom. The meninges and brain were strongly congested; the left lung was œdematous and congested: the upper and lower lobes were retracted, destitute of air, and contained scattered foci of pus. The vesical mucous membrane was of a deep red color, and an ulcer was situated upon its posterior wall. The inter-vertebral cartilage between the fourth and fifth cervical vertebræ was torn by the body of the fourth vertebra, and the adjoining one was torn by the body of the sixth. The arches of the fourth and sixth cervical vertebræ were fractured in a transverse direction. The cord, in the upper half of the cervical enlargement, was crushed and converted into a pulpy mass, streaked with blood.

We must regard the priapism, which lasted a week during life, as due to traumatic irritation of the centre of erection, situated in the cervical and adjacent portion of the dorsal regions. But we are unable to determine whether it is to be attributed to increase of the afflux of blood from paralysis of the vaso-motor nerves, or, as Goltz has lately suggested (Pflueger's Arch., 9. Bd., 1874), to a functional irritation of the vaso-dilator nerves. The post-mortem priapism may be explained by paralysis of the vascular muscles, with venous distention of the corpora cavernosa and glans.

The *diagnosis* of vertebral fractures is based upon the above-mentioned symptoms, upon the traumatic origin of the disease, and upon the appearance of the deformity; if crepitation can be felt the diagnosis is rendered positive. In the majority of cases the fracture is complicated with dislocation, and the entire problem consists in ascertaining to what extent the cord is involved. The absence of displacement greatly increases the difficulty of diagnosis, and the character of the affection can only be determined by examining the mobility of the vertebral column and the attendant phenomena. Independently of the direction of the lesion, the compression of the spinous processes, when exercised upon the posterior part, will especially cause disturbances of sensibility, and the projection forwards, with compression of the anterior part, will cause motor disorders. These data are of importance in deciding upon reduction or trephining.

The *prognosis* of fractures of the vertebræ should always be very guarded. Even when the spinal lesion appears to be slight, we must bear in mind that fractures of the vertebræ unite with difficulty, that the formation of callus is incomplete, and that final displacements, suppuration and caries are frequent, all of which circumstances influence the course of the disease. In fractures of the upper cervical vertebræ the compression of the medulla oblongata or phrenic nerves may produce paralysis of the respiratory centres, severe pulmonary hæmorrhages, and paralysis of the diaphragm. In wounds of the lower portion of the cord life is less threatened, but a fatal termination, after a long illness, is the rule.

Recoveries are of exceptional occurrence. According to Gurlt (Handb. d. Lehre v. d. Knochenbruechen, II. Bd., 1864), fifty-four recoveries occurred in two hundred and seventy cases, but a large number were not permanent. Reduction of the fragments may be attempted by powerful extension and counter-extension. This measure is rarely successful, except, perhaps, in fractures of the lumbar vertebræ, and we can never obtain

retrocession of the lesions already existing in the cord. The patient must then be placed in a proper position and kept immovable. The lesions of the cord and their sequences demand symptomatic treatment such as we have referred to above; especial attention must be paid to the condition of the bladder.

According to Ollivier, trepanation of the vertebræ was practised for the first time by Cline in 1814, but the patient died shortly afterwards. In a similar case published by Tyrrel the patient partially recovered motion and sensation after the operation, but died of pleuro-pneumonia in the second week. The first successful result from trephining the vertebral column was obtained by Gordon (Lancet, Dec., 1865). The operation was performed in fifty minutes without noteworthy hæmorrhage, and was followed, at the end of several days, by a return of sensibility and of the action of the sphincters. Eight weeks later the patient was able to drive out in a carriage in a sitting posture. Walking and standing could not be performed at that time, but the final result is unknown. Of twenty-one cases collected by Gurlt seventeen terminated in death; four patients survived, but their recovery was incomplete.

C.—Dislocations of the Vertebræ.

With the exception of the upper cervical vertebræ, the others, on account of their structure and the strength of their ligamentous attachments, are more exposed to fracture than to dislocation. The latter are divided into traumatic dislocations, produced suddenly by external violence, and into spontaneous dislocations, gradually brought about by internal morbid changes. Traumatic luxations, caused by excessive strain, are usually complete and seriously compromise the cord, on account of their manner of origin. Spontaneous, slow, and incomplete luxations differ in these respects. The luxation may be unilateral or bilateral. The traumatic dislocations, especially in the lower segments of the vertebral column, are often complicated by coexisting fractures, but sometimes simple luxations are observed.

According to Malgaigne (l. c.) and Blasius (Prager Vjschr., 103, 104, Bd., 1869) the most frequent luxations, as well as incomplete subluxations, are those of the lower cervical vertebræ. The causes consist of blows upon the head or neck, external violence, and energetic muscular contractions during the performance of sudden rotatory movements. Martin's experiments (Zeitschr. d. Wien. Ges. d. Aerzte, 18–23, 1864) have thrown light upon the amount of force necessary to produce these results. Slight displacement of one vertebra upon another is only obtained after complete rupture of the ligaments and partial tearing of the intervertebral cartilages. Maisonneuve and Bouvier have obtained analogous results. Luxations occur most frequently between the first and second cervical vertebræ, on account of the great mobility of this articulation. In consequence of the rupture of the ligaments which fix the odontoid process, the head falls forward, and the corresponding spinous process forms a marked projection in the neck. Death soon occurs from compression of the medulla oblongata. In incomplete dislocation of one articular process, the resulting paralysis of motion and sensation may disappear if reduction is promptly performed (observations by Schuh, Maissonneuve, etc.). Simple luxation of the odontoid process, with projection of this appendage through the

transverse ligament of the atlas and fatal compression of the medulla oblongata, may result from a violent blow upon the head or neck.

In luxation of the lower cervical vertebræ we observe a lateral deviation of the spinous processes, sometimes also of the transverse processes, sinking in of the neck, and rotation of the head to the opposite side. In Daucé's case (Gaz. des Hôpit., 91, 1867) double mydriasis occurred. In incomplete subluxations of the lower cervical vertebræ, violent pains develop in the neck, according to Martin (l. c.), and increase when an attempt is made to straighten the head. Projection and tension of the muscles of the neck occur, extending from the occiput to the dorsal region, and a corresponding depression develops upon the opposite side. In addition, temporary paralyses and subsultus develop in the extremities.

Treatment consists, above all, in the reduction of the dislocated vertebræ, but this must be done very cautiously. In recent cases we not infrequently obtain prompt and permanent results thereby. Various kinds of apparatus are employed to maintain the replaced vertebræ in position. In a large number of cases we are unable to perform reduction, and intense inflammation, suppuration, and abscesses result.

D.—Spondylitis Deformans and other Deformities of the Vertebræ.

The osseous growths and anchyloses of the vertebræ in old age did not escape the attention of the older observers. Virchow (Geschichte der Arthrit. deform., Arch., 1869) has found swellings and osseous proliferation upon the cartilages of the cervical and lumbar vertebræ, with considerable narrowing of the spinal canal, in fossil human bones exhumed in a convent. According to Rokitansky (Anat. Path.), atrophy and suppuration of the intervertebral cartilages, or bony spiculæ uniting the articular surfaces, may lead to synostosis of the bodies of the vertebræ and of the lateral articulations. Ossification of the ligaments (Gurlt) and bony tumors occupying the posterior arches or the intervertebral spaces may also cause anchylosis of the vertebræ.

These osteoid products in the spinal articulations may result from inflammatory processes of an exceedingly slow course, and are especially met with in old age, in which ossifications and calcifications are also frequent in various other organs. These products only develop exceptionally in young people. According to the observations which are, at present, at our command, there is no reason for attributing spondylitis deformans to arthritis or to constitutional or diathetic affections.

The principal phenomena in deforming vertebral inflammations of a chronic course (also called vertebral gout), consist of a difficulty in the movements and stiffness in the corresponding vertebral articulations, combined with peripheral pains. The loss of motion is most marked in the cervical column. Thickenings and nodosities are sometimes observed in certain parts of the neck, if the cervical vertebræ are affected, or in the abdominal region when the lumbar vertebræ are involved. At times well-marked creaking is observed in rotatory movements of the neck (Haygarth). The deformities of the vertebral column following spondylitis deformans may sometimes exercise compression upon the cord on account of kyphosis and narrowing of the spinal canal.

Thus, Rotter has recently published (Arch. f. klin. Med., XIII. Bd., 1874) a case of arthritis deformans involving the articulation of the atlas with the axis and both elbows, with slight deformity of the cervical spine. Clonic spasms occurred during life, followed by right hemiparesis and stuttering. The autopsy revealed degeneration with fatty granulations, especially in the right posterior column of the cord, and less marked in the left. The cerebral cortex presented minute hæmorrhages; the central ganglia were normal.

More profound and advanced lesions of the centres are rarely found in the chronic vertebral inflammations at present under discussion. We more frequently observe irritation of the spinal nerves from thickening and narrowing of the intervertebral foramina. The symptoms usually consist of cervico-brachial or cervico-occipital neuralgias, combined with stiffness of the neck, and a sensation of heaviness or weakness in one of the limbs. We are not infrequently able to detect creaking during excursive movements of the head. In certain cases neuritis of the brachial nerves may develop and gradually lead to muscular atrophy.

I have observed thickenings and nodosities of the vertebræ, with pain on pressure and concomitant paresis, in the vertebral periostitis of young scrofulous subjects. In these cases the character of the vertebral affection is rendered evident by the flat thickenings and nodosities which adhere, for the most part, to the sides of the vertebræ, by the coexistence of other periosteal inflammations, or by the traces of previous diathetic diseases of a similar nature.

I have published (Zeitschr. f. prakt. Heilk., 48, 1866) a case of cervical periostitis which belongs to this category. A circumscribed, rugged osseous tumefaction was found, extending on the outside for about two fingers' breadth, from the transverse process of the second to that of the fifth cervical vertebra, and extending upwards as far as the mastoid process and occiput. The movements of the upper and lower limbs were performed with difficulty, but considerable improvement followed the employment of cod-liver oil, iodine preparations, and warm baths.

The *treatment* of spondylitis deformans must be directed towards producing absorption of the neoplasms, and relieving the consequent nervous irritation. For these purposes we employ preparations of iodine, iodated baths, mud baths, and the indifferent mineral waters. Moderate continuous currents may be applied to the irritated nerve trunks and to the brachial plexus. Faradization is useful in cases of muscular atrophy.

In addition to spondylitis deformans, we may refer to certain rarer forms of vertebral deformities, which cause compression of the cord from narrowing of the spinal canal. In the first rank we may mention the vertebral hyperplasiæ, such as bony tumors of the occiput and of the atlas and axis, which produce narrowing of the upper portion of the spinal canal. The narrowing of the foramen magnum from hypertrophy of the basilar process of the occipital bone, or of the posterior arches of the first two cervical vertebræ, has been especially studied by Solbrig (Allg. Zeitschr. f. Psychiatr., 24. Bd., 1867). He has observed the development of atrophy of the medulla oblongata, and the appearance of epileptic attacks (in nine cases).

The articulation of the occipital bone with the atlas may become anchylosed in consequence of inflammation, caries, and tumors of the squamous portion of the occipital bone (Friedlowsky); this anchylosis may also be congenital. Ollivier (l. c.) mentions a case of projection of the odontoid process into the foramen magnum, and atrophy of the olivary bodies and pyramids (with a sudden attack of paraplegia). Osteoid growths

from the odontoid process have been observed by Froriep (in two cases, with fatal choreiform attacks), and very recently by Herz (Arch. f. klin. Med., XIII. Bd., 1874). In the latter case the patient suffered from paralysis and contractures in the limbs and in the muscles of the neck. The autopsy showed the existence of hypertrophy and displacement of the odontoid process, a focus of softening at the decussation of the pyramids, and secondary changes in the antero-lateral columns.

Exostoses of the vertebral arches or bodies may also cause considerable narrowing of the spinal canal. Thus, Eberth (Corresp.-Bl. d. Schweiz. Aerzte, 1872) has seen, in a man ninety-three years old, who suffered from paralysis of all the nerves situated below the seventh dorsal vertebra, an osteoma growing from the seventh and ninth dorsal vertebræ, which had produced gray degeneration of the columns of Goll, from the floor of the fourth ventricle to the portion of the cord corresponding to the lesion. In a case cited by Brown-Séquard (Phys. Anat. and Path. Researches, 1848) the patient suffered from pains in the loins and arms, with a sensation of stiffness and heaviness; the head was turned to the right. A conical exostosis was found growing from the posterior part of the base of the odontoid process and compressing the cord.

More circumscribed vertebral exostoses have been found in rachitis and syphilis (Portal). But they are sometimes found in cases in which the existence of a diathesis cannot be demonstrated, and they are due, especially in old age, to the general tendency towards processes of ossification which manifests itself at this period. In rare cases congenital osseous proliferations also occur within the articulations.

The majority of the vertebral deformities which we have mentioned cannot be diagnosed, and are inaccessible to treatment. We may employ the measures, which we have referred to above, in order to combat the irritation of the nerves and the symptoms of compression of the cord.

E.—Atrophy of the Vertebræ from Compression.

(*In consequence of aneurisms and hydatid cysts*).

Under the influence of long and progressive compression, the vertebræ corrode, like other bones, their volume diminishes more and more, and the vertebral canal is finally opened, usually at the point in which the arch joins the body of the vertebræ. An inflammatory reaction occurs at the same time, with production of osteophytes and thickening of the periosteum, causing partial obliteration of the vertebral openings. Atrophy of the vertebræ is rarely produced by tumors of the mediastinum, but much more frequently by aneurisms and hydatid cysts.

a. *Compression and Atrophy of the Vertebræ from Aortic Aneurisms.*

Aneurisms of the thoracic or abdominal aorta may cause atrophy, either of the dorsal or lumbar vertebræ, especially upon the left side. The compact superficial layers and the spongy tissue of the vertebræ are gradually absorbed, depressions are formed, and fissures penetrate even into the spinal canal. The intervertebral discs, which are only slightly affected by the compression, project between the débris of the vertebræ which still remain. The progressive destruction which involves the bones

may even extend to the walls of the aorta, whose external coat forms adhesions with the thickened and inflamed surrounding tissues. When the spinal canal is opened it is usually found that a direct implication of the cord has been prevented by the interposition of pseudo-membranous products.

The compression and atrophy of the vertebræ by aneurismal sacs produce symptoms of irritation and compression of the cord, severe pains over the sacrum, a peculiar stiffness in walking, retarded motion of the vertebral column, neuralgic pains in the loins and thighs, and paralysis of both lower limbs. These symptoms may be mistaken for those of caries of the lower segment of the vertebral column. Sometimes, in consequence of rupture of the aneurismal sac, a sudden hæmorrhage may occur into the spinal canal, and terminate quickly in death.

Cases of corrosion of the vertebræ from the pressure of aneurisms have been published by Marshall-Hall, Froriep, and Ollivier. In an example mentioned by the latter author, an aneurism of the thoracic aorta had opened into the spinal canal and left pleural sac, causing sudden paraplegia, quickly followed by death. I will here give the particulars of an interesting case which I observed in 1872, in Dr. Scholz's service, and in which the diagnosis of corrosion of the lumbar vertebræ by an aneurism had been made several days before death.

A man, fifty years of age, states that he has suffered for nine months from pains in the left lumbar region and loins, radiating towards the thigh. The lumbar and sciatic nerves were extremely sensitive to pressure and to the electrical current. Two weeks later the patient was unable to mount the stairs. When he walked in his room he maintained a peculiar position; the lumbar region was stiff and deflected. In the fifth week paralysis occurred in the extensors of the left thigh, the patient was unable to leave the bed, and presented an anæmic appearance. At the beginning of the third month a rhythmical pulsation was noticed upon the left side, beginning, anteriorly, above the crest of the ilium and extending posteriorly to the loins. Auscultation, although practised several times, gave negative results. A diagnosis had at first been made of caries of the vertebræ, but was then changed to that of aneurism. The skin, in the region mentioned above, became distended and reddened; three days later the patient suddenly died. At the autopsy a ruptured aneurism of the abdominal aorta was found, with very abundant hæmorrhage and corrosion of the upper lumbar vertebræ.

b. *Compression and Atrophy of the Vertebræ from Hydatid Cysts.*

The cysticercus (in the cervical region, Rokitansky) and, more frequently, the echinococcus, are the only animal parasites that appear in the spinal canal. The acephalocysts are situated between the vertebræ and dura mater, rarely in the subarachnoid cavity. Hitherto the parasites have not been seen in the cord itself. In Morgagni's case (De sed. et caus. morb., nova edit., Lutetiæ, 1822) a tumor was found adherent to the bodies of the second and third lumbar vertebræ, with compression of the left kidney, diaphragm, and adjacent muscular tissues; the intervertebral foramina were enlarged to the size of a thumb, and hydatid masses were found in the spinal canal, around the meninges, and compressing the lumbar nerves. Reydellet's patient (Dict. des sc. méd., T. XXXIII., p. 564) had had anæsthesia of the lower limbs, and then paraplegia and sharp pains in the right thigh. A fluctuating tumor was felt in the lumbar region, an incision into which gave exit to a considerable quantity of hydatids, leaving the spinal canal exposed to the naked eye. The patient

died a year later. The epileptiform attacks observed by Esquirol (Bulletin de la Faculté de Médecine de Paris, T. V., p. 426) must be regarded as a complication. In Foerster's case (Handb. d. spec. path. Anat., 2. Aufl.) inflammation and suppuration had occurred around the cyst between the spinal meninges, and also between the dorsal muscles. In another case the vesicles of echinococci spontaneously opened externally and communicated, at the same time, with the medullary canal.

Davaine (Traité des entozoaires et des maladies vermineuses, Paris, 1869) cites ten cases of echinococci of the spinal canal. To these may be added one by Foerster, another one which I have published (Zeitschr. f. prakt. Heilk., 51, 1866), and finally a thirteenth case reported by Bartels (D. Arch. f. klin. Med., V. Bd., 1868). In only two cases (those of Esquirol and Bartels) were the echinococci situated beneath the spinal arachnoid. The acephalocysts have been especially observed in women between the ages of twenty-two and fifty-six years; my own case was that of a boy aged fifteen years. All the observations were characterized by symptoms of irritation and compression of the cord.

The case which I observed is interesting on account of the anæsthesia, which was confined to Voigt's lines, and also on account of the extremely rare results of the autopsy. An analogous observation was made by Cruveilhier (Anat. pathol., Liv. XXXV., Pl. 6).

An apprentice, fifteen years of age, began to suffer, without known cause, from pains and weakness in both lower limbs. At the end of three months he was unable to leave the bed; spasms of the flexors occurred, followed by spasms of the extensors, of longer duration. At a later period this condition of the extensors disappeared, leaving only spasm of the flexors, of short duration, and subsultus tendinum, the latter symptom being accompanied by formication in the lower limbs. After these symptoms of irritation had disappeared the lower limbs were left in a condition of complete paralysis. Upon examining the patient I found complete anæsthesia (to vigorous pinching, ice, and heat) throughout the whole extent of the lower limbs, extending upwards to a line passing through both mammæ and through the middle portion of the scapulæ. About three fingers' breadth above the limits of the anæsthesia, the normal sensibility gradually returned. Incontinence of urine and fæces, and bed-sores afterwards developed, and the patient died with symptoms of pyæmia.

Autopsy: Brain anæmic; in the right pleura six hundred grammes of an opaque, sanious fluid; the right lung was compressed, pushed upwards, and adherent to a saeculated distention of the costal pleura. This tumor, starting from the lateral portion of the bodies of the third and fifth dorsal vertebræ, appeared in the pleural cavity as a prominence as large as a hen's egg.

The cyst is fluctuating, filled with numerous vesicles of ecchinococci, and has compressed and corroded the bodies of the third and fifth dorsal vertebræ, which are rough to the feel; it has also compressed and driven back the cord upon the right side of the spinal canal. The latter, between the points at which the third and fifth dorsal pairs of nerves emerge from the dura mater, is reduced to an exceedingly thin layer.

F.—Cancer of the Vertebral Column.

Carcinoma is much more infrequent than caries of the vertebræ. Independently of the older works of Abercrombie, Cooper, etc., the characteristics and relations of cancer of the vertebræ have been studied by Cruveilhier (l. c.), Hawkins (Med. Chir. Trans., 1841), Gull (Guy's Hosp. Rep., 1854), Leyden (Charité-Annalen, 1863), Charcot (Bulletin de la Soc. des Hôpit. de Paris, 1865), and Tripier (Du cancer de la colonne vertébrale, etc., 1866).

The most frequent form is the cancer of the bodies of the vertebræ, which are sometimes strewn with cancerous nodes as large as a bean or hazel-nut, sometimes infiltrated with the new-growth and softened throughout their whole extent. The arches of the vertebræ, the trans-

verse and spinous processes, and even the muscles of the back are sometimes affected by the carcinomatous proliferation. The spinal nerves are compressed by the narrowing of the intervertebral foramina. When the vertebral cancer extends to the periosteum and dura mater, the cord also becomes involved, and the antero-lateral and posterior columns are then found in the condition of secondary degeneration to which we have so often referred. The carcinosis may affect different portions of the vertebral column, and usually involves several vertebræ simultaneously.

Vertebral cancer is rarely primary. The most frequent forms in this category are osteo-sarcomata and myxomata, which also involve other bones, and grow very rapidly, especially in young subjects. The fibrous and medullary carcinomata, which constitute the secondary varieties, follow cancer of the breast, stomach, liver, kidneys, uterus, and œsophagus.

The symptoms of vertebral cancer are too obscure and undefined in the beginning to reveal to us the severity of the approaching symptoms. The initial phenomena are those of a beginning spondylitis, but, before long, certain signs make their appearance which enable us to make a diagnosis. The following are the initial symptoms: *vertebral pains*, which are frequently intense, and almost always periodical (they may remain absent in a great many cases); *difficulty in the movements of the vertebral column*, which involves (especially when the process is situated in the upper parts) either the rotatory movements of the head, or the movements of flexion of the trunk; finally, the *dorsal curvature*, from softening and sinking in of the cancerous vertebræ, though the kyphosis never reaches such an extreme degree as in caries. Sometimes we may recognize the presence of hard, flattened tumors upon the transverse and spinous processes and even in the dorsal muscles.

At a more advanced stage a more characteristic and frequent (though not constant) symptom appears, viz., lancinating pains in the paralyzed limbs (paraplegia dolorosa of Cruveilhier and Charcot), due to compression of the nerve trunks at their exit, and to lesions produced in the nerves by the softened and flattened vertebræ. These pains, according to the situation of the disease, are manifested in the nerves of the brachial and cervical plexuses, but more frequently in the nerves of the abdomen and lower limbs. They increase paroxysmally (with remissions which gradually become shorter and more incomplete). The paroxysms occur especially during the night, and after active or passive movements. They become a terrible torture to the unhappy patient, and large doses of narcotics often prove powerless to afford the least relief. Sometimes these excessive pains subside spontaneously. According to Gull (l. c.) the pain is localized upon the side of the vertebral column when the lesion occupies the dorsal region. In the intervals between the paroxysms, when the condition of the patient has not become too depressed, he may still be able to move the limbs in bed.

As the disease progresses various accidents may develop, to which Charcot has especially called attention. These are: the appearance of *zona* in the territory of the affected nerves; *cutaneous anæsthesia* in small spots (generally under the form of anæsthesia dolorosa); *atrophy* and *contracture* of the muscles, and *arterial thromboses* (independent of *cachectic venous thromboses*). In Charcot's four cases the arterial thromboses occurred once in the middle cerebral artery, twice in the brachial, and once in the femoral artery. In the latter case the lower limbs presented paralysis of motion and sensation, cadaveric coldness, and livid spots.

The cord is variously affected in the different forms of cancer of the vertebræ. In moderate softening of the vertebræ the medullary parenchyma is only slightly involved, and over a slight extent. But when the vertebral lesions are more intense, the compression may cause softening and destruction of a considerable portion of the cord. I published a case of this kind in 1864 (2d edition of my Treatise on Electrotherapeutics), and this observation demonstrated how small a number of nerve fibres will suffice to maintain a satisfactory amount of motor power.

A man, æt. forty-seven years, stated that, for the past year, his legs have become weaker and weaker. On examination a prominence was found extending from the last cervical to the first dorsal vertebræ. The lower limbs were paralyzed and there was partial anæsthesia and abolition of electro-muscular contractility; reflex action can still be produced from the inner portion of the thighs. The patient was able to walk slowly when he was supported under the arms by an assistant. Upon the morning of the third day of his admission I found him completely paraplegic. There was complete anæsthesia of the lower limbs, the buttocks and the back as far as the upper dorsal vertebræ, and extending forwards to the sides of the abdomen. Electro-muscular contractility, the galvanic excitability of the nerves and reflex movements were completely abolished. Upon the following day paralysis of the sphincters occurred, with extreme dilatation of the right pupil. The patient died at the commencement of the second week.

At the autopsy the spinal column was found slightly curved in the form of a capital S, and, from the last cervical to the third dorsal vertebra, was replaced by a cancerous mass.

The cord was compressed to such an extent that the meninges were in apposition and only enclosed a small quantity of softened medullary tissue. The dura mater was thickened, and adherent to the arachnoid; the dorsal and lumbar portions of the cord were indurated. On microscopical examination, the degeneration was found to affect chiefly the posterior and lateral columns.

The diagnosis of cancer of the vertebræ is frequently encompassed with great difficulties. Certain cancerous products may remain latent during life, and, as the preceding history shows, carcinomatous compression of the cord may develop without the production of severe pain. On the other hand, curvatures of the spine and lancinating pains also occur in caries of the vertebræ and in compression of the spinal nerves by aortic aneurisms or by hydatid cysts. The peculiar qualities of the pain, in cancer of the vertebræ, consist in their extreme violence, their increasing intensity, and their resistance to narcotics. A paraplegia which becomes more and more marked, or a hemi-paraplegia dolorosa (Charcot), following a unilateral subsidence of the vertebræ, enables us to exclude caries of the vertebræ and indicate the existence of carcinoma or of a tumor developing in the spinal canal. The final appearance of arterial or venous thromboses, cancerous infiltration of the thyroid gland, digestive tract, breasts, testicles, abdominal viscera, lymphatic glands or bones, the increase of the cachexia, and, finally, the much more frequent occurrence of carcinoma at an advanced age, enable us to diagnose cancer of the vertebræ in certain cases in the living subject.

It goes without saying that the *prognosis* is absolutely unfavorable. The patient may survive for several months, a year or even more. They finally succumb to marasmus and dropsy, and, in lesions of the upper cervical vertebræ, to intercurrent cerebral symptoms.

The *treatment* consists in repeated injections of morphine, and in continually increasing doses of opium, chloral, or chloroform; these remedies may alleviate the excessive pains for some time, but they become more and more ineffective, and the patient greets death as the only deliverer from torture.

II.—COMPRESSION-MYELITIS, FROM PERI-MENINGEAL, INTRA-MENINGEAL, AND INTRA-MEDULLARY NEW-GROWTHS.

a. *Morbid Peri-Meningeal Processes.*

As Ollivier had already observed (l. c., T. II., p. 272), and as recent observations have more clearly shown, the loose cellular tissue situated between the spinal canal and the dura mater may be the seat of idiopathic inflammation, independently of the meninges. The resulting suppurations are either circumscribed or diffuse. They may constitute limited foci, or more or less extended collections of pus, and may produce compression of the cord, usually on the posterior surface.

The inflammations of the peri-meningeal cellular tissue are most frequently caused by extra-vertebral suppurations which have penetrated the inter-vertebral foramina. These suppurations occur near the vertebral column and pass through the inter-vertebral foramina to the spinal canal, as in Traube's and Mannkopf's cases, or the point of departure of the process is either in the psoas muscle or between the cervical vertebræ and the pharynx (Angina Ludovici of the Germans). In H. Mueller's case (l. c.) the suppuration occurred in the sub-pleural connective tissue. Caries of the vertebræ is also a frequent cause of peri-meningeal purulent infiltrations, when the pus, instead of pressing outwards, forms, so to speak, an internal congestive abscess in the spinal canal. We then find a circumscribed (usually round), purulent collection between the laminæ of the dura mater, which may be followed by compression of the cord, with the symptoms to which we have previously referred.

With regard to the symptoms and diagnostic signs, we refer to the previous remarks upon page 183 et seq.

The following new-growths are found in the peri-meningeal cellular tissue: lipomata (Virchow and Johnson), sarcomata, mixed enchondroma (Virchow), hydatid cysts, and many carcinomata, which break through the dura mater and compress the cord.

b. *Intra-Meningeal Neoplasms.*

The inflammatory neoplasms of this class consist of internal pachymeningitis (which terminates in rare cases in hematoma) and hypertrophic cervical pachymeningitis (Charcot). The clinical signs of this latter form of inflammation of the spinal dura mater, and of the circular compression which it exercises upon the spinal cord, have been discussed upon page 185. These meningeal tumors develop most frequently upon the inner surface of the dura mater, and more rarely upon the arachnoid and pia mater. If they are very small they exercise a unilateral compression upon the cord; when they increase in size the compression becomes greater, and secondary atrophy and softening occur. Apart from the great importance of the situation of the tumors, their volume, consistence, and development exert considerable influence upon the symptomatic manifestations. Small tumors, as large as a pea or bean (cysts, fibromata, neu-

romata of the cauda equina), present no appreciable symptoms during life. But tumors as large as a hazel-nut or walnut, especially when they develop within the cavity of the arachnoid, may produce severe lesions terminating in death.

Sarcoma and its derivatives (myxosarcoma, gliosarcoma, fibrosarcoma) are the most frequent among the tumors of the spinal meninges. Melanosarcoma (according to Virchow and Sander) develops especially in the arachnoid and pia mater, under the form of malignant pseudoplasms. Carcinoma rarely develops in the spinal meninges. The psammoma, formed of small crystalline masses deposited in a white substance, the myxoma, of a mucoid, gelatinous consistence, with a delicate areolar stroma, and the epithelioma, occur upon the arachnoid and pia mater, and sometimes also upon the nerve roots.

In unilateral lesions and compression of the cord by tumors, spinal hemiplegia sometimes occurs (Brown-Séquard), or when the symptoms are confined to the lower limbs, hemiparaplegia, with alternate hemianæsthesia, may develop. Charcot observed a case of this kind (Arch. de physiol., T. II., 1869) in a woman fifty years of age, who had complained for five weeks of weakness and pains in the left leg. There was paralysis of motion in this limb and in the abdominal muscles of the same side, with considerable hyperæsthesia; motion was less affected in the right leg, but sensation was abolished. At the autopsy an ovoid tumor, three and a half centimetres long and one and a half centimetres wide, was found about five centimetres above the thickest portion of the lumbar enlargement, and strongly adherent to the inner surface of the dura mater (psammoma, angiolithic sarcoma of Cornil and Ranvier).

We may finally mention the hydatids of the cavity of the spinal dura mater, of which two cases have been observed; the first, an old one, by Esquirol; the second, more recent, by Bartels (Arch. f. klin. Med., V. Bd., 1869). The latter case occurred in a man, æt. twenty-five years, who suffered from the onset from pains in the right hand, and then in the arm, shoulder, and clavicular region, and from pains in the neck and a feeling of constriction in the chest. Three months later numbness of the left and then of the right leg developed, followed by anæsthesia

FIG. 13.—Cystic sarcoma of the arachnoid, compressing the spinal cord, in the lower dorsal region.

and paralysis of these limbs, by paralysis of the sphincters, contraction and immobility of the left pupil, and finally by pains and weakness in the right arm, by bed-sores and pyæmia. At the autopsy an echinococcus vesicle was found at the lower half of the cervical enlargement, and the cord was compressed, flattened, and channelled with fissures upon its posterior surface.

The symptoms of intra-meningeal tumors correspond to the signs which we have ascribed, in a preceding chapter, to paralyses from progressive compression of the cord. At first the compression of the meninges and nerve roots causes intense lancinating, neuralgic pains, which radiate, according to the situation of the tumor, into the upper or lower half of the body, and are very frequently accompanied by vaso-motor irritation. The movements of the vertebral column soon become stiff, and circumscribed motor and sensory paralyses make their appearance. The signs of progressive compression of the cord then become manifest, especially the following: increase of reflex excitability, contractures of the limbs, spontaneous convulsions, hyperæsthesias, paralyses, rarely a spinal hemiplegia (in unilateral compression), generally paraplegia (often paraplegia dolorosa), and, finally, paralysis of the sphincters, cystitis, and bed-sores, which carry off the patient.

With regard to *diagnosis*, we must be especially careful in excluding vertebral affections (accompanied by deformity) and diseases of the meninges. A careful study of the symptoms, especially of the intense, radiating pains, the progressive increase of the paralysis, the muscular atrophy which rapidly increases under the influence of the softening of the cord, and the abolition of the electrical excitability of the muscles and nerve trunks, may, in certain cases, enable us to make a probable diagnosis of intra-meningeal tumors.

c. *Intra-Medullary Tumors.*

Tumors of the cord may proceed from the meninges, or develop in the parenchyma of the cord. New-growths of the first variety act, with regard to their influence upon the cord, like tumors of the meninges. But the neoplasms which form in the substance of the cord present numerous dissimilarities in their symptomatic manifestations.

In older observations, tumors of this category were ranged in the great class of sarcomata and carcinomata; but we cannot place much reliance upon these data with regard to the symptomatology. A very important observation was made by Hutin (Gottschalk's Samml., II. Bd., 1838), who found a fibrous tumor, as large as a hazel-nut, in the centre of the lumbar region, in an old man who had suffered from paraplegia and pain.

Foerster's case (l. c.) occurred in a boy, eighteen years of age, in whom a sarcoma was found in the centre of the markedly distended cord, and extending almost throughout its whole length. Carcinomatous infiltration of the cord has been observed in a similar case. A patient of Brown-Séquard (Course of Lect., 1860) suffered from stiffness in the neck, weakness of the right arm, and then of the leg, and finally died of meningitis. A cancer as large as an olive, and developing from the meninges, was found in the lower half of the cervical portion of the cord.

Among the intra-medullary tumors, whose history has been recently cleared up, we may mention the glioma (Virchow), which results from hyperplasia of the neuroglia, and consists of a finely reticulated basis sub-

stance, containing a soft tissue formed of nuclei and round or stellate cells. If the meshes of the network of cells enlarge and the mucous tissue increases, the tumor will be transformed into a myxoma. The glioma of the cord, like that of the brain, develops chiefly from the white substance; it does not pass beyond the pia mater, but gives rise to frequent hæmorrhages, on account of its great vascularity. It appears to grow less rapidly than the allied variety known as glio-myxoma.

In Schueppel's first observation (Arch. d. Heilk., VIII. Bd., 1867), the patient (a toper, fifty years of age) suffered, in the beginning, from weakness in the right arm, and then from stiffness in the neck and back. Power of motion was gradually abolished in the upper and lower limbs. A glioma was found in the right half of the lower cervical region, with old hæmorrhages in the adjacent parts and in the medulla oblongata, and a more recent hæmorhage in the gray substance of the dorsal cord. In the second case, the patient suffered, after a fall, from constricting pains, which radiated towards the abdomen, with heaviness and difficulty in the movements of the legs and persistent, but not painful, subsultus. After a safe confinement and marked improvement of the paralyses during the following four months, the woman suddenly sank into collapse and died, a week later, in a condition of general paralysis. The autopsy revealed the presence of a glio-myxoma in the gray column of the cord and extending from the medulla oblongata to the cauda equina; scoliosis existed without disease of the bones.

Tubercles of the cord are rare, and are generally accompanied by tuberculosis of the lungs or brain. They are most frequently situated in the cervical and lumbar regions and either in the white or gray matter. They vary in size from a millet-seed to a hazel-nut. Tubercles of the cord are usually isolated, and are very rarely grouped in several places. According to Lebert's statistics (Traité d'anat. path., T. II.), they occur with the greatest frequency from the ages of fifteen to twenty years (six times), and from twenty-five to forty years (four times); they are also proportionately frequent in childhood (two cases by Lebert). Tubercles of the cord, like other tumors, produce softening and inflammation of the medullary parenchyma by compression. When they involve the meninges, they may give rise to a spinal meningitis, and the inflammation may then extend into the cranial cavity. In tuberculous products limited to one side of the cord, unilateral paralyses (Eager) and paralysis of an arm (Laurence) may occur. Epileptiform attacks have been observed (Gendrin) when the tubercle occupies the cervical region near the medulla oblongata.

Gull's case (Guy's Hosp. Rep., 1858), of a tubercular tumor in the lower portion of the cervical enlargement, occurred in a child eight months old, and gave rise to stiffness of the neck, rotation of the head backwards, and paraplegia with spasmodic contractions. The two most recent observations upon tuberculosis of the cord were made by Chvostek (Med. Presse, 33–39, 1873). The first was that of a man, æt. thirty years, who presented the symptoms of a myelitis of rapid course (seven weeks), at first with hyperæsthesia, and then with anæsthesia in the lower and paraplegic limbs. At the autopsy, a tubercle as large as a pea was found in the lower dorsal region, with myelitis in the adjacent portions and in the inferior cervical region. The second patient suffered in the beginning from a sensation of cold, numbness, and violent pains in the bones of the left arm, and, later, in both legs. Paralysis of all the limbs and of the muscles of the trunk soon supervened. The muscles of the limbs were very much atrophied; the electro-muscular contractility and galvanic excitability of the nerves were markedly diminished. Convulsions occurred in the legs, both spontaneously and upon contact with the ground.

Death occurred in the ninth month, after increase of the paraplegia of motion and sensation. Autopsy: Tubercle of the inferior cervical region, as large as a hazel-nut, containing in its centre a cavity the size of a millet-seed with consecutive myelitis of the adjacent parts and of the left anterior horn.

It is extremely difficult to diagnose intra-medullary neoplasms. The deflections of the vertebral column from unilateral muscular paralysis (Schueppel's second case) will readily lead us to diagnose caries with myelitis from compression. The suddenly fatal hæmorrhages in gliomata of the cord may be mistaken for simple spinal apoplexies. In *differential diagnosis* we must pay especial attention (according to Cruveilhier, Charcot, Gull) to the absence of severe pains in intra-spinal lesions, as opposed to the intense pains occurring in chronic myelitis, from extra spinal compression. But there are exceptions to this rule, as shown by Chvostek's second case (quoted above), in which the patient complained of violent pains (with nocturnal exacerbations, as if the bones had been broken) in both legs and in the lumbar region. This patient had, furthermore, arrived at that advanced age at which carcinoma of the vertebræ is most frequently observed. Finally, increased reflex excitability and spontaneous convulsions (with or without pain) are also observed in tumors of the parenchyma of the cord.

One of the best diagnostic signs in intra-medullary neoplasms is the muscular atrophy consecutive to a central myelitis of the anterior horns, with its continuous progress and peculiar electrical reactions. The diagnosis of an intra-medullary product of a tuberculous character can possess no solid foundation unless we are able to discover, in a young subject, other signs of pulmonary, intestinal, or meningeal, tuberculosis.

CHAPTER XXI.

SYPHILITIC MYELITIS.

A CONSIDERATION of neoplasms of the cord and its membranes is naturally followed by a discussion of the myelitic processes and hyperplasiæ which follow syphilis, whether intra-meningeal, intra-medullary, or under the form of diffuse sclerosis. The researches made within recent times have considerably increased our knowledge, both of the semeiology and of the pathological anatomy of specific affections of the cord.

Pathological Anatomy.

Under the influence of constitutional syphilis, the vertebræ, like the bones of the skull, may be the seat, either in their periosteum or in their osseous tissue, of an inflammation which terminates in caries. These ulcerative lesions sometimes extend to the membranes and parenchyma of the cord. Although a large proportion of the vertebral exostoses referred to in medical literature are not positively due to syphilis, but rather to spondylitis deformans, of which we have previously spoken, there are, nevertheless, undeniable cases of syphilitic vertebral caries and of compression of the cord from periostitis or exostosis. Ollivier mentions cases of this kind (syphilitic caries of the cervical vertebræ, with dislocation, paraplegia, and rapid death; or with ulceration of the spinal meninges and hæmorrhage into the vertebral canal). The destruction of the anterior arch of the atlas or of the first cervical vertebræ, with ulceration and perforation of the spinal column, may also involve the cord (Autenrieth, Colles, Ollivier). Godelier, Piorry, etc., have seen periostitis or exostosis encroaching upon the lower part of the spinal canal (with symptoms of paraplegia). More recently, Virchow has observed syphilitic softening of the vertebræ with adhesion of the meninges; the cord was pale and firm.

The following changes occur in the spinal meninges: induration, thickening and adhesion of the dura mater to the bodies of the vertebræ, and marked pigmentation and adhesions of the meninges to each other and to the cord. Syphilomata, which develop upon the spinal dura mater, may also compress the cord (as in a personal observation which I shall report at a later period). Finally, the nerves alone may be torn, thickened, and adherent to the dura mater (Delafield, cheesy tumor, as large as a pea, in the nerves of the cauda equina, with thickening of the interfibrillary connective tissue, cellular proliferation and fatty degeneration of the nerve fibres).

Gummy tumors (Moxon, Charcot, and Gombault), abnormal coloration or partial sclerosis of the posterior and lateral columns, atrophy of the gray substance (Bruberger), partial atrophy with sclerosis of the cornua, and deformity of the nerve cells (Charcot), have been observed in the

spinal parenchyma. We may mention, in conclusion, that Petrow (Virch. Arch., 57. Bd., 1873) has twice seen degeneration of the sympathetic nerve in syphilis. The nerve cells presented pigmentary or colloid degeneration, with fatty degeneration of the epithelium. The interstitial tissue was in a condition of active proliferation, terminating in atrophy of the nerve fibres and cells.

Etiology.

The first medullary symptoms are usually manifested several years after the infection. In exceptional cases, they are combined, at a later period, with alterations of the cranial nerves. The largest number of these cases are observed in young and middle-aged subjects. No observations have been made, up to the present time, concerning the vascular system of the cord in syphilitic patients, analogous to the specific degenerations of the vessels seen in cerebral syphilis and to their influence upon the development of thromboses and foci of softening.

The inflammatory lesions of the meninges and nerve roots, and the gummy tumors and diffuse sclerosis of the white and gray substance, suffice to explain the more serious changes in the medullary centres. It is, therefore, superfluous to admit functional disorders and paralyses of the spinal functions which are directly due to the diathesis. Judging from the initial symptoms, the vascular irritations may also serve as the starting-point of the morbid processes.

Symptomatology.

The myelitis due to syphilis, like simple myelitis, attacks the patient insidiously. Slight, vague pains, tingling in the limbs, temporary, localized sensations of heat or cold, pricking and formication, are usually the symptoms which attest the development of these latent irritations of the cord.

At a later period they are characterized by abnormal sensibility of the spine, fugitive muscular spasms, weakness of both legs or arms, and a feeling of lassitude. At a more advanced stage the paralysis of the lower limbs becomes more and more manifest, the different varieties of sensibility are unequally affected (either analgesia or anæsthesia), weakness of the sphincters occurs, very frequently with diminution of electro-muscular sensibility and contractility, and, finally, all the well-known symptoms of myelitis.

The cases of this kind published by Potain, MacDowel, Wilks, and E. Wagner are allied to an observation which I published in 1865, and which may be regarded as the first published example of a syphiloma of the spinal meninges.

A working woman, æt. twenty-eight years, had suffered, since the beginning of Jan., 1865, from neuralgia in the legs, followed by rapid paralysis of motion. I examined the patient in the autumn; she was anæmic and paraplegic in the lower limbs, which were considerably emaciated, and presented anæsthesia, analgesia, and very marked diminution of electro-muscular contractility (especially of the extensors). Towards the close of the year the patient died from cystitis and bed-sores. At the autopsy, a rounded gumma, as large as a hazel-nut, and whose free surface pressed against the dura mater, was found in the centre of the left parietal bone. A second gumma, of the thickness of a finger and about three centimetres long, developed from the spinal dura mater and compressed the cord upon the left side, from the second to the fifth cervical vertebræ.

I afterwards learned that this patient had suffered, during the year 1863, from symptoms of secondary syphilis.

In a more recent case reported by Moxon (Guy's Hosp. Rep., V., XVI., 1871) the patient, æt. thirty years, had suffered, seven years previously, from chancre, bubo, and a cutaneous eruption. After a long period of quiescence of the disease, he complained of numbness and weakness in the legs, and abolition of sensibility to contact, pain and temperature in both lower limbs. Electro-muscular contractility was diminished and electro-muscular sensibility entirely lost; reflex action was preserved. Vesical paralysis, abscess of the left buttock, and fever developed toward the close of life.

Autopsy: foyers of sclerosis in the cranial vault, as if from old nodes; considerable pigmentation of the medulla oblongata and spinal cord; the cord was indurated in its upper fourth, and very much softened below. The sclerosed patches in the cord were composed, externally, of a fibrous layer, and, internally, of a mass of whitish yellow, gummy matter, and were found especially in the posterior and lateral columns. The testicles were inflamed, and also contained gummy nodules similar in character to the foregoing.

Unilateral lesions of the cord, produced by syphilitic tumors, are especially interesting from a clinical and diagnostic point of view. The first observation of this nature was made by Brown-Séquard (Lect. on Diag. and Treat. of Paral. of the Lower Extremities, London, 1861) in a man, who presented several patches of periostitis upon the head and face, and who suffered from paraplegia, with paralysis of the right and anæsthesia of the left leg. This led to a diagnosis of a syphilitic tumor compressing the right half of the cord. We shall now cite a much more important case, in which the investigations were made during life and post-mortem. The case is one of disseminated syphilis of the brain and cord, which was published in detail by Charcot and Gombault (Arch. de physiol., March, 1873).

A woman, forty years of age, had contracted syphilis twenty years previously. A long time afterwards she began to experience twinges, numbness, and articular pains in the left leg, with gradual weakness of motion. Still later, she suffered from headache at the vertex, with dilatation of the left pupil. Upon examination of the patient the symptoms found were: paralysis and hyperæsthesia of the left leg, which was considerably emaciated; anæsthesia without paralysis in the right leg; spontaneous pains over the third and fourth dorsal vertebræ, radiating into the anæsthetic intercostal spaces (anæsthesia dolorosa); below these points, upon the right side, there was analgesia without tactile anæsthesia; the left side was hyperæsthetic. Epileptiform attacks then made their appearance, followed by paralysis of the left external rectus and right facial paralysis (with diminution of faradic, and increase of galvanic reactions). The right optic papilla was cloudy and infiltrated with serum. Towards the end, paralysis of the right motor oculi communis and externus occurred, with double optic neuritis, marasmus, somnolence, fever, bed-sores, and fatal pulmonary œdema.

Autopsy: The skull and cranial meninges were normal. The left optic tract and right peduncle contained gummy plaques (formed externally of branching cells and abundant nuclei and, in the centre, of granular cells); the optic nerves and corresponding ocular nerves were atrophied. Two patches of a reddish-gray color were situated upon the anterior surface of the pons, a small one upon the left and a larger one upon the right side. Another yellowish patch was found in the interior of the pons, at the level of the origin of the trigeminus. Other plaques were present in the floor of the fourth ventricle, in the anterior portion of the lateral columns of the isthmus, upon the left side, and in the neighborhood of the posterior tubercula quadrigemina. A hard tumor, one centimetre in diameter, was situated between the roots of the third pair of dorsal nerves upon the left side. The pia mater was thickened and the nerve roots were sclerosed at this point. Below there was sclerosis of the lateral columns, and above,

sclerosis of the posterior columns. The left anterior and posterior horns were confused and poorly defined; a portion of the nerve-cells was replaced by a thick reticulated tissue; a large number of the cells of the anterior horns, however, were intact. The commissure, a part of the right anterior horn and the two posterior columns, were sclerosed.

In a very recent case reported by Bruberger (Virch. Arch., 60 Bd., 1874), paralysis occurred upon both halves of the body (with almost complete preservation of sensibility), in addition to the apoplectiform symptoms of cerebral syphilis. The autopsy revealed the presence of extensive basilar meningitis, thickening of the cerebral arteries, and normal condition of all the other portions of the vascular system. The cervical meninges, which were matted to one another, formed a thick membrane which adhered firmly to the cord, and loosely to the inner wall of the spinal canal. The gray substance was in a condition of atrophy (no microscopical examination) and the central canal was dilated.

Diagnosis and Prognosis.

If we refer to the symptomatology which we have sketched of the disease under consideration, it will be found that a very favorable grouping must be present, in order that the diagnosis of spinal syphilis may be made with certainty during life.

In Charcot's and Gombault's patient, who suffered from inveterate syphilis, the crossed hemiparaplegia and hemianæsthesia indicated a lesion of the left half of the cord, while the coexistence of epileptiform attacks, of ocular paralyses, of alternate facial paralysis (with abnormal electrical reactions), and of double optic neuritis, pointed to the existence of multiple foci in the medulla oblongata, pons varolii, cerebral peduncles, and in the optic tracts. The localization of specific lesions of the brain and cord depended, therefore, in this case, upon the grouping of the symptoms.

In the other cases the arrangement of the symptoms was not, by any means, so favorable, and the autopsy alone revealed the connection of the medullary symptoms with the syphilitic diathesis. In observations of this kind we can only make a positive diagnosis if we are able to determine the coexistence of syphilis and myelitis. When any vague symptoms of spinal disease are presented, especially in a young subject, we should carefully examine into the antecedents with regard to syphilis. We must not be deceived by the denials of the patient, and must search with the laryngoscope and the ophthalmoscope, for traces of syphilis wherever they may be concealed. Whenever a diathetic affection of the medullary centres has developed we will also find other vestiges of the constitutional infection. In the case which I observed a careful examination would have discovered the characteristic cicatrices in the vagina, and would have permitted a diagnosis during life.

The *prognosis* of syphilitic myelitis should be very guarded. When the economy has been infected for several years, the appearance of significant disorders on the part of the cord must be regarded as a serious event. Although in these cases, as in cerebral syphilis, we can not deny that therapeutic intervention may prove successful in benign forms, on the other hand, the examples referred to above serve to show that neither youth nor the appearances of good health enable us to affirm that the sequences of the diathesis will ever be removed from the spinal system. In

one case of inveterate syphilis, with ataxic symptoms, I obtained improvement by repeated medicinal and hydrotherapeutic measures; but the amelioration was not of long duration, the spinal lesion again assumed the upper hand and the symptoms grew gradually worse.

Treatment.

In all spinal affections in which there is a suspicion of syphilis, and especially when the patients present, in addition to the medullary symptoms, other signs of the diathesis, we must immediately institute a methodical specific treatment, regulated by the general condition of the patient. In young subjects we should prescribe a trip to the country and long-continued hydrotherapeutic measures (wet and then dry packs, continued until moderate perspiration is produced, and followed by gradually cooled half-baths). In neuralgias and conditions of weakness we often derive benefit from the cautious combination of hydro-therapeutics and galvano-therapeutics.

DISEASES LIMITED TO CERTAIN REGIONS OF THE CORD.

After having considered the general inflammatory processes which may extend throughout the entire cord, we shall now study the morbid types which are limited to certain regions of the cord. Considerable differences are here manifested, both from a histological and physiological point of view, and are evidenced by perfectly distinct clinical forms. The progress made in recent times has especially aided in furthering our knowledge of the characteristics of the diseases in question.

Starting with the posterior portion of the cord, we shall first examine the forms of tabes or locomotor ataxia, resulting from sclerosis of this region. Then passing to the lateral halves of the cord we shall investigate lateral sclerosis and its varieties, and the crossed hemiplegia and hemianæsthesia, with which experimentation and clinical observation have made us acquainted. Finally, we will pass in review the motor and trophic disturbances due to lesions of the anterior circumference of the cord, and especially of the anterior gray horns, as has been demonstrated in infantile spinal paralysis, in the acute spinal paralysis of adults, in progressive muscular atrophy, and in the mixed forms of these various affections.

CHAPTER XXII.

DISEASES OF THE POSTERIOR COLUMNS OF THE CORD.

Hutin (Bull. de la Soc. Anat., T. II., 1827), Monod (Bull. de la Soc. Anat., 1836), and Ollivier (loc. cit.) were the first to demonstrate that the anatomical lesion of tabes consisted of a degeneration of the posterior columns. Abandoning the narrow etiological views which had been hitherto entertained, Horn, Hecker, Naumann, Brach, Romberg, Steinthal, and Wunderlich, were forced to pay more attention to the clinical aspects of the disease in question. The ataxic disorders of movement were first attributed by Bouillaud (Nosographie Médicale, V., 1840) and then by Todd (Cyclop. of Anat. and Physiol., 1847) to a disturbance of coordination. Twelve years later Duchenne (Arch. Gén., Dec., 1858, and Jan., Feb., and April, 1859), in his remarkable investigations upon progressive locomotor ataxia, called the attention of physicians to this disease and its symptoms, with a clearness and precision which Romberg and Todd were unable to attain.

Pathological Anatomy.

Upon fresh sections of the cord the posterior columns are found very much diminished in size, retracted, of a grayish color and a firm (rarely soft) consistence. According as the disease has involved the upper or lower limbs the lesions will predominate either in the cervical or lumbar region. In many cases the degeneration extends even higher, and may

be traced into the gray substance of the floor of the fourth ventricle, and into the pons and cerebral ganglia. We find upon microscopical examination of transparent sections of the hardened cord that the nerve fibres are changed into a substance which is strewn with fine granulations and fatty molecules, and are replaced by fibrillary connective tissue. If the preparation is treated with a weak solution of ammonia carmine, the axis cylinders will become more distinct, and we can frequently recognize, even with the naked eye, a cuneiform imbibition, corresponding to the degenerated posterior columns. Accumulations of fat and pigment are often observed in the vessels, and some are filled with nuclear proliferations. The fundamental granular substance contains varicose capillaries, with thickened walls, and is infiltrated with granules and amyloid corpuscles. Marotte, and especially Lockhart-Clarke, have found the nerve cells of the *posterior horns* partially affected by sclerosis. The *posterior nerve roots* are thin, hard, and affected by fibrous degeneration. The sciatic, crural, and brachial nerves are sometimes strewn with an interstitial connective tissue, rich in nuclei, with diminution of the primitive fibres (Friedreich). Several *cranial nerves*, especially the optic, motor oculi, and hypoglossus, also present gray degeneration and atrophy. Finally, the *meninges* are often, but not always, thickened and adherent along the posterior columns.

According to the later researches of Charcot and Pierret (Arch. de Physiol., 1872–3) the lesion involves not alone the median cuneiform portion of the posterior columns (columns of Goll), but also extends to the external portion which borders upon the posterior horns. This portion has been regarded by Stilling, L. Clarke, and Koelliker, as the intra-medullary expansion of the posterior roots (internal root fibres of Koelliker), and unites various parts of the posterior gray substance by means of arched commissural fibres. The sclerosis of the posterior columns develops from the centre of this portion, and then extends to the posterior roots. *The lesion of these postero-external columns, without participation of the postero-median columns, will suffice*, according to the above-mentioned authors, *to give rise to ataxia*. In advanced cases the degenerations finally involve the median columns.

The sclerotic process not alone tends to propagate itself from the lateral to the median portions of the posterior columns, but it may also be transmitted to the posterior horns, and sometimes to the posterior portion of the lateral columns. In the vertical direction, the sclerosis of the lateral portions of the posterior columns may be traced into the medulla oblongata, where it involves the restiform bodies. Usually the median portion of the posterior columns is also the seat of a secondary degeneration, which proceeds from below upwards. The complication with progressive muscular atrophy, which is sometimes observed in ataxia, is due to an extension of the morbid process to the network of nerve fibres which, according to Gerlach (Stricker, Handb. der Gewebelehre, II. Bd.), connect the internal root fibres with the cells of the anterior gray horns. In a case of this kind, Charcot found atrophy of the right anterior horn, in addition to the degeneration of the posterior columns. I have had the opportunity of examining (Sept., 1871) the interesting preparations made by Charcot upon this subject.

Etiology.

The alarming number of cases of ataxia and other spinal diseases observed in our country proves that this predisposition must have its

source in causes which form an intimate part of the social conditions of our times. If we consider the enormous frequency of anæmia in large cities (and especially in Vienna); the large number of females who suffer from nervous and hysterical affections; the numerous examples of precocious excitement, perversion or abuse of the genital functions; the many hardships to which men are exposed in the struggle for existence—can we wonder that, after such numerous and profound disturbances of the energy and force of resistance of the nervous system, debility and nervous diseases, with all their sad consequences, should be transmitted from parent to child? We can thus explain the fact that nervous diseases are so numerous in the present generation, and that ataxia is found so often in families whose members present, almost continuously, some varieties of nervous disease.

Trousseau has several times observed ataxia in families in which closely related members suffered from hypochondria, monomania, epilepsy, or convulsions. In Friedreich's observations, in one case two, and, in another, four relatives were affected with ataxia. Carré mentions a family of ataxics in whom the grandmother, mother, seven brothers, and sisters, and nine relatives were undoubtedly ataxic. In a family under my care, the mother, who died of tuberculosis, had frequently had convulsions, one son was epileptic, another ataxic, and a daughter died of hydrocephalus. In another family, two brothers, and in a third, the father and son, were ataxic; in a fourth the grandfather died of apoplexy, the father became ataxic, and the son suffered from chorea.

It is evident, from the preceding statements, that the development of ataxia in certain families may be due to a predisposition to nervous disorders. When a morbid congenital predisposition exists, the nervous system will suffer from certain external influences, which would have no injurious effects upon healthier and more resistent individuals. Certain delicate constitutions will succumb more rapidly to anxiety and over-work, and are more seriously and persistently affected by outside influences, than others who were originally stronger, or better accustomed to and prepared for resistance.

Among the external influences which are injurious to the nervous system, we must mention the effects of cold as among the most important. I have several times had occasion to point out the rheumatic origin of ataxia. Among the considerable number of cases which I have observed in the Vienna General Hospital, during a long series of years, cold has figured as the most frequent cause, especially in the working-classes, in which the men are so frequently and seriously exposed, by their work in the open air or in the water, to injuries from cold and from the strong winds which are prevalent in our climate. The remarkable frequency of ataxia among our laborers, upon whom the severity of their daily labor and the hardships of life impose moderation in sexual gratification, is opposed to the opinion that venereal excesses are the chief cause of ataxia. We also find ataxia (more rarely chronic myelitis) developing after exposure in different professions. I have seen the first irritative symptoms of ataxia follow exposure among merchants, farmers, hunters, engineers, etc., who are compelled to travel or work in the cold, and in snow or rain; among architects, who must often work in water and undergo various hardships; and among laborers, who work in canals, in ice, and at the most inclement seasons of the year. According to the individual idosyncrasy, or to the extent and duration of the action of cold, the irritation will affect the skin and muscular nerves (muscular rheumatism), or will produce

neuralgia of more important nerve-trunks, like the sciatic nerve, or it will give rise to rheumatic paralyses of certain groups of muscles, or even to vascular excitation of the spinal system, in which the repeated or prolonged congestions may give rise to morbid processes, as has been already demonstrated with regard to the brain. The abnormal irritation produced by cold upon the peripheral sensory nerves may be transmitted to the cord by following the nerve trunks and roots. According to Feinberg's recent experiments, intense cold applied to the cord in animals may also produce myelitis.

Other testimony also evidences the influence which cold possesses upon the functions of the nervous system. Thus, while venereal excesses and other causes of exhaustion only give rise to ataxia after a long time, a severe exposure of short duration will suffice to immediately develop the first germs of ataxia, which then progress rapidly, especially if the action of the cold is repeated. The affection of the nervous system will depend very much upon the period at which the exposure acts. When the nervous system is in a condition of repose, it is able to sustain, with impunity, many external shocks. But when the nerves are in a condition of excitement and have been overworked for a long time, they will not only feel the action of cold acutely, but this irritation may extend to the central organs, even to the nerve cells, and give rise to the first elements of disorder in their delicate structure.

Venereal excesses also add a considerable contingent to this disease. But if sexual excess were really such a frequent cause of ataxia as is generally believed, large cities would be filled with ataxics. A much larger proportion of cases of ataxia originates from two other forms of genital excitement, viz.: masturbation and habitual pollutions.

There are numerous occasions for observing the profound influence of unnatural genital irritation upon the spinal functions and upon general nutrition. I believe that unnatural intercourse will affect the medullary activity less than the excessive excitement accompanying ejaculation in onanism. I know that pathological changes, following masturbation, are sometimes observed in monkeys. But the male population would be in a very deplorable condition if masturbation would produce ataxia in the majority of cases in which it is practised. The more infrequent the practise of onanism, the better the forces of nature will be able to repair its evil effects. On the other hand, if masturbation is practised to an extreme degree for a number of years, and is even continued to a ripe age, a morbid condition of the nervous system will be produced, and the germs of spinal diseases may develop at a later period, when other noxious influences are brought into play. Seminal losses may also affect the nervous system, by their frequency and long duration. These pollutions are more frequent in men who have previously practised onanism. The pollutions from psychical excitement are relatively less grave. The longer the duration and persistence of these seminal losses, the more serious will be the effect of the repeated congestions of the cord which follow them. When this condition continues several years and the resistance of the nervous system is shaken, a central affection may develop.

Among the pathogenic factors of ataxia we must also mention certain causes of exhaustion, such as severe exertion and mental anxiety, which may seriously compromise the spinal functions, especially in very impressionable patients. Ataxia also develops, at times, after syphilis, severe typhoid fever, repeated confinements, chronic hemorrhagic fluxes, and frequent and prolonged lactation.

The male sex furnishes a much larger proportion of ataxics than the female. The largest number of cases develop between the ages of thirty and fifty years; they are most frequent between forty and fifty. I have never seen ataxia in children, but merely rapid myelitis after severe exposure.

Symptomatology.

Ataxia, when accompanied by palpable disorders of co-ordination, anomalies of sensation, and functional paralyses, is a disease which it is impossible to mistake. It is much more difficult, however, to recognize the first stages of this affection. The onset of ataxia often passes unnoticed for a long time, and disguises itself under the appearance of vague neuralgias, rheumatic or gouty pains, hemorrhoidal troubles, or spinal irritation. Nevertheless, in the majority of cases, the careful physician will discover symptoms which reveal to him the gravity of the latent affection, despite the difficulties connected with diagnosis in examining the patient for the first time.

Disorders of Sensation.—These disturbances are due, in the majority of cases, to vascular or inflammatory irritation of the cord, especially of the nerve roots and of their prolongations into the gray substance. *The shooting pains in the limbs*, which follow one another more or less rapidly, *are usually accompanied by cutaneous hyperæsthesia, chills, and acceleration of the pulse* (from irritation of the internal root fibres, Charcot). They occur more frequently along the inferior than along the superior branches of one of the sciatic nerves, or along the lumbar nerves. In other cases the patients suffer from deep-seated piercing pains; girdling pains (around the trunk) often predominate. These intermittent neuralgias only yield incompletely to therapeutic means. After they have lasted a certain length of time (sometimes even from the beginning), pains occur in the branches of the cervical or brachial plexus upon one side (with painful spots in the posterior scapular region and tenderness of the brachial nerves upon pressure), with numbness and tingling in the hands, fingers, or toes, and temporary twinges of pain in certain intercostal spaces. When the irritation extends to higher parts, it produces cerebral congestion, with a sensation of fulness, compression or constriction, and with or without notable elevation of temperature. Painful twinges in the course of the trigeminus, and a feeling of tension in the neck, occiput, and in the eyes often occur. In rarer cases, cardiac palpitation and syncope make their appearance in consequence of reflex spasm of the cerebral vessels.

Rachialgia presents various characteristics in ataxia. The spinous and transverse processes and the skin and muscles which cover them are unusually sensitive to pressure from irritation of the posterior roots of the spinal nerves. This hyperæsthesia is not a constant sign of ataxia or myelitis. Schiff has shown that if turpentine is poured upon one of the axillary nerves, the trunk of the nerve will become red and painful, although the sensibility of its terminal branches was not markedly increased. In an analogous manner, the congested or even inflamed nerve roots may give rise to a subjective, though not objective, hyperæsthesia of their terminal branches. In certain ataxics, in whom neither pressure nor exaggerated rotation of the trunk produced pain, I have seen the application of strong galvanic or faradic currents produce peripheral symptoms of irritation, such as burning or shooting pains in the legs or soles of the feet. Galvanic exploration of the lower cervical and upper dorsal vertebræ in an ataxic

produced muscular contractions in the left arm (secondary irritation of the corresponding nerve fibres), while irritation of the right upper limb produced no appreciable action.

The pains may extend to the anterior portions of the thigh and leg, in which certain portions of the nerves are found sensitive to pressure, and prolonged compression, lying upon the side, or the wearing of a tight-fitting shoe, soon become insupportable. Pressure of the finger upon the sciatic notch, or on the points of emergence of the lumbar nerves, produces pain (though not always), and is accompanied by a distressing sensation, on account of the frequent coexistence of cutaneous and muscular hyperæsthesia.

In the majority of cases the *sensibility to tickling* disappears first; *tactile sensibility* is profoundly changed and often abolished; the *sensibility to pain* rarely disappears completely but merely presents perversions. *Sensibility to temperature* is only altered at a later period, and the sensibility to cold is especially increased. *Muscular sensibility* is usually profoundly changed; *the delay of sensory impressions* (on account of obstacles to transmission in the gray substance) may even amount to two or three seconds. When a more extended anæsthesia develops at a later period (from lesion of the posterior roots and horns), it may even affect the articulations. Movements communicated to the toes, metatarsus, ankle, or even to the knee, do not penetrate the consciousness of the patient. Great difficulty is experienced in assuming the vertical position (on account of the want of stability in the knees) and in walking (the foot does not disengage itself from the ground); the movements of rotation are also poorly executed. At this period the muscular sense is generally involved. When the eyes are closed, or during the night, the patients are unable to distinguish the position of their limbs or of their relations to one another. By means of a descending galvanic current applied to the nerves or muscles, marked contractions are sometimes obtained, which are perceived only slightly or not at all by the patient when his attention is diverted. Cutaneous sensibility is also profoundly changed over the surface extending from the loins to the buttocks, scrotum, and penis.

Motor Disorders.—These disturbances usually appear at an early period. The patients experience a distressing sense of tension in the limbs, the knees bend under from time to time, and they soon complain of fatigue when walking (especially without a cane). This feeling of prostration often inconveniences the patient to a very marked extent when rising from bed (morbid perversion of the muscular sense ?). From time to time, and especially after exertion, temporary muscular spasms occur in the upper and lower limbs and in the trunk and neck. The vertical position, rapid rotation upon one foot, rapid walking, ascending a flight of stairs, and running are performed with difficulty, and are usually followed by an exacerbation of the pains in the legs or back, and by a more marked feeling of constriction in the head.

This weakness of movement may increase slowly for the space of a year, but in unfavorable conditions, or on account of the original intensity of the disease, it may promptly terminate in abolition of motion. The energy of the movements then diminishes rapidly, stiffness and stamping become very evident during walking, the legs act unequally and are thrown apart when placed upon the ground. In order to sustain the vertebral column in its unstable equilibrium, a curve is formed towards the sound side from interruption of the antagonism between the dorsal muscles and from paresis of one side.

The stability of the knee-joint is compromised from weakness and atrophy of the extensors of the thigh. The paresis of the muscles of the pelvis causes great difficulty in straightening the trunk after bending forwards and in performing the oscillations, which are normally executed upon the head of the femur. Other characteristic signs also develop, such as oscillations when the patient remains for some time with the eyes closed and the feet close together (Brach's symptom); inability to stand or hop upon one foot with the eyes closed; inability to turn to the right or left when the feet are kept close together. Clonic spasms often occur in the extensors of the thigh and in the buttocks, especially in the vertical position with the eyes closed. The oscillations are caused, in my opinion, by relaxation of the muscular tonus in the extensors of the muscles of the pelvis. At a more advanced period we find, in a number of cases, the movements of projection of the limbs, the stamping, and the want of harmony in the combined action of the muscles, which is known as ataxia (paralysis of co-ordination). In very advanced cases, even paralysis may occur in a larger or smaller number of muscles. Ataxic weakness of the upper limbs rarely occurs at the onset of the affection, but only after the motor disturbances have appeared in the lower limbs (sclerosis of the inner root fibres of the posterior columns in the cervical region, Charcot and Pierret). We then observe formication and anæsthesia in the hands, and inability to grasp objects firmly; the writing becomes labored and hesitating, and the patient experiences great difficulty in rounding the letters. Walking is only possible with the aid of a cane, but the latter is readily entangled between the legs of the patient. I have seen many cases of this kind confounded with writer's cramp.

Voluntary movements are hindered, on account of the disturbances of co-ordination by antagonistic movements of the limbs, attended usually with abnormal increase of reflex action. In two cases which I observed the reflex movements were so marked that the patients, when once started, were incapable of coming to a stand-still, but continued to walk with an involuntary impulse. They were compelled to seize hold of some object in order to arrest their progress.

The galvanic excitability is abnormally increased in the irritative forms of ataxia. This is manifested by the appearance of contractions at the closure of the cathode, often by tetanic contractions upon application of weak currents, or by the enormous increase in the intensity of the contractions from currents of short duration or inverse direction (even with weak currents), or when the resistance is diminished by the use of the rheostat. Contractions often appear more rapidly and strongly upon closure at the anode than at the cathode, or contractions may even appear with more intensity when interrupting at the cathode. The farado-muscular contractility may also be increased at first, but is markedly diminished in the chronic forms and when paralysis begins.

Fever sometimes occurs in the initial period of ataxia, and has been investigated of late years by Finkelburg (Verh. d. Niederrhein. Ges. f. Natur- u. Heilk., 1864) and by Clemens. These febrile symptoms are especially important with regard to the nature of the disease. In the majority of cases they correspond to an exacerbation in the inflammatory processes occurring in the medullary parenchyma and meninges. The onset of ataxia is marked, more or less frequently, by changes in the pupils and by phenomena of irritation on the part of the bladder, genital organs, and vascular nerves, which are due to irritation of the corresponding centres in the cord.

The *pupillary changes* are due to a lesion of the cilio-spinal centre in the upper portion of the cervical cord. Both pupils may be contracted to the size of a pin's head, may be insensible to light and atropine (paralysis of the iris with anterior convexity), and only become dilated during paroxysms of pain. More frequently, however, one of the pupils, especially upon the weaker side, is markedly dilated and sluggish, and sometimes both pupils are in this condition; the dilatation may also disappear from time to time, and the contraction becomes more energetic under the influence of light, or we may even notice alternations of contraction and dilatation.

We sometimes observe neuralgia of the urethra, the neck of the bladder, and the rectum, from irritation of the genito-spinal and ano-spinal centres (Budge). The neck of the bladder and the urethra present intense hyperæsthesia, often attended with vesical tenesmus, pain during micturition and violent periodical pains in the urethra, which may be combined with voluptuous sensations. In rare cases the pains radiate into the spermatic cord, the testicles, and lower limbs. Very frequently, also, a few drops of a clear fluid appear at the meatus after violent genital excitement and after micturition. This fluid proceeds partly from the prostate gland, and partly from Cowper's glands, and is usually regarded as indicative of chronic urethritis. Some patients complain of a sensation of burning, compression or lancinating pains in the rectum.

Disorders of the Cranial Nerves.—These constitute some of the most serious and frequent complications of ataxia. The optic nerve is most commonly involved (fifty-one times in one hundred and two cases, according to Topinard), and gray degeneration is much more marked in it than in the other cranial nerves. There is, at first, a concentric narrowing of the field of vision, with diminution in the sharpness of sight, which finally terminates in complete blindness (amaurosis). With the aid of the ophthalmoscope the papilla is found to have sharp outlines, has a shining white color, and the retinal arteries are diminished in size. Both eyes are usually affected to an equal extent. In some cases the early amaurosis is the first symptom indicative of ataxia.

In many cases the diminution in the power of sight is accompanied by partial abolition of the perception of colors (Daltonism). After a gradual diminution in the perception of colors at the external boundary of the field of vision (Schoen, Birmer), the perception of green and red is first weakened, and then entirely abolished. Yellow, with its infinite variety of shades, and blue remain perceptible for the longest period. As long as the colors persist at the outer limit of the field of vision, the prognosis, according to Schirmer, is not unfavorable.

The *motor nerves of the eye* also become involved at an early period. The paresis or paralysis occurs most frequently in the motor oculi communis, less frequently in the external motor oculi, and very rarely in the patheticus. Ptosis, diplopia, and strabismus may appear as temporary symptoms of irritation, and disappear with or without treatment and without any noteworthy effect upon the primary disease. They may reappear after long absence, or, on the other hand, they may persist with the other symptoms of the disease. Paresis of accommodation may also develop and is likewise susceptible of recovery. Oculi-motor paralysis of cerebral origin is usually accompanied by intermittent cephalalgia, vertigo, intellectual disturbances, simultaneous affection of other cranial nerves, and coincident or alternating paralyses of the limbs. Oculo-motor paralyses of spinal origin are distinguished by the coexistence of frequent

sciatic neuralgia, with cutaneous hyperæsthesia or other neuralgic pains, especially in the lower limbs, by the rapid exhaustion of the motor functions, the weakness of the genital system, the different results obtained by ophthalmoscopic examination (cf. page 101), and, finally, by the increased electrical excitability to which we have referred above.

The *trigeminal* nerve is sometimes implicated in ataxia. In a case observed by Duchenne, double paralysis of the fifth pair of nerves occurred, with paralysis of the left motor oculi communis. In four of my cases, paralysis of the trigeminal and motor oculi communis occurred twice; paralysis of the latter nerve, and of the facial and trigeminal, developed once; and, in the fourth case, anæsthesia was observed upon the buccal mucous membrane and perversion of taste upon the left side. The paralysis of the trigeminal is usually incomplete. The *facial* nerve is rarely involved in its totality, but certain muscles of the face manifest diminished tonus. Duchenne, however, has seen complete paralysis of the facial nerve in a case of ataxia.

The *acoustic* nerve is not always exempt. In Topinard's one hundred and two cases, hearing was affected ten times, and I have observed disturbances of audition in five cases (enfeebled hearing upon one side). In the majority of cases, the patients complain of annoying noises in the ears, which prove intractable to treatment. The lesion of the semi-circular canals, observed by Lucas in a case of gray degeneration of the posterior columns, must be regarded as an accidental complication, since Politzer made a smilar observation in a case of tuberculosis.

Functional disorders of the *hypoglossus* not infrequently develop during the course of ataxia. I have noticed disturbances of speech in eight cases of ataxiæ; Friedreich has seen it six times, and Topinard twenty times in one hundred and two cases. The patients experience some difficulty in speaking, especially in the pronunciation of certain words. The motion of the tongue is little affected, except when the organ is protruded, when it is seized with strong tremor and spasmodic movements. The stammering must be looked upon as a variety of ataxia of the muscles which take part in speech. The disturbances of speech usually appear after other disorders of the senses or of the organs of movement. When the hypoglossus is involved at the same time with the other nerves which take part in deglutition, the latter function is rendered very difficult of performance.

Irritative phenomena also appear, at times, on the part of the *pneumogastric* and *spinal accessory* nerves. Irregularity of the heart's action develops in certain patients and increases, at times, without any appreciable cause. I have, for a long time, observed a patient who often presents an acceleration of the pulse to 108 or 112, without any assignable cause. Eulenburg (Berl. klin. Wschr., 28, 1868) has demonstrated, with the sphygmograph, the dicrotism of the pulse in ataxics, and attributes it to a diminution of the vascular tonus (of spinal origin). Periodical *gastralgia* and *vomiting*, with acceleration of the pulse, occur, in some cases, during the stage of irritation (crises gastriques of Charcot). They are accompanied by other symptoms of spinal irritation, especially by lancinating pains. These gastric crises may be attributed to irritation of the sensory fibres furnished by the pneumogastric to the stomach, rather than to hyperæsthesia of the solar plexus, whose sensibility has not been hitherto established.

Disorders of phonation are very rare in this disease. In one of my patients, paresis of one of the vocal cords was observed with the laryngoscope.

In another case the periodical difficulty in walking was accompanied by marked feebleness of the voice. The diminution in the tension of the vocal cords, in such cases, is probably due to disturbances in central innervation.

The *sense of taste* is often changed. The appetite is not infrequently lost, the tongue is thickly coated, and gas forms in the stomach and intestines, both at the beginning of the disease (with or without febrile symptoms) and in the later stages (nervous dyspepsia). In certain chronic forms of this dyspepsia, the sensibility of the buccal mucous membrane is diminished. All kinds of food taste insipid and doughy, and only markedly sweet or acid substances are perceived. In a case referred to above, the left half of the mouth and gums was markedly insensible to touch and to the action of an induced current. In two of Topinard's patients, the sense of taste was either enfeebled or lost.

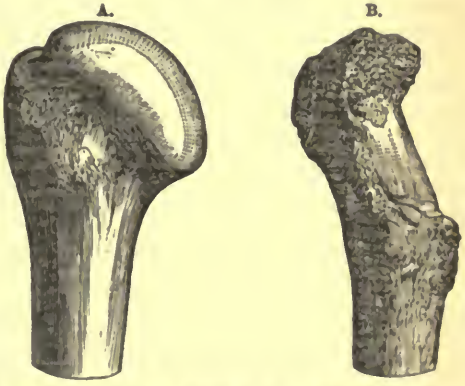

FIG. 14.—A, Upper extremity of a healthy humerus; B, of a humerus presenting the arthropathic lesions of ataxia.

Vaso-motor and Trophic Disturbances.—In addition to the burning sensations which the patients sometimes complain of in the legs or soles of the feet, and to the frequent and annoying subjective sensations of cold, attended by diminution of the perspiration and cutaneous secretions, desquamation of the skin, etc., I have also observed the appearance of reddish blue spots, which were very sensitive to pressure. To this category also belong the arthropathies described by Charcot, by whom they were observed five times in fifty cases. They usually belong to the initial symptoms of the disease, and, when they appear at a later period, are confined to the upper limb. The arthropathy develops without prodromata, the creaking and swelling of the joint being the first signs of the hydarthrosis. It usually occurs without pain or rise of temperature. The most frequently affected joints are the knee, shoulder, elbow, hips, wrist, and, more rarely, the smaller articulations. There are two varieties of arthropathies—a benign form, which terminates in recovery in several weeks or months, and a severe form, which causes lesions of the joint surfaces, dislocations, etc. This arthropathy differs from dry arthritis by the increase of the intra-articular exudation, by its manner of development, its sudden appearance, and frequent recovery. Charcot, Joffroy, Pierret, and Gombault have shown that the anatomical substratum consists in atrophy of the anterior horns (muscular atrophy often occurs in the affected limbs). In a recent case the atrophy of the anterior horns was wanting, but the spinal ganglia were enlarged and markedly changed.

The *general nutrition* may remain good for years. Usually, however, the size and firmness of the muscles diminish, although the subcutaneous adipose tissue appears well developed. Many of the patients have a peculiar, pale yellow color, especially upon certain days and after excitement, insomnia, etc.

The patients are subject, at the onset, to frequent erections and pollu-

tions. The sexual appetite is inordinately increased, but their ability to perform the sexual act does not equal their desire. The ejaculation of semen occurs prematurely, erection is often incomplete, and after coitus the patients are extremely prostrated. In the majority of cases the impotence progressively increases. Sometimes, however, complete or incomplete impotence heralds the onset of the disease, and is soon followed by neuralgias, diplopia, and rapid deterioration of motor power.

The remote *complications* of ataxia consist of *dementia paralytica*, and *melancholia* (with secondary lesions in the brain). *Progressive muscular atrophy* has been observed by Foucart, Duchenne, and Charcot, and has been attributed by the latter to lesions of the cells of the anterior horns. The symptoms of *paralysis, with or without contracture*, which sometimes occur in the last stages, are due to secondary degeneration of the posterior segment of the lateral columns.

Duchenne has divided this disease into three stages. The first stage comprises the ocular paralyses, degeneration of the optic nerves, and the characteristic shooting pains. The second period is characterized by loss of co-ordination, and of the muscular and cutaneous sensibility of the lower limbs (more rarely in the upper limbs). To the third period belongs the generalization of the disease. But since the ocular disturbances and cephalic symptoms do not exist in all forms of ataxia, and since even the pains may be wanting in certain cases, it appears preferable to distinguish only two stages in ataxia.

The first stage, or *stage of excitation* (with or without cephalic symptoms) is due to the development of the irritative process in the nerve centres, and is characterized by irritative phenomena of motion, sensation, and the vegetative functions. To this period belong the ocular paralyses with diplopia, the primary retinal changes, muscular spasms (usually of a reflex nature), the ready fatigue of the muscular power with integrity of isolated contractions of the muscles, neuralgias, and hyperæsthesias in the course of different nerves, febrile symptoms, phenomena of irritation of the genital organs, stomach, bladder, and rectum, arthropathies, etc. The second stage, or *stage of abolition of motion and sensation*, commences with the beginning of the degeneration, and comprises the manifest weakness in the harmonious action of the muscular system, frequently followed, if not accompanied, by diminution in the sensibility of the muscles, skin, and joints. It also includes the functional decay of the genital organs, the bladder, and rectum, and finally terminates in the more or less rapid suppression of all these functions, in partial paralyses, muscular atrophy, insanity, etc.

In many cases, however, it is impossible to adhere rigorously to this classification, since various symptoms of both stages may be present at the same time.

The Theory of Ataxia.

It has been established by a considerable number of observations that ataxia consists essentially in an affection of the posterior portion of the cord and that the sclerosis attacks, by preference, the posterior columns. If the process gains in extent and intensity, the adjacent posterior roots and the posterior gray horns will also become involved. The lesion often extends to the base of the brain and cranial nerves; it more rarely involves the anterior roots or horns. Experimentation has proven that the posterior columns serve only to a slight extent for the transmission of sensa-

tion, but that the latter occurs chiefly through the gray substance. On the other hand, section of the posterior columns produces considerable disorder in co-ordination of movements. *The centrifugal influence of the posterior roots upon the excitability of the anterior roots* (Harless and Cyon) has been disproven by the negative results obtained by Bezold, Uspensky, and G. Heidenhain. This is, therefore, a problematical phenomenon which cannot be utilized in the interpretation of disease, and especially of ataxia.

The rôle played by cutaneous sensibility in preserving equilibrium has been determined by Vierordt and Heyd (Tastsinn d. Fusssohle, etc., Tübingen, 1862) by anæsthetizing the soles of the feet with chloroform or ice, and these experiments have shown that the amplitude of the oscillations of the body is increased in these cases. In an ataxic, with incomplete insensibility of the soles of the feet, the local anæsthesia manifestly increases the uncertainty in the erect position and in walking with the eyes closed.

From these experiments, and from the fact that patients with myelitis themselves remark the anæsthesia of their limbs, while hysterical patients scarcely notice it, it follows that the loss of sensibility exercises some influence upon the awkwardness of movement observed in ataxia.

According to Leyden's observations (Ueber Muskelsinn in Ataxie, Virch. Arch., 47. Bd.), *the sensibility of the deeper parts (the muscles and articulations) is affected earlier and more profoundly than the cutaneous sensibility.* When the perception of movements is lost, together with the stability of the articulations of the foot or even of the knee, the patient finally loses all consciousness of the position of the limbs while walking. The preservation of equilibrium, the force, certainty, and harmony of action of the lower limbs, are, therefore, more and more compromised, and the patient must rely upon the sense of sight to supply the imperfections of movement.

If we attentively consider the mechanism of movements, from the first attempts of the child to the secure walk of more advanced age, we will find that our first efforts, which are entirely reflex, engrave the first motor impressions upon the cerebral cortex.

The frequent and regular succession of these phenomena occur in well-determined reflex paths. It is only at a later period that the cerebral activity, having arrived at maturity, intervenes directly in the production of movements, the latter obeying not only unconscious impulses, but also the conscious volition. The mechanism of these movements results from a continual succession of voluntary and involuntary muscular actions. The voluntary stimulus from the cerebral cortex passes to the anterior roots, through the fibres of the pes pedunculi, which have their central origin in the corpus striatum and lenticular nucleus. Reflex and unconscious impulses, which have their origin in the posterior roots and sensory nerves, pass to the optic thalamus and tubercula quadrigemina, and these transmit the stimuli to the anterior roots through the tegmentum pedunculi. Each movement occurs from the harmonious combination of contractions in certain groups of muscles, and the simultaneous innervation of these groups occurs by reflex means. These reflex acts are not only produced (as Cyon believes) by the sensitive muscular nerves which originate, according to Cl. Bernard, in the posterior roots, but also in other parts endowed with sensibility, like the skin and articulations. The degree of tension of the skin, the compression exercised by the muscles upon their nerve terminations, and the resistance of the articulations, give rise, in a reflex manner,

to corresponding synergic muscular actions. The great influence exerted by the posterior roots upon the motor activities is still further confirmed by the pathological facts observed by Charcot and Pierret. They have observed, in all their cases of ataxia, a degeneration of the external fibres of the posterior columns, which are continuous with the posterior roots and even with their prolongations into the posterior horns.

Duchenne has discovered that every movement of the trunk and limbs results from a double stimulation, which causes a simultaneous contraction in the muscles directly involved and also in their antagonists (or moderators). In walking, for example, when the trunk is carried horizontally forwards, it is necessary that the leg be elongated, and this is produced by extension of the knee and then of the ankle. In this manner the trunk may be carried forwards the whole length of the foot, which is raised from the ground. The elongation, which is still further necessary, is obtained by extension of the thigh; when the maximum elongation is reached, the leg is then raised by means of flexion of the knee, in order to move forwards without grazing the ground. During this series of movements of extension in the various segments of the lower limbs, it is necessary that the corresponding flexor muscles, as well as the external and internal rotators, should acquire greater tonicity and oppose a certain resistance, in the absence of which the movements would become sudden and convulsive.

Bishops has shown that, with the exception of the hand and forearm, the extensor muscles throughout the body are stronger than the flexors. We must therefore conclude that a sufficient equilibrium between the power of the different muscles is not maintained by the muscles themselves, but through the intervention of the nervous system.

Let us suppose that the apparatus of co-ordination is affected by an extended process in the nervous centres, such as occurs especially in ataxia. The disorders of innervation will then have a profound effect upon the manner of the production of movements. In the majority of cases it will be extremely difficult to differentiate the disorders of innervation from those of co-ordination. The different muscular groups may be unequally affected according as the various nerves are involved in the spinal disease. Usually, in ataxia, the extensors of the thigh and gluteal muscles (innervated by the sciatic nerve) suffer less than the flexors (psoas and iliacus), which are supplied by the lumbar nerves. On the other hand, the extensors of the knee (triceps femoralis), which are innervated by the crural nerve, are less affected than the flexors (biceps, semi-tendinosus, and semi-membranosus).

In proportion as the motor disturbances increase in ataxics, we sometimes observe that the patients do not carry their legs forwards, but that they throw them outwards in the arc of a circle before placing the foot upon the ground, and that the trunk consequently undergoes a very marked movement of extension. This depends upon excess of action in the extensors, with relative weakness of the flexors of the hip and extensors of the thigh.

The centres of co-ordination include the cerebellum and pons varolii; the part played by the tubercular quadrigemina in this function is less positively demonstrated. The cerebral hemispheres do not participate directly in the co-ordination of movements, but the centres of co-ordination, in order to become active, need a motor impulse originating in the cerebral ganglia. The posterior columns, by reason of the fibres of the posterior roots which enter into their composition, act merely as conductors for the

transmission of sensory impressions to the centres of co-ordination in the cerebellum and mesocephalon. Serious lesions of these conductors destroy the intermediate paths and, consequently, the essential unity of the apparatus of co-ordination. From the statements hitherto made, we must therefore conclude that progressive locomotor ataxia is a paralysis of co-ordination, a synergic paralysis of spinal origin.

Diagnosis.

The appearance of lancinating pains in the lower or upper limbs, appearing periodically and increasing after exertion or excitement should lead us to suspect the beginning of locomotor ataxia. Pains in the spinal column, and especially sciatic pains (with or without partial cutaneous hyperæsthesia), persistent upon one side and intermittent upon the other, are very significant. Intercurrent diplopia, marked inequality of the pupils, changes in the optic nerves recognizable with the ophthalmoscope, disturbances of vision and color-blindness, and the less frequent irritative phenomena on the part of the digestive and genito-urinary organs, are also very characteristic symptoms. Rapidly developing fatigue, especially in the vertical position; tremor and oscillations occurring when the patient attempts to stand upon one foot, with the eyes closed; phenomena of irritation on the part of the genital organs (increased sexual desire, frequent pollutions, incomplete erections, etc.); extreme sensibility to atmospheric changes; finally, the abnormal electrical excitability of the nerves, —all these symptoms, together with their manner of appearance and their characteristic relations, will usually enable us to detect the latent course of ataxia at an early period. In its more advanced stages, ataxia will hardly be overlooked by even the most careless observer.

With regard to differential diagnosis, we must especially take into consideration certain central affections whose symptoms are similar to those of ataxia.

Chronic myelitis is distinguished from ataxia by an evident diminution of motor power (varying from abolition of certain isolated movements to general paralysis); in walking, the patient drags the limbs slowly and with difficulty. There are no disorders of co-ordination or intermittent ocular paralyses. The different varieties of sensibility disappear rapidly, and galvanic excitability is more frequently diminished than in ataxia. *Chronic spinal meningitis* also presents the signs of a chronic myelitis, with the trophic disorders and electrical reactions resulting from local compression of the cord (vide p. 189).

In *general paralysis* we sometimes observe movements of projection of the limbs at an advanced stage of the disease. But general paralysis commences with tremor of the limbs, tongue, and lips, followed by embarrassment of speech, dullness of the senses, disorders of memory and judgment. Not infrequently certain filaments of the facial nerve are more or less paretic at the onset. On the other hand, ocular paralysis and amaurosis do not occur. If ataxia is complicated with insanity (a very rare occurrence), this will only develop after secondary affection of the cerebral cortex, and we must then rely, for diagnosis, upon the previous history of the patient.

The *differential diagnosis between ataxia and cerebellar affections* has been discussed upon p. 134. *Unilateral ataxia* is rare, and is readily distinguished, with a little care, from cerebral hemiplegia. We can recog-

nize the real nature of the hemiplegic symptoms by the coexistence of motor or sensory disturbances upon the other half of the body, by the characteristic neuralgia, and by other evident signs of ataxia.

Hysterical ataxia is characterized by rachialgia with intense hyperæsthesia, by the variable sensory disturbances in the lower limbs, by the abolition of electro-cutaneous and electro-muscular sensibility, the appearance of intermittent hysterical attacks, etc.

Ataxia following acute diseases (diphtheria, variola, typhoid fever) may be interpreted correctly by taking into consideration the previous symptoms, the simultaneous disorders of speech, and the absence of other signs of ataxia. In Ebstein's case (Arch. f. klin. Med., X. Bd.) sclerosis of the medulla oblongata and spinal cord was found in ataxia following typhoid fever.

Finally, in *cerebro-spinal sclerosis* we may also observe symptoms of ataxia and shooting pains, when the lesion extends to the posterior columns. But, in these cases, the initial cerebral symptoms, the embarrassment of speech, nystagmus, increased reflex excitability, and tremor during movements or after excitement, will prevent us from hesitating long concerning the diagnosis.

Prognosis.

The prognosis of ataxia is usually grave, but it is not desperate in all cases. It depends upon the violence of the initial symptoms of central irritation, upon their duration previous to treatment, and, finally, upon the habits of the patient. The more slowly the ataxia develops under the influence of known causes, the less threatening will be its further progress.

Therapeutic measures meet with the greatest success when directed against the initial stage of ataxia. But even in these cases, examples of complete and permanent recovery are exceedingly rare, and, hitherto, no case of cure has been as well authenticated as we might wish. Charcot and Vulpian have twice observed regeneration of nerve fibres in the cord, but this statement has not been confirmed. Even when periods of arrest occur in the progress of the symptoms, we should distrust the final triumph of therapeutic remedies.

Cases of ataxia traceable to hereditary influence, or in which an especial intensity is manifested by the rapid abolition of motion, of the genital functions and of the activity of the sphincters; those in which the disorders of motion and sensation rapidly increase or in which emaciation occurs after prolonged excitement or fatigue; those in which several cranial nerves, and especially the nerves of special sense, are involved, after a long period of characteristic neuralgia and motor disorders—all these cases present an unfavorable prognosis.

The paralysis of the ocular muscles, especially at the onset, diminishes with or without the aid of the electric current. In advanced cases persistent contractures sometimes develop. The disorders of co-ordination in the muscles presiding over deglutition and speech, and asthmatic difficulties, are rare but grave symptoms, the appropriate treatment of which (galvanization of the hypoglossal or phrenic nerves) may diminish their dangerous character.

The diminished power of sight presents an extremely gloomy prognosis. I have only seen one case of severe ataxic amblyopia in which hydrotherapeutic measures, continued for several months, produced such an improve-

ment that the patient could recognize surrounding objects with tolerable distinctness. A similar case is reported in Eisenmann's work, Obs. 65. But, in general, nothing will arrest the progress of the amaurosis (subcutaneous injections of strychnine, etc.). It is almost always superadded to other serious disturbances of motion and sensation.

The persistent neuralgias, the dulling of sensibility over a large surface, genital irritation (especially frequent pollutions), the rapid diminution of nutrition and early paralyis of the sphincters, render the prognosis much more grave. Local or general treatment, hydrotherapeutics, and electricity may relieve some of the annoying symptoms, but the effects of treatment will not prove permanent unless motion is palpably benefited. The diminution of vertigo, greater resistance to fatigue, and more sustained continuity of movements are important indications of improvement. We may sometimes succeed in keeping the ataxic in the same condition for several years (these cases are frequently reported as recoveries), but we will usually find that the progress of the disease presents an ebb and flow. This dire affection may exist for years, with very insignificant symptoms, when a fresh exposure, excesses, or mental excitement may suddenly precipitate its progress. In the severer forms, the ataxia pursues its course without being influenced in the least by any method of treatment.

Ataxia is generally a disease of long duration. Death does not occur in less than five years, except in those forms which run a rapid course. The largest mortality occurs at the end of the fifth or tenth years of the disease, and certain cases may last fifteen or twenty years and even longer (including the long period of the phenomena of irritation). The majority of ataxics die from pulmonary affections (tuberculosis, pleurisy, pneumonia), and a few from bronchitis, cystitis, suppurations, and bed-sores. Paralysis of the respiratory muscles, asthmatic disorders, and insanity are extremely rare causes of death.

Treatment.

We shall only consider a few of the remedies that have been recommended in this affection.

Iodide of potassium has been highly praised by Brown-Séquard and Duchenne in exudations into the spinal system. I have employed this drug faithfully in the beginning of ataxia, but without having obtained any appreciable effect.

Nitrate of silver has been highly extolled by Wunderlich, Charcot, and Vulpian, Herschell, Klinger, Duguet, and Vidal. It is most conveniently prescribed in pills (two to three decigr. (with some extract) to sixty pills, of which three to five are given daily). We may employ this remedy when the motor disturbances are very marked, until four to five grammes have been administered. The development of argyrism need not be apprehended before four grammes of nitrate of silver have been administered. In patients suffering from cardialgia and in those who complain, after the employment of this remedy, of a disagreeable sensation in the stomach, a metallic taste in the mouth, and diminution of appetite, treatment must be suspended for a certain length of time, and begun again, in smaller doses, after these symptoms have disappeared.

Charcot and Vulpian have observed the following symptoms after the employment of nitrate of silver: formication, slight convulsions throughout the whole body, and lichenoid eruptions with pruritis.

Among twenty cases treated with nitrate of silver, I have only seen one in which the motor power improved rapidly and to a marked extent. This treatment has little effect in inveterate cases. The remarks made concerning the nitrate of silver will also apply to the phosphide of silver.

Brown-Séquard has strongly recommended belladonna and ergot. I refer for further details to page 199, and will only remark that I have never seen any satisfactory results from their employment.

Bromide of potassium is most useful in those forms which are attended by increased reflex excitability, by nervous agitation, wandering neuralgias, muscular spasms, and sexual excitement. In these cases bromide of potassium will relieve the irritability of the spinal centres. We may prescribe two or three grammes daily, in pills or powder (in a little sugar-water). It is well tolerated by the majority of patients, but in some cases it produces redness of the pharynx, coryza, urinary hypersecretion from time to time, and diarrhœal evacuations. We must suspend the use of the bromide as soon as weakness of the limbs develops in consequence of its employment.

Phosphorus has been recently prescribed in ataxia by Dujardin-Beaumetz. He administers the drug dissolved in chloroform (1 gramme to 1000), in gelatine capsules, the dose varying from 1–10 milligrammes daily. I have frequently seen phosphorus produce disorders of digestion, but have observed no benefit from its administration.

Subcutaneous injections of morphine, and other preparations of opium, are useful as palliatives in ataxia dolorosa and in the severe neuralgic paroxysms of ordinary ataxia. The patients finally become accustomed to enormous doses without experiencing any inconvenience. The excessive pains should, nevertheless, be combated in these cases by the use of mineral waters, hydrotherapeutics, and the methodical application of electricity, since the neuralgias sometimes yield to one or the other of these remedies. But in some cases, fortunately comparatively rare, hypodermic injections are necessary to relieve the pains. I have obtained temporary good results, in purely nervous insomnia, from injections of morphine, chloral, and large doses of codeine and extract of thebaia (3–8 centigr. before going to bed).

As a rule, bleeding does not produce permanent benefit in ataxia. Even in the stage of congestion, it relieves the irritative symptoms to a very slight extent or not at all. Experience, also, indirectly confirms the uselessness of bleeding, since some patients suffer, from time to time, from an abundant hæmorrhoidal flux, without experiencing any appreciable improvement in the symptoms of irritation.

It has been the fashion, for a long time past, to send all ataxic patients to a warm mineral spring. Waters, possessing a temperature of 24°–28° C., are usually borne well by the patients, and the neuralgias, spasms, and distressing sensations of cold may disappear. In impressionable patients, mineral waters are frequently useful as a preparation for hydrotherapeutic treatment. But mineral waters with a high temperature are positively injurious, since they increase the condition of congestion and the sensibility of the patients to variations in temperature. If their use is prolonged, they increase the want of resistance on the part of the nervous system, and very frequently hasten the appearance of motor disturbances. Vapor baths are also contraindicated in ataxics. By their increased temperature, and the douche which is subsequently administered, they may, in the beginning of the affection, increase the condition of hyperæmia of the nervous system. It is well known, also, that, in the chronic forms, vapor baths have a debilitating action, and produce vertigo, nausea, and

exhaustion. Sea baths do not possess as many advantages as methodically applied hydrotherapeutic measures.

Hydrotherapeutics form one of the most efficient methods of treatment in ataxia. By methodically stimulating the vast expanse of sensory nerves, and by rendering the peripheral circulation and the cutaneous functions more active, hydropathic treatment allays the central irritation, strengthens the nervous system, and diminishes its great excitability and the dangers to which it is exposed on account of its sensibility to atmospheric changes (vide p. 206 for further details).

The first rule in the application of hydrotherapeutics to easily excited patients consists in the avoidance of all measures which are capable of increasing the subjective phenomena of congestion and irritation already existing. Everything depends upon a cautious and circumspect application of the method. The most useful measure appears to consist of friction with a cloth dipped in water possessing a temperature of 18° or 15° C. At the same time a cold compress may be applied to the head, and the patient is then placed in a bath of 24° to 20° C., into which cold water is slowly poured until the temperature is lowered to 18° or 16° C. The patient remains in this bath from four to eight minutes; he is then showered, and friction is applied to the back. After the entire procedure is complete, the patient should experience a sensation of comfort; after being thoroughly dried, he may take moderate exercise in the open air. We should not employ cold water, strong affusions to the head, douches, or wet packs, since they produce a permanent exciting effect upon impressionable patients.

Wet packs, continued until there is a gradual return of general warmth, especially to the legs, combined with cold applications to the head and followed by cool half-baths, may be employed in ataxia, but only when the patient suffers from spasms or distressing neuralgia. This measure is also employed in violent rachialgia, although we may also be compelled to employ frequently renewed cold compresses to the dorsal region or prolonged cool baths, local packing, and injections of morphine.

Hydrotherapeutic treatment may even be practised in winter, with the necessary precautions and in a moderately warm establishment. By persevering for a long time, we may restore the energy of the motor functions, cause the disappearance of the neuralgias, improve digestion, and, up to a certain point, the activity of the sphincters (by employing, for a short time, ascending douches to the perineum, and douches to the sacral and hypogastric regions).

The electrical treatment should consist, in the stage of irritation, of the application of constant currents, for a few minutes, through the vertebral column (preferably the ascending current). In the more advanced stages of the disease, the faradic current, which readily produces irritation on account of its tension, is less useful than the galvanic current (moderate interrupted currents, passing from the dorsal spine to the nerves or muscles, each seance lasting 3–5 minutes). We must avoid violent and painful contractions.

In ataxia of slow progress, electricity may have a favorable influence upon the motor and sensory disturbances. When the power of movement rapidly fails, the prudent application of the constant current may reinforce the motor functions, and the electrical brush sometimes causes improvement of the sensory disorders, although these measures do not arrest the progress of the disease. I have obtained marked improvement, in some cases, by combining electricity with hydrotherapeutics.

CHAPTER XXIII.

DISEASES OF THE LATERAL PORTIONS OF THE CORD.

The lateral portions of the spinal cord may present special forms of symmetrical sclerosis (gray degeneration). The lesion may also extend to the entire half of the cord, and may lead to what is known as spinal hemiplegia. It is only recently that the anatomical conditions and clinical forms of this group of spinal affections have been made the subject of exact investigations.

A.—Primary Sclerosis of the Lateral Columns.

We have previously discussed the secondary degenerations of the lateral columns which develop in localized lesions of the brain and cord (Tuerck). In dementia paralytica, the posterior segment of the lateral columns may also present an isolated lesion or combined with degeneration of the posterior column (formation of granular cells and enlargement of the interstices of the connective tissue, Westphal). Finally, the researches of Charcot (Arch. de physiol., Vol. II., 1869; Vol. IV., 1872; and Leçons sur

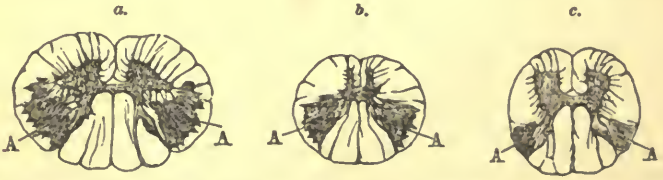

Fig. 15.—*a*. Transverse section through middle of cervical enlargement; *b*, middle of dorsal region; *c*, middle of lumbar enlargement. A, Plaque of primary lateral sclerosis.

les maladies du système nerveux, 5e fasc., 1874) have demonstrated, from an anatomical and clinical standpoint, the existence of primary sclerosis of the lateral columns. The origin of our knowledge concerning this affection may be traced to the observation published by Tuerck in 1856, of double degeneration of the lateral columns without cerebral lesion, and to the primary sclerosis of both lateral columns observed by Charcot, in 1865, in a case of permanent hysterical contracture of all the limbs.

The *anatomical characteristics* of primary lateral sclerosis appear to the naked eye as a gray, gelatinous degeneration of the affected column, and, under the microscope, as an interstitial hyperplasia of the connective tissue and corresponding atrophy of the nerve elements. The sclerosis affects, by preference, the posterior segment of the lateral columns. It extends from the inferior portion of the spinal cord into the pyramids and pons varolii, and may, in some cases, be traced into the pes pedunculi. With regard to the anatomical differentiation between secondary descend-

ing sclerosis in cerebral lesions and primary lateral sclerosis, Charcot has recently made the following distinctions (Progrès médic., 5, 1876): Secondary sclerosis, when it arises from a single foyer, only involves the lateral column of the opposite side, and never occupies more than a very circumscribed portion of the posterior lateral column; primary sclerosis, on the other hand, extends further, and is not clearly defined; it is more diffused, and may extend anteriorly to the external angle of the anterior horn, posteriorly to the posterior gray substance and, internally, to the bundle of nerve fibres (perhaps sensory) which forms the deeper portions of the lateral columns. Woroschiloff (Ber. d. k. Sachs. Akad. d. Wiss., Leipzig, 1874), in his later experiments, made isolated transverse incisions through certain portions of the spinal cord, and then determined, with the microscope, the lesion produced thereby. He found that a transverse incision of the posterior and anterior white columns (and of the entire gray substance), made upon a limited portion of the length of the cord, did not produce any appreciable disturbances in the transmission of motion and sensation. These columns do not, therefore, contain fibres which unite the brain to the spinal nerves, but merely short communications connecting the nerve fibres with one another. The real bond of union between the brain and the nerve roots is found in the lateral columns, the lateral column of each half of the cord containing motor and sensory fibres passing to two limbs. The lateral columns grow thicker from below upwards, since they receive new fibres from each nerve which they transmit to the brain. Henle (Handb. d. Nervenlehre, 1871, p. 70) states that he has only been able to recognize in the lateral columns fibres which originate in the gray substance and then bend upwards. This fact confirms the experimental conclusions of Woroschiloff, but further investigations are necessary in order to pronounce definitely upon the functional significance of the lateral columns in man.

The *symptomatology* presents many peculiarities, according to Charcot and to the observations recently published by Erb and O. Berger, although the latter were not verified anatomically. The disease commences with slight symptoms of paresis in the lower limbs, walking or prolonged standing becoming difficult. At a later period, *stiffness and want of suppleness develop in the legs*, with involuntary tonic movements of extension (more rarely, of flexion), which continue a short time, and are manifested chiefly in the knee. These phenomena of muscular tension are not very painful. Slight clonic spasms and tremor of the legs appear after exertion, excitement, or even after sudden passive movements. Patients affected with this paraparesis have stiff, very incompletely flexed knees, and take short steps, the legs being closely approximated and dragging upon the ground. When the eyes are closed, they present no symptoms of ataxia and do not stagger. The nutrition of the muscles which are affected with this paresis does not suffer to any appreciable extent, even after the disease has lasted several years. This also holds good with regard to their electrical contractility and to the excitability of the peripheral nerve trunks. It is only in the last stages that we observe slight emaciation and relaxation of the muscles. Sensibility is normal, the reflex excitability of the skin often increased, and the tendon-reflexes are almost always exaggerated (Erb, Berger). The functions of the brain and cranial nerves, and of the bladder, rectum, and genital organs remain intact during the entire duration of the disease. As a rule, bed-sores do not develop even when the patient is confined to his bed for several years.

At a more advanced stage, the paraplegic limbs become the seat of

permanent rigidity and of *immobile contractures*, which are subject, at intervals, to paroxysmal increase, attended with intense pain. The contracture usually fixes the legs in forced extension, the feet in equino-varus, the knees firmly pressed against one another by the contracture of the adductors; contracture of the flexors rarely occurs. Paresis and stiffness of the muscles of the back and abdomen are also observed in some cases. Permanent contracture of the upper limbs, with forced extension and application of the arm against the trunk, only occur after a very long duration of the disease and, exceptionally, in the first stages. The contracture of the upper limbs is generally less pronounced or barely noticeable.

Primary lateral sclerosis usually continues for a number of years. According to Charcot, it is not incurable even after it has reached the highest stage of its development. Erb has seen one case recover completely and several others markedly improved. Death is almost always due to intercurrent diseases.

We frequently recognize characteristics of primary symmetrical lateral sclerosis among the morbid forms classified under the general term of chronic myelitis. Paralyses associated with muscular tension and contracture indicate the participation of the lateral columns in the spinal lesion. These phenomena, however, become lost and disappear in a host of other symptoms and functional disturbances.

Primary lateral sclerosis is distinguished from *interstitial myelitis* and from *sclerosis of the posterior columns* by the slow progress of the paraparesis, by the muscular tension and early contracture, by the increase of the tendon-reflexes, often also of cutaneous reflex action, and finally by the absence of other spinal symptoms and of disturbances of co-ordination. In order to differentiate lateral sclerosis from *amyotrophic spinal affections*, we must be guided by the prolonged preservation of muscular nutrition and of the electrical reactions. In *peripheral paralysis* we do not observe a symmetrical development of the spasmodic and paretic symptoms, and, on the other hand, there are other positive signs, such as the combination with disturbances of sensation and nutrition, rapidly developing feebleness of the electro-muscular contractility, and loss of reflex excitability. By continuing our investigations and increasing the number of our observations, we will arrive at more positive conclusions with regard to this new disease.

The *treatment* coincides with what has been already formulated in the preceding chapters in regard to chronic diseases of the spinal cord. Electricity, hydrotherapeutics, and the use of mineral waters are the measures which furnish the best results. If advanced paraplegia exists with extended contractures, we cannot hope for improvement under any plan of treatment.

Symmetrical lateral sclerosis also includes a second variety, viz.: *amyotrophic lateral sclerosis*, whose anatomical and clinical history has been established by Charcot (Prog. méd., 23–29, 1874; Leçons, etc., 3e fasc.; Arch. de physiol., 1875, p. 739). The analysis of this new disease has been made by Charcot, from five personal observations combined with examination of the anatomical lesions, and from the analysis of other cases of progressive muscular atrophy and of bulbar paralysis, scattered throughout medical literature (Duménil, Barth, Leyden, L. Clarke, etc.).

The anatomical lesion of amyotrophic lateral sclerosis consists in a primary symmetrical sclerosis of the lateral columns, with a corresponding change in the anterior gray horns. The gray degeneration occupies

the posterior segment of the lateral columns, reaches its maximum in the cervical enlargement, diminishes progressively from above downwards, and, in the lumbar region, does not occupy more than the posterior fourth. In the medulla oblongata we can follow the lesion throughout the entire length of the pyramids, and from thence into the lower part of the pons and sometimes into the foot of the cerebral peduncle; the internal capsule is usually intact. In the anterior horns the sclerosis involves the neuroglia and the large motor nerve cells, as in progressive muscular atrophy. The cervical region is usually involved to the greatest extent. In the floor of the fourth ventricle, the cells in the nuclei of the spinal accessory, facial, and especially the hypoglossal nerves, are in a similar condition of degeneration. The anterior roots and peripheral nerves are merely atrophied. The trophic lesions of the muscular system are the same as in progressive muscular atrophy, but their inflammatory character appears more marked in amyotrophic lateral sclerosis, and the hyperplasia of the perimysium is more pronounced.

The *symptomatology* of amyotrophic lateral sclerosis bears the impress of the two medullary lesions, that of the lateral columns and that of the anterior horns. It is a combination of lateral sclerosis and progressive muscular atrophy. The course of the disease may be divided, according to Charcot, into three stages. The *first period* comprises the phenomena on the part of the upper-limbs. They present paralysis, atrophy, and intense fibrillary spasm with preservation of electrical contractility. Primary contracture soon appears in the paralyzed and atrophied limbs, as in the analogous forms of lateral sclerosis. These contractures are accompanied in the beginning by tremors. At a later period, when the entire arm is atrophied, the spasmodic rigidity, which sometimes extends to the muscles of the neck and jaw, disappears. Usually the upper limbs are successively involved, and, at the end of four to six months or a year, the alterations in the arms have attained a very marked extent. The disease, after remaining stationary for several months, then passes into the *second period.* The lower limbs then become involved in the motor paralysis, without affection of the bladder or rectum, without tendency to bed-sores, but with characteristic attacks of tonic and clonic spasms, terminating in permanent muscular rigidity, and with increase of the cutaneous and tendon reflexes. A very long time elapses before the rigidity diminishes in the lower limbs, giving place to fibrillary spasms and atrophy of the muscles. In the *third period* all the preceding symptoms are aggravated, and *bulbar symptoms* supervene, assuming the well-known characteristics of paralysis of the bulbar nuclei. Serious disturbances of circulation and respiration, from lesion of the nuclei of the pneumogastric nerves, terminate the sad spectacle of this disease.

The three stages which we have thus briefly sketched terminate in a comparatively short time. All the symptoms have usually made their appearance in six months or a year from the beginning of the disease, and death occurs in from two to three years. Charcot regards the hemiplegic forms and those which begin with bulbar symptoms as very rare anomalies.

Secondary lateral sclerosis, from localized lesions of the brain or cord, may also, according to Charcot and others, extend to the anterior horns, but only in very rare cases. Charcot mentions an observation in a woman, aet. seventy years, who had sudden left hemiplegia, soon followed by contractures, muscular atrophy, and diminution of electrical contractility. At the autopsy, a hæmorrhagic foyer was found in the centrum ovale of

the right hemisphere, with descending sclerosis of the left lateral columns and atrophy of the cells of the left anterior horn in the cervical and lumbar enlargements. In an analogous case, recently published by Pitres (Prog. méd., 8, 1876), an old hæmorrhagic foyer was found in the right lenticular nucleus, with destruction of a portion of the internal capsule, and descending sclerosis situated in the peduncle, pyramid, and left lateral column; the left anterior horn was sclerosed in the upper portion of the cervical enlargement. The atrophied muscles were emaciated, yellowish, and affected, in great part, by fibrous degeneration. Even secondary sclerosis, following medullary lesions en foyer, may produce muscular atrophy by extension of the lesion to the anterior horns.

With regard to *diagnosis*, the important point is to distinguish amyotrophic lateral sclerosis from progressive muscular atrophy, with which it has hitherto been confounded. Progressive muscular atrophy is characterized by its very slow course and its long duration. Even after the disease has lasted for several years, and the arms are very much atrophied, the lower limbs may be in a satisfactory condition. In idiopathic progressive muscular atrophy, the symptoms of bulbar paralysis are rare and exceptional. According to Duchenne's observations, it was found thirteen times in one hundred and eighty-nine cases of muscular atrophy. On the other hand, the course is rapid and the duration very short in amyotrophic lateral sclerosis. All the limbs are successively involved within a short period, and the lower limbs present characteristic changes, shortly after the beginning of the disease. The atrophy of the limbs is superadded to a previously existing paralysis, and this atrophic paralysis is soon accompanied by primary spasmodic rigidity of the muscles. Finally, Charcot mentions the following as a characteristic sign: partial affection of certain groups of muscles in progressive muscular atrophy, while in amyotrophic lateral sclerosis the muscles of the limbs are involved en masse. In the latter affection we also observe disorders of sensation, and tenderness of the muscles upon pressure and extension.

The *anterior spinal paralysis of adults* of Duchenne (subacute anterior poliomyelitis of Kussmaul) is differentiated from amyotrophic lateral sclerosis by the initial fever, sometimes attended with cerebral symptoms, by the rapid spread of the muscular atrophy, with weakness of the electrical reactions, by the absence of muscular tension and spasmodic deformities, by the paralysis which generally extends from the lower to the upper limbs, by the rarer, but always fatal, complication with bulbar symptoms, and finally by the rapid improvement of the legs, with longer persistence of the paralysis in the upper limbs. The anterior paralysis may recover or, at least, improve and its progress be arrested. We can readily distinguish amyotrophic lateral sclerosis from other forms of chronic medullary affections, if we pay attention to the symptoms and characteristic course of the disease. The action of cold and damp is regarded as a causative agent; in one case the affection was traumatic in its origin. The female sex has furnished the larger number of patients; their age varies from twenty-six to fifty years. From the facts hitherto obtained, the *prognosis* of amyotrophic lateral sclerosis must be regarded as absolutely unfavorable, since not a single case of recovery has been observed. All the cases terminated in death after a relatively short duration of the disease. The *treatment* consists of the methods in vogue in other spinal affections, but it has always proved entirely futile in this terrible disease.

B.—Diseases of one of the Lateral Halves of the Spinal Cord.

(With Crossed Hemiplegia and Hemianæsthesia.)

Among the advances made in recent times in the pathology of the spinal cord must be placed a disease, our knowledge of which has been due, in equal measure, to clinical observation and experimentation. I refer to lesions of one of the lateral halves of the cord. The affection had been recognized and described in its principal characteristics by some of the older authors, but these descriptions were deficient in the anatomical demonstration of the lesions and in the insight afforded by experimentation.

Cases of unilateral disorders of motion, with disturbances of sensation upon the opposite side, have been reported by Burserius, Morgagni, Ramazzini, Sénac, and Ollivier. A more important and characteristic case was published by Boyer (Traité des maladies chirurgicales, T. VII., p. 9).

Schilling's, Eigenbrodt's, and Koelliker's investigations upon the laws of spinal conduction have less reference to the disorders of sensation than to those of motion. The last two authors performed section of the cord in animals, and demonstrated the decussation of the anterior roots of the spinal nerves in the anterior commissure of the cord. They also proved by these experiments that a second decussation of the motor fibres occurs in the cord itself, independently of the decussation in the pyramids. Schiff and Brown-Séquard have observed, in their recent experiments, that in transverse section of the posterior columns the parts situated behind the incision present increased sensibility to stimulation. Weak stimuli, which pass unnoticed in healthy animals, provoked cries of pain in these experiments, and the animals endeavored to escape. After a certain length of time a manifest *diminution of excitability* follows this first stage of *increased sensibility*. Section of the cord, involving the posterior columns, does not abolish the transmission of sensation. The latter was preserved if a thin column of the central gray substance or a portion of the horns (especially the posterior) remained intact. The transmission of sensory excitation occurs, therefore, in the gray matter or æsthesodic substance of Schiff (although this tissue is itself insensible). According to Brown-Séquard's experiments (Journ. de l'anat et de la physiol., T. VI., 1863), the motor fibres, in man, only decussate in the pyramids, but *the decussation of the sensory fibres occurs along the whole length of the cord* and probably at a slight distance from the point of entrance of the posterior roots. Two groups of symptoms will appear after section of the lateral half of the cervical cord, including the antero-lateral and posterior columns and the gray substance. Upon the same side as the section, the phenomena are: first, paralysis of motion, muscular sensibility and of the vessels (with elevation of temperature); secondly, hyperæsthesia of the trunk and limbs to contact, pricking, electricity, and changes of temperature; thirdly, anæsthesia in a small zone between the upper limit of the hyperæsthesia and the healthy portions of the body; fourthly, symptoms of vaso-motor paralysis in the face and eyes (elevation of temperature, increased sensibility, slight contracture of some of the muscles of the face). *Upon the side of the body opposite to the section of the cord*, we find: first, preservation of motion and muscular sensibility; secondly, anæsthesia of the limbs and of nearly all the varieties of sensation.

According as the lesions involve a part or the whole of a lateral half of the cord, and according to their height and depth, the symptoms will present some variations, although always manifesting certain fundamental characteristics. In severe lesions of the cervical or lumbar regions, paralysis of motion occurs in one leg, and paralysis of sensation in the other, *i. e.*, hemiplegia with crossed hemianæsthesia. If the lesion does not penetrate so deeply as to involve the central organ of sensory conduction, motor paralysis of one side may alone develop.

The experimental investigations, which we have mentioned, throw considerable light upon the pathology of spinal lesions. If the affection involves the entire lateral half of the lower segment of the medulla oblongata (at the decussation in the pyramids), voluntary motion will be partially preserved upon both sides of the body, with complete abolition of sensation upon the side opposite to the lesion. If the morbid foyer occupies the entire thickness of the lateral half of the cord at a certain height, all parts of the body situated below the lesion, and upon the same side, will be deprived of voluntary motion, but not of sensation, while, on the opposite side, abolition of sensation will occur, with preservation of motion. We may also add that different portions of the two halves of the body may be affected with paralysis of sensation, even in unilateral affections of the cord. If, for example, the lesion is sufficiently extensive to involve, at the same time, the origins of the nerves which supply one limb or a portion of the trunk upon the same side, these nerves will lose their power of conduction as well as the sensory nerves of all portions of the body situated below the lesion and upon the opposite side. If anæsthesia limited to the same side follows a unilateral lesion of the cord, it is due to irritation of the posterior nerve roots.

Brown-Séquard reports twenty-four clinical observations in confirmation of the results of his experimental investigations upon the lesions of a lateral half of the cord. Other cases of a similar character have been since published by Radcliffe (Lancet, May 27, 1865), Bazire (Lancet, Vol. II., 5, 1865), by myself, by Charcot (Arch. de physiol., 1869, and 1873), Cantani (Il Morgagni, 1870), W. Mueller (Beitr. z. Anat. u. Physiol. des Rueckenmarks, Leipzig, 1870), Joffroy and Salmon (Gaz. méd., 1872), Riegel (Berl. klin. Wschr., 1873), and Troisier (Arch. de physiol., 1873).

Pathological Anatomy.—In Monod's case, published by Ollivier (incomplete motor paralysis of the right leg and complete insensibility of the left side, from the thorax to the toes), a hæmorrhagic extravasation was found in the lower portion of the right half of the cord; a brownish hæmorrhagic infiltration also extended to half of the gray substance, and the left horns and most external portions of the white substance alone remained intact. Oré has recorded two observations (Mem. de la Soc. de Biol., 1853); in one (paralysis of sensation on the right, and of motion on the left side), a blood clot was found in the left half of the cervical cord; in the other case (abolition of motion on the right, and of sensation on the left side), the cervical cord was compressed from right to left by a spongy excrescence growing from the dura mater. In the two cases reported by Charcot, the unilateral lesion was caused, in one instance, by an intra-meningeal spinal tumor, and, in the other, by an intra-medullary syphiloma. In W. Mueller's case, the cord was cut transversely below the third pair of dorsal nerves, from behind forwards, and from right to left (the point of a dagger was left in the wound). The left half of the cord was completely divided, and the surface of the section was swollen, of a reddish-brown color, and covered with pus; the meninges were adherent

to one another around this point. In Troisier's case (weakness of the left leg, incomplete anæsthesia of the right leg) the lesion consisted of sclerosis of the inferior half of the dorsal cord, especially on the left side. Upon the right side the lesion only occupied a few points in the posterior and lateral columns.

After this general review, I shall report two personal observations, which are rendered the more interesting because they have given rise to investigations into the neurology of the external integument.

A peasant, æt. twenty-seven years, stated that towards the end of Sept., 1866, while walking beside his horses and sowing seed, he was struck by a violent gust of wind. Shortly afterwards he experienced a burning sensation in the sole of the right foot, which, after several days, involved the right lower limb, and, a week later, the upper limb; this terminated in an insensibility to blows and pricks, which was accidentally discovered by the patient. At the end of about two months he experienced sensations of cold, formication, spasms of the extensors in the left lower limb, and in the thumb and index finger of the right hand. The patient lost the use of the left arm completely, and experienced lancinating pains in the left leg; he dragged this leg in walking.

When he entered the hospital, a year later, incomplete hemiplegia of the left side was noted with normal sensibility. The hand was very weak, the arm could not be raised above the horizontal, active extension, abduction and adduction of the hand were impossible, as well as flexion of the arm backwards. Active extension and flexion at the hip or knee were accomplished with slowness and evident difficulty. In walking, the left leg was stiff and dragged considerably. Sensibility, reflex excitability, and sensibility to temperature were normal. Electro-muscular contractility was markedly diminished in the extensor communis of the fingers, the extensors of the index-finger and thumb, the interossei, and the muscles of the ball of the thumb. The galvanic excitability of the nerve trunks, especially of the radial nerve, was very much diminished.

The right half of the body presented, on the other hand, anæsthesia without motor disturbances. With the exception of the anterior and posterior halves of the head and the lateral portion of the neck, the right upper and lower limbs and the lateral portion of the trunk, were insensible to pinching, pricking, and to the electrical brush. A space, about as large as three fingers, had preserved its sensibility in the lumbar region. The perineum and scrotum were also sensitive as far as the root of the penis. The patient was unable to distinguish hot and cold water upon the side of the body affected with anæsthesia and analgesia. When the right hand or foot was plunged into cold water, it produced a sensation of pain but none of temperature. Reflex excitability was abolished, but voluntary movements and muscular sensibility were retained. Strong faradic stimulation of the muscles of the forearm produced reflex contractions in the triceps, deltoid, and pectoralis major. The patient experienced a frequent and annoying sensation of flashes of heat in the anæsthetic portions.

The lesions of the sensory paths extended, therefore, from the fifth pair of cervical nerves downwards, with the exception of a small surface corresponding to the coccygeal nerves. The incomplete motor disturbances indicated a partial lesion of the left lateral half of the cord and of the motor fibres which decussate higher up. The well-defined symptoms in this case rendered the diagnosis positive. In the *hemiplegia of cerebral apoplexy* the cranial nerves are usually more or less involved. When anæsthesia and analgesia coexist (which is rare), they are always found upon the same side as the motor paralysis (for further details, vide p. 46). Severe lesions of the brain also produce, in such cases, disorders of consciousness and of the psychical faculties; electro-muscular contractility and sensibility, on the other hand, remain intact. *The unilateral form of ataxia is rare;* it presents the symptoms of spinal irritation for several years; the anæsthesia does not progress steadily until at an advanced stage, and is accompanied by weakness of movement and by disturbances of co-ordination. Generally analogous phenomena are observed upon the other side of the body. The *rheumatic hemiplegia*, described by Hoppe,

Romberg, and E. H. Weber (which I regard as a paralysis from exposure (Wiener Medicinalhalle, 1864)) is sometimes associated with diminution of sensation. But the latter only exists in the paretic or paralyzed limbs; muscular sensibility is very often abolished, and electrical excitability is slightly changed. In women, we must also exclude *hysterical hemiplegia*, which is distinguished, without reference to the other signs of hysteria, by the anæsthesia of the deeper parts, of the mucous membranes, and even of the special senses upon the same side, by the diminution or abolition of electro-cutaneous and electro-muscular sensibility, and by the integrity of the electrical reactions of the muscles.

We may finally add, with regard to treatment, that the patient was first given iodide of potassium (7 decigrammes daily); warm baths had a beneficial effect upon the condition of medullary irritation, but vapor baths and douches, on the other hand, were injurious. At a later period, strong descending currents were employed (from the vertebral column to the brachial plexus and nerves), alternating with the application of the electrical brush to the insensible parts. After seven weeks of treatment the power of motion was markedly improved, the movements being less circumscribed in the left limbs, while, upon the right side, the paralysis of sensation had made no appreciable progress towards recovery. In the tenth week it began to disappear from the centre to the periphery. When the patient left the hospital (at the end of the third month), the motor and sensory disturbances had disappeared. The exudation (?) into the left lateral half of the cord had been completely removed at this period.

After describing this form of spinal hemiplegia from exposure to cold, I will now publish the notes of a case in which the affection of a lateral half of the cord was due to traumatism.

An apprentice, aet. twenty-two years, was cut with a knife on the left side of the spinous processes of the third and fourth dorsal vertebræ. Almost at the same time he was felled, by a blow from a club, in the right lumbar region. *The power of motion immediately disappeared in the left leg, even the toes being paralyzed.* Soon after the patient noticed that the *right lower limb, in which motion was preserved*, was insensible. The left arm had also become heavy, but not, by any means, so stiff and unwieldy as the left leg.

Upon carefully examining the patient, a cicatrix was observed in the back, at the point already indicated, to the left of the dorsal spine (the wound had been received three days previously). There was paralysis of the left lower limb, and paresis of the left arm and hand. Sensation on the left half of the body was entirely normal; the electro-muscular contractility and galvanic excitability were markedly diminished. The right lower limbs retained perfect freedom of motion; sensibility to pressure was preserved, but sensibility to pain, temperature, and to the electrical brush had entirely disappeared; muscular sensibility was normal. The patient immediately began to stagger, when he stood with his feet together, the eyes being closed. Analgesia not only existed throughout the right lower limb, but also upon the anterior and posterior surfaces of the trunk. This analgesia extended upwards to the right loin, and upon the abdomen as far as the median line, below to the right groin, the right half of the scrotum and penis and to the right testicle (which was insensible to pressure and electricity); posteriorly it extended from the right half of the perineum to a line passing through the upper lumbar vertebræ. *The lesion was, therefore, situated in the vicinity of the lumbar enlargement.*

The limits of the analgesia, in these two cases, correspond exactly to the distribution and to the lines of demarcation of the cutaneous nerves (described by Voigt). The second case was under treatment about two months, but only incomplete recovery was obtained. The faradization of the paralyzed muscles contributed largely to the restoration of motor power, so that the patient could walk, with the aid of a cane, at the end

of five weeks, and, three weeks later, could walk freely, merely dragging the left leg slightly. But the analgesia had undergone no change, despite active treatment with the electrical brush.

An analysis of these cases and of those published by other authors will lead to very interesting conclusions, both from a physiological and pathological point of view.

The *motor paralysis* occurs upon the same side as the lesion, while sensibility is abolished upon the opposite side, because the sensory fibres decussate along the whole length of the cord, and the motor fibres high up. The motor paralysis may assume the form of an incomplete hemiplegia or chiefly involve one extremity (usually the lower limb). On the other hand, in Radcliffe's second observation, both arms were alone involved in the paralysis and anæsthesia. This incomplete paralysis proved that the lesion of the lateral half of the cord was only partial and situated high up towards the cervical region. The abolition of motion is usually preceded by the irritative symptoms so frequently observed in other spinal paralyses. These irritative phenomena consist of clonic or tonic spasms in the muscles of the limbs and in certain muscles of the fingers, or in contracture of the muscles of the neck and jaw. At a later period, exhaustion after motion, and tremor develop as precursors of the paresis or paralysis.

The *sensory disturbances* usually appear at the onset. They consist of sensations of burning or pricking, formication, cold, and heaviness. Neuralgia develops if the irritation of the sensory fibres becomes more intense. Hyperæsthesia then usually occurs, but lasts merely a short time, and soon gives place to anæsthesia, the various forms of sensibility being involved in different degrees. Not only the skin, but the tendons, ligaments, bones, muscles, and nerves, may entirely lose their sensibility to mechanical, thermic, and electrical irritation upon the side opposite to the section of the cord. In other cases the perception of temperature or of pain is alone abolished. Lancinating pains sometimes occur in the anæsthetic limbs (anæsthesia dolorosa). In my first patient, the reflex excitability to tickling and the sensibility to temperature had disappeared in the anæsthetic portions. The muscular sense was preserved in both cases, the patients being able to move the limbs freely without being aided by the sense of sight. They could distinguish changes in position, the different degrees of resistance opposed to them, and difference in weights with perfect accuracy. The similarity of these symptoms in the cases hitherto observed has led Brown-Séquard to believe that the nerves presiding over the muscular sense, and the vaso-motor nerves, follow the same course as the motor fibres. The unequal affection of the different varieties of sensation appears to indicate, according to Brown-Séquard, that special nerves exist in the cord for the transmission of the various forms of sensibility. This hypothesis is also corroborated by the fact that the different varieties of sensation may recover separately. When the lesion occupies the upper portions of the cord, *respiration is impeded and accelerated.*

In addition to the disorders of sensation and motion which we have described, we not infrequently observe inertia and paralysis of the vesical and anal sphincters, abolition of the sexual power, and oscillations and vertigo in the erect position when the legs are placed in apposition (Brach's symptom).

Electrical exploration of the paralyzed limbs gives different results, according to the greater or less intensity of the spinal lesion. In both of

our cases we observed diminution of the electro-muscular contractility and of the galvanic excitability of the nerves. In Joffroy and Salmon's case the electro-muscular contractility had disappeared at the end of six weeks, but was re-established under the influence of faradization.

The *reflex excitability*, both to electricity and to mechanical irritation, is usually increased in the beginning. At a later period this reflex sensibility becomes lost.

There is a very intimate relation between the extent of the anæsthetic regions and the mode of distribution of the cutaneous nerves, as Voigt has demonstrated by numerous and delicate preparations (from within outwards and inversely) (*vide* Denkschriften der k. k. Acad. d. Wiss., XXII. Bd., 1863). This relation existed very distinctly in two cases under our own observation. In two cases of paralysis following vertebral caries, which have been previously discussed, in chronic sclerosis of the cord, and in the hysterical paralyses, to which we shall refer hereafter, I have found anæsthesia of considerable extent limited by Voigt's lines, and the relation between these two facts appears to me to be perfectly well established. More extended investigations, carried on in this sense, will furnish clear indications concerning the changes in certain spinal columns, and concerning their anatomical and pathological relations, and from these considerations we will be able to draw conclusions concerning the situation of the lesion and its sequences.

The mosaic which is found in the central nervous system must necessarily correspond to the peripheral nervous expansions. We must first comprehend the law of these peripheral expansions, before undertaking, with any chance of success, the infinitely more difficult task of penetrating into the complex texture of the central nervous system.

Voigt has communicated to me another fact, which is interesting from the same point of view, viz. that in birds the various territories occupied by the feathers correspond to the lines of demarcation of the cutaneous nerve filaments; these facts are confirmed by the researches of His upon the same question.

It now remains for us to refer to the *vaso-motor and trophic* disturbances. In Joffroy-Salmon's and Mueller's cases contraction of the pupils was observed, with hyperæmia of the fundus of the eye and an elevation of temperature upon the same side as the lesion. Upon the insensible side of the body bed-sores formed after a few days, and disappeared in the first case, as soon as the condition of congestion of the eye was relieved. In the same patient the knee upon the paralyzed side was red, swollen, and painful when movements were communicated to it.

In Mueller's and Riegel's patients considerable muscular atrophy occurred, with diminution in the electrical excitability and lowering of the temperature. We know, from previous considerations, that these symptoms indicate an affection of the gray substance, and especially of its anterior horns.

The *prognosis* of spinal hemiplegia depends upon the nature of its causes. It is unfavorable in the case of tumors compressing a lateral half of the cord, in sclerosis, and in abundant hæmorrhages into the parenchyma of the cord. In unilateral hæmorrhage, especially when the patient has been previously healthy, we may hope for absorption of the extravasation and for improvement, if not complete disappearance of the paralysis.

Spinal hemiplegias of rheumatic origin may also terminate in complete recovery. The termination naturally depends upon the degree of intensity of the disease, upon its extent, and upon the character of the phe-

nomena. A moderate exudation or extravasation may recover almost entirely, but more severe hæmorrhages and solutions of continuity will give rise to disorders which do not disappear.

The *treatment* in the spinal lesions now under consideration should be directed towards increasing absorption, and rendering the circulation and innervation, in the parts affected with paralysis, more active. The iodide of potassium in moderate doses and the use of warm baths are indicated in the beginning. Vapor baths, cold douches, and very hot baths are not beneficial in this form of spinal irritation. In these cases English physicians prescribe sulphurous mineral waters. From what I have observed in closely allied spinal affections, they act like mineral waters in general. In Riegel's case, the annoying symptoms resulting from the increase of reflex action and of sensibility were successfully combated by subcutaneous injections of Fowler's solution.

The *electrical treatment* consists in the application of interrupted galvanic currents (produced by stroking the skin with the electrodes) from the nerve roots and plexuses to the nerve trunks of the paralyzed limbs. The descending currents should be preferred, but it is not necessary to produce strong contractions. In paralyses of sensation, the faradic brush should be applied to the dry skin. If the anæsthesia is very profound, the secondary current may be applied to the previously moistened skin. The application of the electric brush may be effected by means of the negative pole of a galvanic chain, by giving a descending direction to the interrupted current. The anode is preferably placed upon the trunks of the nerves which are distributed to the affected part. In persistent paralyses of motion, it is well to alternate the treatment of the nerves by the galvanic current with the treatment of the paralyzed muscles by the induced current.

INDEX.

INDEX.