PART VII
DISEASES OF THE NERVOUS SYSTEM

CHAPTER XXXIII

DISEASES OF THE MENINGES

GENERAL DISCUSSION

Both spinal and cerebral meninges are subject to infection. Tumors are rare and are usually of a benign nature. The bacterial invasion may be through direct extension around the roots of the cerebro-spinal nerves or the infectious agent may be carried by the blood; occasionally there may be direct invasion of the meninges from the rupture of an abscess or from injuries. Mastoid abscesses may rupture into the meninges. The direct extension from the nasal membrane upward around the roots of the olfactory nerves is certainly responsible for some meningeal infections. Meningeal tuberculosis is probably the result of blood-born infection as is the case with syphilis.

The blood vessels of the meninges are controlled by the vasomotor nerves from sympathetic ganglia; these, in turn, are controlled from the lateral horn cells of the spinal cord, and homologous centers in the medulla and the midbrain. The spinal meninges thus may have their circulation disturbed by bony lesions of the first thoracic to the third lumbar vertebræ, and the cerebral meninges may be affected by lesions of the upper thoracic vertebræ or by axis, atlas, or occiput lesions.

Gravity is a factor in causing meningeal congestion when the blood vessels have deficient tone, as may be the case in the presence of bony lesions, such as have been mentioned, or in the presence of exhausting illness, as typhoid, or when toxins affect the vascular walls, as in scurvy, influenza, and certain other acute or cachectic diseases. For this reason the supine position is to be avoided, during any serious or long-continued illness.

The pia and arachnoid are to be considered as one membrane, in disease; inflammation of these usually attacks the dura to some extent, and vice versa. Disease of the dura is associated with nerve-root symptoms more often and more intimately than is disease of the pia-arachnoid.

Meningeal diseases have usually a grave prognosis, and satisfactory treatment is difficult. Surgical interference is usually dubious, though it may give good results in selected cases. By

far the most important thing is the prophylaxis of meningeal invasion; this must be based upon the consideration of the etiological factors.

Epidemic cerebro-spinal meningitis is discussed with other acute infectious diseases.

PACHYMENINGITIS

Inflammation of the dura mater may affect chiefly either the extradural space (external pachymeningitis), or the subdural space (internal pachymeningitis). Either the cerebral or the spinal dura may be involved.

Cerebral External Pachymeningitis is due to trauma, middle ear disease, syphilis, or disease of the skull. The symptoms are often indefinite, but include constant, dull headache in nearly all cases. Chills and fever, drowsiness and stupor, rarely convulsions and paralysis, may suggest the diagnosis. Choked disk is present, and is due to the increased intracranial pressure. Symptoms of pyemia, with leucocytosis, may appear. The history of injury, previous middle ear disease, etc., may help in the diagnosis.

Cerebral Internal Pachymeningitis may follow the external form, or may not be associated with it. It is found in alcoholics, epileptics, the insane, and paralytic dements. Rarely it may appear in childhood, after acute infections. The condition is hemorrhagic; successive subdural hemorrhages occur, and these become organized, so that at autopsy a laminated false membrane may be found; this may or may not be stained with hemoglobin and its derivatives. Headache, convulsions, paralysis are the most common symptoms. Each hemorrhage may be associated with a fairly typical epileptic attack; the progress of events may be much slower than ordinary epileptic fits in some cases. Cortical epilepsy is frequent; this may lead to localizing symptoms, and to surgical relief. The paralysis may be upon the same side as the hemorrhage; this may be due to the pressure of the opposite side against the skull; or to flexion of the brain stem, thus causing pressure upon the pyramidal tracts of the opposite side. After organization of the clot, the paralysis is upon the opposite side of the body, as is to be expected from the anatomical relations.

Spinal External Pachymeningitis results from trauma; vertebral disease, either syphilitic, tubercular, or other; from the pressure of tumors, etc.; it is always secondary. The symptoms are mostly referable to involvement of the nerve roots.

Spinal Internal Pachymeningitis is due to alcoholism, syphilis, trauma, or extensions from vertebral disease. It is usually hemorrhagic, and is most frequent in the cervical enlargement (pachy-

meningitis cervicalis hypertrophica). First, involvement of the sensory nerves causes neuralgic and neuritic symptoms of the arms and shoulder girdle; paresthesias, pains, formication, and various reflex muscular symptoms appear. Later, paralysis of the hands, arms and shoulders appears; this is of the lower neuron type, and atrophy may be speedy. Third, the legs show spastic or upper neuron paralysis, due to the pressure upon the descending tracts. Death is from exhaustion, after months of illness, or earlier, from involvement of the phrenic centers.

Diagnosis. The symptoms may make the diagnosis evident. Spinal puncture and the examination of the cerebro-spinal fluid may show the etiological agent. Traumatic cases, some tumors, especially osteoma, and cases with increased intracranial pressure, may be recognized by the X-ray, especially in stereoscopic views. Blood examination shows leucocytosis in pyogenic cases; diminished eosinophiles in tubercular, and sometimes eosinophilia plus lymphocytosis in syphilitic cases. Wasserman's, Noguchi's and other biological tests for syphilis should be made.

Treatment. In traumatic cases, the removal of bits of bone or of thickened dural areas may give relief. Drainage of the cerebro-spinal fluid, several times repeated, is of value under certain conditions. Tumors may sometimes be removed.

Palliative measures may give much relief. Very gentle general spinal treatment gives relief which may last for a week or more. Counter-irritation, ice bags, mustard plasters, heat, may give temporary relief.

Hygienic conditions must be corrected. Alcohol, sexual indulgence, excessive meat diet, stimulating foods and drinks of all kinds, must be forbidden for a long time, even though symptoms abate markedly.

Prognosis. Complete recovery is not to be expected, except in early trauma, where the pressure can be removed before tissue degeneration has begun. Partial recovery is to be expected when the causative factor is amenable to treatment. Improvement may be hoped for with palliative measures. Death may occur at almost any time, but may be postponed for months or even years.

LEPTOMENINGITIS

Inflammation of the pia-arachnoid occurs as the result of an infection; rarely trauma gives entrance; usually the infectious agent is borne by the blood. The meningococci, the pneumococci, typhoid bacilli, tubercle bacilli, and the streptococci and staphylococci of various types are the most frequent. The disease may follow any of the acute infectious diseases, measles, mumps, diphtheria, influenza, or any others. Tubercular meningitis is

more common in children. The inflammation may involve the spinal membranes alone, the cerebral alone, or both. Of the cerebral, the basal area is more frequently involved, probably partly on account of gravity, and partly on account of the many nerve roots and blood vessels, which present so great areas of folding membranes to the action of the invading agents.

Cerebral Leptomeningitis of the convexity is characterized by headache, fever, stupor and delirium. Constipation, coated tongue, nausea, projectile vomiting, convulsions, are common symptoms. Photophobia is constant; hypersensitiveness to all sensory impressions amounts to severe pain upon any stimulation whatever.

Basilar Cerebral Leptomeningitis is characterized by the symptoms associated with inflammation of the convexity, and also by ptosis, pupillary changes, strabismus, facial spasm or paralysis, athetoid movements of the hands, especially, and the delirium is sometimes characterized by weeping, laughter, and apparent expressions of rage or other passion.

Spinal Leptomeningitis is characterized by marked pain in the back, with rigidity, opisthotonos and retraction of the head. Reflexes are first marked, then diminished or absent. Varying sensory disturbances are present, according to the early irritating, later paralyzing effects upon the sensory nerve trunks, roots, and ganglia. The spinal cord may be involved, with paralysis, bed sores, incontinence of urine and feces, and other symptoms of meningo-myelitis.

Serous Meningitis (wet brain; meningitis serosa) may be either acute or chronic. It involves the cerebral membrane almost exclusively. In its chronic form it gives symptoms of brain tumor. In this type there is excessive formation of cerebrospinal fluid. The condition is due to chronic alcoholism. The basilar membrane is usually involved, as well as the convexity, and the symptoms referable to the involvement of the cranial nerves are often severe.

Diagnosis. The history of an acute infectious disease, of chronic alcoholism, or the presence of the signs of tuberculosis or syphilis elsewhere in the body, with the symptoms as given, should suggest the diagnosis. The examination of the cerebrospinal fluid, obtained by means of lumbar puncture, should establish the nature of the disease. In serous meningitis this fluid is great in amount, escapes under pressure, and is clear. In infectious cases there is usually a rather small amount of a cloudy or flocculent fluid, which usually contains the infectious agent. Sometimes this is not to be found either on slides or in culture; injection into animals may give the diagnosis when other methods fail.

The blood examination shows marked leucocytosis in purulent cases; diminished eosinophiles in tubercular cases, and sometimes indications of other less common etiological factors.

Treatment. The treatment of all types of leptomeningitis follows closely after that given for acute infectious cerebrospinal meningitis. (q. v.) The prognosis is always grave. Recovery may occur, but usually with more or less permanent injury.

CHRONIC LEPTOMENINGITIS

This may follow the acute form, or may occur as a slow and chronic form from the beginning, as the result of alcoholism, syphilis, and possibly as the result of intense overstrain of the muscles of the back with exposure to extremes of heat and cold.

The symptoms are referable to involvement of the nerve roots, and include hyperesthesias, paresthesias, and anesthesias, affecting the sensations of heat, cold and pain, or of touch and muscle sense, variably and at different times. Motor symptoms include spasms and weakness, rarely paralysis. Herpes, slight and varying disturbances of bladder, rectum, and genital functions, and progressive loss of all powers until death from exhaustion or intercurrent disease are included in the ordinary history of the disease.

Treatment is limited to the correction of the causative factors, and such general spinal treatment as may be indicated on examination.

CHAPTER XXXIV

DISEASES AFFECTING BOTH BRAIN AND CORD

MULTIPLE SCLEROSIS

(Insular sclerosis; lacunar sclerosis; disseminated sclerosis)

Multiple sclerosis is one of the most common of organic nervous diseases. It is characterized by the formation of plaques of neurogliar overgrowth in many widely distant areas of the nervous system, and clinically by tremors, weakness, speech disturbances, emotional instability and visual disturbances, which appear and disappear suddenly or slowly, and which constantly progress to more serious symptoms, to helplessness and death.

Pathology. The plaques are usually slightly denser than the normal nerve tissue, and are of a pearly luster. They are composed of neuroglia cells, and usually surround a small blood vessel. Through them the naked axons pass, often fairly normal except for the loss of the fatty sheaths, and which seem to carry nerve impulses in a fairly normal manner. The nerve cells often retain their normal appearance, in the midst of the plaques. The pathogenesis is not known. It has been supposed that the degenerated fatty sheaths give the stimulus to the neuroglia which causes the overgrowth; the proximity of the vessels leads to the view that the disease is altogether circulatory in origin; it is supposed that congenital peculiarities of the neuroglia predispose to overgrowth, and that this is excited by toxic substances in the circulating blood.

Etiology. The sexes are probably about equally affected, though statistics disagree considerably. Rarely cases are reported before puberty; after that age they are frequent until after thirty years; after that age they are again very rarely found. The exanthemata, malaria, sunstroke, typhoid are all mentioned as causes; this incidence is little if any greater than might be expected from the laws of coincidence. Strains of various kinds are rather more frequent as possible etiological factors. A neuropathic ancestry is probably one factor. Metallic poisons, lead, mercury, and probably arsenic, are considered as causes. A history of fright, or some other profound emotional storm, is sometimes given as the cause of the disease; in such cases further investigation usually elucidates earlier symptoms; the emotional storm is usually one of the first recognizable symptoms, rather than a real cause of the disease.

Pseudosclerosis. Certain cases diagnosed as multiple sclerosis have come to autopsy, and no signs of sclerosis have been found; doubtless in some of these cases the patches were so small as to be overlooked. On the other hand, the possibility of functional imitation of the disease must not be forgotten.

Familial Sclerosis (aplasia axialis extra-corticocollis) is a very rare form of multiple sclerosis which appears rather constantly in certain families. It is sometimes directly hereditary.

Diagnosis. This rests chiefly upon the symptoms and history. The onset, in early adult life, is insidious. Usually the first symptom is a weakness in one leg; soon the other is affected, then the arms, and other muscles. Complete paralysis is rare, in the early stages, and the weakness may pass away for days or weeks at a time. Nystagmus appears early; it may not be noticeable except upon voluntary movements of the eye-balls. Retrobulbar optic nerve atrophy may be the first symptom. The retina often shows marginal pallor before the symptoms of the disease appear elsewhere. After a few weeks the intention tremor becomes marked; the speech becomes slow and drawling, and later scanning. Writing becomes difficult, on account of the tremor. Vertigo is common, rarely projectile or ordinary vomiting. Paresthesias are rather rare. Bladder symptoms are frequent and variable. Rectal and sexual functions are usually normal until late in the disease.

Mentality usually suffers. Emotional instability is constant; forced laughing and weeping are frequent. Less commonly dementia or mania is present.

The location and nature of the symptoms depend upon the location of the plaques. Sometimes involvement of the lateral funiculi gives an imitation of lateral sclerosis; in other cases implication of the posterior funiculi causes symptoms of tabes dorsalis. Occasionally the diagnosis is impossible. (Formes frustes.)

The disease resembles hysteria in many ways, and certain other organic nervous diseases may give difficulty in diagnosis.

Treatment. This can be only palliative. Rest, good hygiene, and such corrective work as may be indicated upon examination are helpful.

It must be remembered that these patients have prospect of remissions, followed by exacerbations, and that they are apt to live many years, unless some accident or intercurrent disease interferes. Hysteria certainly is often associated with sclerosis, and functional disorders are also frequent; treatment for the relief of symptoms may give gratifying, though probably temporary, results. Patients must be taught to make the best of their lives, and to understand that while serious symptoms may occur at any time, yet that these are not to be thought of as permanent; it is much better that they understand these things, rather than that they are taught not to expect further accidents. They should engage in such pursuits as are possible, and should live as happily and usefully as possible. Interest and good cheer go far toward pro-

moting general health, and toward preventing the functional disorders so often associated with the organic disease.

Especially when the prognosis is not frankly given, these patients go from one doctor to another, and from one patent medicine to another. Being erratic, in the very nature of things, they try everything that promises relief, without much judgment. "Nerve tonics" and purgatives seem especially attractive to them, and are, of course, either inert or harmful.

Prognosis. Recovery is not to be expected. Improvement is probable, and may last for months. Later symptoms may appear at any time. Life is probably not shortened by the disease; rarely it may involve cardiac or respiratory centers. Death usually occurs from some intercurrent affection, or some accident.

TABES DORSALIS

(Locomotor ataxia; posterior leucomyelitis)

This is a parasyphilitic disease, characterized by symptoms indicative of the degenerations of sensory neuron systems. These include lancinating pains in the legs, loss of the knee-jerk, Argyll-Robertson pupil, analgesia of the lateral surfaces of the legs with tactile hyperesthesia of the trunk, and other variable symptoms. The girdle sensation, visceral crises and ataxia due to the sensory disturbances are usually present. Lymphocytosis of the cerebrospinal fluid and increase of its globulins are significant; Wasserman and Noguchi are usually positive. The disease is characterized anatomically by degeneration of the fasciculus gracilis (the tract of Goll) and sometimes of the fasciculus cuneatus (tract of Burdach).

Etiology. Syphilis is the most important cause of the disease. Exposure to violent climatic changes, especially standing in cold water, heavy lifting, or violent exertion, and injury to the lumbar spinal column are sometimes concerned in the etiology. Atypical cases in which the eye symptoms are always absent and the gastrointestinal symptoms are usually wanting sometimes occur as the result of the factors just mentioned in the absence of syphilitic history.

Pathology. The constant pathological findings include the atrophy of the long sensory nerve fibers in the posterior funiculi of the cord. The posterior nerve roots and the cells of the sensory ganglia become atrophied later in the course of the disease. A diffuse pachymeningitis of the cerebral concavity is usually present and this may be responsible for the ocular symptoms. Other syphilitic evidences are usually present in typical cases of locomotor ataxia. The brain and the cardio-vascular system are usually affected.

Diagnosis. The symptoms are very typical, though there is much variation in the time of their onset. Sometimes the digestive, sometimes the ocular, sometimes the sensory, and sometimes

the motor symptoms may first appear. In the typical case the **gait** is first affected. The patient finds himself stumbling more frequently than usual, especially in the dark. He is unable to walk as well as usual, and it is noticed that the toes turn outward and that the foot drops when the forward step is taken; the legs are swung out in a semicircle in order to prevent the toes from scraping the ground. The ataxia becomes gradually more marked until the patient is unable to walk at all. The arms are rarely affected. The typical gait is almost pathognomonic. The **lightning pains** usually appear about the time of the ataxia. These are excruciatingly severe and come and go with lightning rapidity. It is very difficult to relieve this suffering; even moderate doses of morphine are often ineffective. The **girdle** sensation is a sense of constriction which may appear first around the legs, but which usually is noted first around the abdomen. The sensation follows the disturbance of the spinal nerves and in typical cases the girdle rises with the progressive degeneration of the sensory neurons. The **gastric crises** usually resemble severe attacks of acute gastritis. They may not be associated with any dietetic indiscretion, but sometimes appear to be precipitated by irregular meals, by alcohol or by emotional storms. Diarrhea with intestinal colic is sometimes present. More rarely the place of the gastric crisis is taken by crises involving the larynx, heart, vessels, or other viscera. The **Argyll-Robertson pupil** is present. This means that the pupils react normally to distance, but do not change in size with changes in the light. Pupils constantly dilated, constantly contracted, or of an oval or comma shaped outline are sometimes found. Vision is not disturbed, except as the result of retinal hemorrhages or optic nerve atrophy, or some other ocular lesion; these usually occur. Diplopia may be an early symptom.

Impotence and incontinence may occur early or late.

The tendon **reflexes** are first exaggerated, usually for only a very short time, then diminish and finally disappear. The loss of the sensations of heat, cold and pain during the later stages is associated with diminished nutrition of tissues affected. **Charcot's joint** usually affects the knee. This is a rarefying osteitis and arthritis. The knee may reach a size almost or quite equal to the waist of the patient. It is not usually painful but adds greatly to the difficulty of walking. A lax condition of the joints of the legs, especially of the hips, is present. It is not rare for a patient to be able to wrap his legs around his neck in much the same way that a normal individual could twist his arms around his neck. Injury to the feet is unnoticed, and burrowing abscesses may result from infections. The bones of the foot may be destroyed in this way with no pain to the patient. **Romberg's sign** consists in the patient's inability to stand alone with the eyes closed. It is present in other ataxias as well as in this. No characteristic blood or

urinary changes have been reported. The examination of the cerebrospinal fluid shows lymphocytosis. A positive reaction is given to the Wasserman test or any of the later modifications of this.

H. F. Goetz shows by spinograms a posterior lumbar spine in typical locomotor ataxia. Spinograms of syphilitics without locomotor ataxia do not show this spinal conformation. Hence, if all cases of locomotor ataxia have this posterior displacement of the lumbar vertebræ, then all cases of syphilis must be examined with the object in view of discovering whether they have posterior displacement of the lumbar vertebræ, and if so, this lumbar displacement must be corrected with the second object in view of preventing locomotor ataxia.

"The importance of this point is also apparent if the diagnosis of locomotor ataxia is made early, for then by correcting this displacement or disalignment of the lumbar vertebræ, we may not only prevent further advancement of the condition but also by reëstablishing the normal blood supply and nutrition, cure those cases in which no great havoc has been wrought. In other words:

"Removing this posterior disalignment of the lumbar vertebræ should act as a preventive or prophylaxis in locomotor ataxia."—H. F. Goetz.

Treatment. In very early cases, increased mobility of the dorso-lumbar spinal column, rest of the affected part and a hygienic manner of living usually result in stopping the course of the disease, and frequently in partial restoration of the loss of function. The bones are fragile; careless treating may result in fracture of the ribs or the bones of the legs or arms.

The older medical treatment with mercury and the iodides has been largely superseded by the present methods which are based upon the use of certain delicate arsenic compounds. If these are to be employed in any case they should be given by doctors who have made a special study of their administration.

The **educational** treatment is extremely important. The patient should be taught to perform first very simple movements and then gradually more complex movements until in many cases walking again becomes possible. This fact seems to be produced through the education of nerve centers or nerve paths not ordinarily functional under normal conditions and not injured by the syphilitic poison.

The **prognosis** for complete recovery is very serious. The prognosis for considerable improvement under the osteopathic treatment without drugs is very good. In untreated cases the disease may stop at any time and the condition of the patient remain stationary for many years. When no intermissions in the progress of the disease occur, death is to be expected within five to ten years after the occurrence of the first symptoms.

GENERAL PARALYTIC DEMENTIA

(General paresis; general paralysis of the insane)

This is a parasyphilitic disorder occurring in late middle life and characterized by successive attacks of paralysis associated with progressive dementia.

Etiology. It is probably always due to syphilis plus alcohol. Sexual excesses are also accessory etiological factors. The disease usually begins in the second or third decade after the occurrence of the primary lesion. Overwork, overworry, nervous strain and other mental injuries are frequently considered by the patient and his friends to be responsible for the disease. Investigation shows, however, that the actual importance of these factors is considerably overestimated.

Pathology. The pathological changes in the brain are very conspicuous. Thickenings, hemorrhages, and adhesions are found in the meninges and the cranium. In the brain itself are the evidences of syphilitic vascular disease associated with small foci of softening and neurogliar proliferation. Microscopic examination shows the cerebellar neurons undergoing various forms of degeneration and atrophy. Old and fresh hemorrhagic areas are scattered through the brain substance. Yellow pigment is abundant in the large nerve cells.

Diagnosis. The symptoms are very characteristic. At first the patient shows signs of what is ordinarily called a nervous breakdown. A superficial examination at this time gives a diagnosis of neurasthenia. Sometimes this period of nervous depression is preceded and sometimes it is followed by a period of marked exaltation. During the time of exaltation the patient is full of big plans for the future; he borrows money to extend his business; he buys many things on the installment plan; he begins work whose completion might require several lifetimes; he invents impossible machines; he sleeps little and considers himself fortunate in being able to devote more than the ordinary time to the pursuance of his new-found ambitions. He may suffer from exaltation in the sexual sphere; if he is a widower or bachelor, he is likely to marry a young girl, or he may cause grief to his family by his infatuations for young women. It is unfortunate that during this stage of exaltation the true nature of the condition is so rarely recognized, for it often happens that men introduce such absurd business enterprises that they jeopardize the futures of children and wife.

During the stage of nervous depression, melancholia may be marked. The character becomes suspicious, irritable and careless; the patient may show apparently more than normal ingenuity in devising methods of circumventing the members of his family; emotional irritability becomes more and more marked; he laughs and cries easily upon slight or no provocation; convulsive attacks resembling epilepsy are likely to occur; slight cerebral hemorrhages precipitate paralysis, which is more likely to affect the right side of the body, and which usually involves the speech centers; one attack of paralysis follows another until finally the whole body is involved. The dementia is progressive; the patient gradually losing interest in himself, or his surroundings, and finally becoming mindless and completely paralyzed. He may live in this pitiable condition for a number of months until paralysis involving the cardiac or respiratory centers brings a welcome death.

In the early stages, that is, during the period of exaltation or the period of nervous depression, diagnosis may be doubtful. It should be a matter of ordinary routine to make a Wassermann or other biological test for syphilis in every case in which apparently causeless neurasthenia occurs in men in middle life. Pupillary changes are usually present. The Argyll-Robertson pupil, the oval pupil, or inequality in the two pupils, are all important factors in an early diagnosis. The absence of these findings has no significance, but when they are present syphilitic history should be strongly suspected.

Treatment. Since the symptoms of the disease are due to actual nervous degeneration and since serious structural perversions precede any symptoms, it is evident that treatment is commonly of very little value after the diagnosis is possible.

Prophylaxis is important. The prevention of syphilis is the prevention of paretic dementia. Men who have had syphilis may avoid this form of insanity by living continent and temperate lives.

The **prognosis** is hopeless unless an early diagnosis is made. The course of the disease is sometimes halted for some months or years, but its further progress to death is inevitable.

TABO-PARALYSIS

There are certain cases of parasyphilitic disease in which the spinal degeneration resembles that of locomotor ataxia, and the cerebral degeneration resembles that of paretic dementia. To these cases the term "tabo-paralysis" has been applied. The mental derangement is less pronounced than in the ordinary case of paralytic dementia; the pupillary changes are early and marked; the ataxia is variably marked but never absent; the occurrence of epileptoid attacks is rather rare. Speech is less frequently an early symptom, and the paralysis is oftener a weakness with incoördination than a true paralysis. The disease is probably to be considered an intermediate type between locomotor ataxia and paretic dementia, rather than a combination of the two diseases. The treatment and pathology are practically the same as in paretic dementia. The prognosis is somewhat different; the progress of tabo-paralysis is more constant, and less subject to remissions, than is either paretic dementia or locomotor ataxia.

DISSEMINATED MYELITIS

In this form there are many patchy areas of inflammation in the cord, medulla, and brain. The disease is due to almost any of the ordinary infectious agents, and may follow any fever. The symptoms are those of acute myelitis, plus the symptoms of bulbar and brain involvement. The ocular disturbances include variations in the size of the pupils, various incoördinations of the extrinsic eye muscles, and visual disturbances. Mentality may be variously affected according to the area involved. Coma and delirium may

precede death. The bulbar symptoms may be immediately followed by death, or may be limited to the centers of the cranial nerves; twitchings and paralysis of the face, paresthesias of sight, taste, smell and hearing may occur.

Treatment is almost useless, and death is apt to occur at any time within a week or so after diagnosis becomes possible.

BONY LESIONS AS LOCALIZING FACTORS

In diseases affecting both brain and cord, it is very evident that some localizing factors are present. Different patients present different symptoms, according to the location of the lesions in each case, but what factors determine the location of the lesions in any case is not yet to be determined exactly.

Vasomotor nerves have been demonstrated for the brain and for the meninges of both brain and cord. Vasomotor nerves for the cord itself have not yet been certainly demonstrated but their existence seems fairly certain. Bony lesions are certainly not less efficient in modifying spinal and cerebral circulation than in modifying renal and pulmonary circulation. Thus, lesions affecting any segment of the spinal cord must be considered efficient in localizing general disease in that, and neighboring, segments. Lesions of the occiput and the cervical vertebræ are efficient in modifying the cerebral circulation, and thus in localizing the effects of disease in the brain. The localization of vasomotor control within the brain has not yet been completed.

The functional activities of nerve centers depend chiefly upon the nerve impulses reaching them. Bony lesions which limit the mobility of any joint lessen the normal stimulation to the related centers. Bony lesions which are irritating send abnormal impulses into the related centers, and thus these have undue stimulation. When a lesion associated with limited mobility is associated with increased mobility above and below the affected joint, or when it is associated with marked hypersensitiveness, the disturbance of the function of the related nerve centers may be profound.

All these factors, disturbed circulation through the nerve centers, lessened stimulation, and irritation, are concerned in lessening the resistance of the nerve centers to infection and to the influence of poisons, and thus are localizing factors in diseases affecting the central nervous system in a somewhat general manner.

CHAPTER XXXV

DISEASES OF THE SPINAL CORD

GENERAL DISCUSSION

The diseases which affect the spinal cord itself are characterized by various motor, sensory and trophic disturbances varying as the injury destroys the anterior or posterior gray matter, the white matter or the nerve roots or the spinal ganglia.

Pathology. Spinal cord diseases are characterized by pathological findings which vary from those practically negligible in the functional disease to absolute destruction of the cord or of some area in it as in syringo-myelia or myelomalacia.

In the gray matter the nerve cells may show merely a slow progressive diminution in size which goes on to complete destruction as in chronic anterior poliomyelitis or they may show various degenerated types with chromatolysis, extrusion of nuclei, vacuolization and swelling of nucleus and protoplasm, fatty degeneration, pigmentation and other less easily recognized changes. The neuroglia may be unaffected; may increase in amount either by multiplication of the nuclei with cellular division or without (this leading to a syncytium-like appearance), or the fibers may increase, forming a dense felt-like tissue in the injured spinal matter. The blood vessels may be uninjured or they may show the effects of ischemia, hyperemia, congestion, or inflammation in varying degrees. In the spinal diseases due to syphilis changes in the walls of the blood vessels are usually to be found. In myelitis vascular changes are probably an important factor in the determination of the characteristics of the disease. The walls of the blood vessels may show arterio-sclerotic changes; the intima may show thickening which may go on to the point of total occlusion. When this process occurs inside the spinal cord itself the death and degeneration of the infarct area are inevitable since there are no anastomoses within the cord. In the meninges similar conditions are usually associated with chronic pachymeningitis of varying degrees. The wealth of anastomotic relations in the meninges maintains a fairly good arterial supply even when rather large vessels are occluded.

Etiology. The causes of disease of the spinal cord are extremely varied and numerous. Of all these, however, the infections hold first place and of all the infections syphilis either directly or indirectly is a factor of paramount importance. Acute anterior poliomyelitis or infantile paralysis is the next most important of the spinal diseases due to specific infection. The organisms responsible for many of the ordinary infectious diseases, such as typhoid fever, pneumonia, scarlet fever, measles, as well as those found in pyemia may gain entrance into the spinal cord itself and there set up extremely rapid and usually fatal spinal inflammations. Inflammatory processes in the meninges may extend into the spinal cord and this condition also is usually very rapid and fatal.

Toxic influences of all kinds may be considered in connection with diseases of the spinal cord. Alcoholism goes with syphilis

as an etiological factor in spinal diseases; as indeed these belong together in most discussions of personal and social pathology. Lead, mercury, and other inorganic salts sometimes exert serious influence upon the spinal cord. The diminished use of calomel as a drug is removing one cause of spinal disease. The poisons, whatever they may be, that are responsible for pernicious anemia, insular sclerosis and a few other diseases of doubtful etiology bring about the disease of islands of the nerve tissue with symptoms localized according to the area affected. Spinal diseases due to this factor are usually easily recognized by the history. Whether concussions, jars or blows can be considered important in the cause of the spinal disease in any given case is not always easily determined. Certainly the occurrence of injuries to the spinal column does seem to localize and often to predispose to spinal disease when other conditions are favorable to the development of a pathological condition.

The place of the bony lesion as an etiological factor in spinal cord disease has not yet been positively determined. There is very good reason for believing that bony lesions exert a detrimental influence upon the circulation in the meninges and at least indirectly upon the circulation in the segments of the cord. Though the presence of vasomotor nerves within the spinal cord itself has not been demonstrated, the fact remains that spinal diseases appear to affect first those areas of the spinal cord which send sensory nerve fibers to subluxated vertebræ and ribs. It must not be forgotten in this connection that resistance to infection generally, elimination of poisons, and the nutrition of the body are all subject to profound variations as the result of slight malpositions of the bones and ligaments and the abnormal muscular tension so often associated with these.

Perhaps there is no one factor much more responsible for spinal diseases than inheritance. Except for infantile paralysis, most of the spinal diseases in children are due to inheritance. This inheritance follows Mendel's law so that such diseases are more frequently referred to as familial than hereditary. Many of the diseases whose chief cause is infectious or toxic have bad inheritance as a predisposing factor. In many of the diseases associated with syphilis, alcoholism, sexual excesses and so on, it is difficult to determine the relative proportion of blame due to these things in themselves, to the inheritance of nervous weakness or to the fact that the different members of the same family are usually educated to the same habits and the same use of life.

Diagnosis. Some general factors in diagnosis may be given here. Lesions of the anterior gray matter alone bring atrophy and paralysis of the skeletal muscles innervated from the area affected. In such a case there are no sensory disturbances except that the

weakened muscles may sometimes ache or feel sore. Such muscles undergo atrophy with varying degrees of rapidity. There is present a reaction of degeneration and loss of reflexes; there is loss of muscular tone so that in early stages the muscle is very flaccid and soft. Later the overgrowth of connective tissue usually associated with the atrophy of the muscles, makes them hard and dense and the contraction of these connective tissues together with the unbalanced action of antagonists leads to various deformities.

Disease of the posterior gray matter is usually associated with more or less destruction of the anterior gray matter. In such diseases sensory disturbances are likely to be very profound. Paresthesias include the sense of formication, tinglings, pain, sensations of heat and cold and the girdle or stocking or glove sensations. Total loss of sensation in some area is usually present. Anesthesias, analgesias, lack of temperature sense or of the sense of muscular effort may appear in varying degrees according as the injury is or is not strictly localized in the gray matter. It must be remembered that the sensations of heat, cold and pain are carried chiefly by way of the nerve cells in the posterior gray matter, while the sensations of muscular effort and touch are carried chiefly by the long white tracts in the posterior funiculi. To a certain extent, however, it is probable that each of these pathways includes at least some of all the sensations named so that any destruction of the gray matter of the cord is associated with diminution of all somatic and visceral sensations.

Diseases of the white matter of the cord are mostly limited to those of the long tracts so far as our present methods of diagnosis are concerned. The most important of these on the sensory side is locomotor ataxia in which the long fibers of the fasciculus gracilis and to a less extent those of the fasciculus cuneatus are involved. On the motor side destruction of the lateral and the anterior descending cerebrospinal or pyramidal tracts leads to the symptoms of lateral sclerosis.

In those diseases in which heredity or a congenital condition is responsible for the disease the blood cells usually show the presence of many immature and atavistic types. In the infectious diseases the blood shows the same characteristics that are present in the same or similar infections occurring in other parts of the body. In many cases of spinal disease of doubtful diagnosis the blood examination will make this real condition clear.

It often happens that cases of pernicious anemia show their first symptoms as paralysis or atrophy referable to the spinal cord lesion. Under such conditions the blood examination may throw much light upon the condition and it should be made in every case in which there is the least doubt of the diagnosis. The blood-pressure is usually very high in senile and syphilitic diseases.

The urine often shows no changes whatever. When there is any marked destruction of nerve matter phosphorus may be increased beyond the amount expected from the patient's diet. In making this test it is best to put the patient upon an almost phosphorus-free diet, for a few days before the 24-hour sample of urine is collected. A good test for this is to have the patient begin his phosphorus-free diet with a few charcoal tablets. Then when the black color due to the charcoal has ceased to appear in the feces, collection of the urine may be begun. In diseases with marked degeneration of the nerve tissue or with abscess formation an excess of indican is present. In diseases associated with trophic symptoms the kidneys may be seriously involved either directly or as the result of the harm due to the elimination of the products of the abnormal metabolism elsewhere in the body. When the bladder is involved the urine may show the effects of this condition. When catheterization is necessary, the bladder is very likely to become infected and the urine then shows the results of the cystitis so produced.

At present the X-ray is limited to the recognition of the diseases of the vertebræ as these may affect the spinal cord. Tumors and bone diseases may be recognized by the X-ray and since such conditions cause symptoms which are atypical, the X-ray should be used in all cases of doubtful diagnosis.

Treatment. In many cases of disease of the spinal cord there is very little efficient treatment. Nearly everything in the way of drugs has received at least one voice of commendation as to its use in spinal cord diseases and several times as many voices of condemnation for its use in these same diseases. The diagnosis usually gives the indications for treatment. During the acute stage of any of the infections the treatment must include suitable measures for reducing the fever, such as are used in fevers in general. The patient should not be permitted to lie upon his back but must be kept upon the side or in the left lateral or the right lateral position. The prone position is good, except for the difficulty of getting the head in a comfortable place. In most cases an extremely gentle relaxation of the spinal muscles is usually very grateful to the patient and should be repeated from one to several times each day. Ice packs are sometimes of value. Massage of the arms and legs is sometimes good. Usually no foods except fruit juices are given during the acute stage. In the chronic diseases the treatment varies according to the condition. Contractures of the limbs and deformities of various kinds are best treated by suitable orthopedic surgery. Massage of the affected muscles and very mild faradization are sometimes helpful in the treatment of the muscles paralyzed. In diseases associated with incoördination of the muscles but no true paralysis or where the

paralysis is of the upper neuron type exercises looking toward re-education are of considerable value. This is especially true of the diseases of the long sensory tracts such as locomotor ataxia.

In both acute and chronic conditions the osteopathic measures to be employed depends entirely upon the indications as these are interpreted by the experience and good judgment of the practitioner.

The sensory disturbances are very hard to deal with. Generally ice packs give more relief than do applications of heat. Alternations of heat and cold may give relief when neither alone is efficient. Usually in the girdle sensation massage is grateful. Sometimes a rather tight bandage placed over the girdle relieves the discomfort. The same thing is true of painting the skin with celloidin. Great care must be employed to prevent the skin from being injured in any of these measures. The danger of bed sores and of serious infection from slight abrasions of the skin must be kept in mind. Careful nursing is the best thing in these conditions. An important factor in dealing with the disturbed sensations is the education of the patient. He must find interest in life and must thus be made to forget as far as possible the things that are so annoying and uncomfortable. The occurrence of these diseases which are so often hopeless so far as recovery is concerned is in itself a dreadful thing and the patient must be encouraged to find such occupations and interests as to get the most good out of such a life as will be possible to him.

The **prognosis** varies according to the area and amount of the spinal tissue destroyed and to a less extent upon the possibility of securing compensatory development of other nerve centers and tracts. The amount of coöperation which the patient is willing to give is sometimes very important. Under suitable cases nerve surgery gives a good prognosis. The prevention of the spinal cord diseases lies in the prevention of the causes. Stringent isolation of infantile paralysis and other contagious diseases is important. Perhaps the greatest thing of all is the prevention of alcoholism and syphilis. Those diseases of the spinal cord which come on during middle age or later should be lessened with the diminished use of alcohol and drugs. A large preventive field lies in keeping the structure thoroughly adjusted.

HEMORRHAGE OF THE SPINAL CORD

(Meningeal apoplexy; hematorrachis; hematomyelia; spinal apoplexy)

Hemorrhage into the spinal membranes occurs from rupture of an aneurysm into the extrameningeal space, or from erosion of an artery by malignant neoplasms, caries of vertebræ, or as the result of hemorrhagic diseases, hemophilia, scurvy, and others.

Hemorrhage into the cord itself is usually due to trauma, or to rupture of small vessels which have become atheromatous or otherwise diseased; it may occur in the hemorrhagic diseases.

In either case, the symptoms depend upon the extent and the area of the hemorrhage. The onset may be extremely sudden (apoplectic), with paralysis and various sensory disturbances; or the injury may be so slight as to make diagnosis impossible. The extradural and the subdural spaces may contain a large amount of blood without any particular difficulty or pressure. The symptoms, if any, are due to pressure upon the nerve roots. Hemorrhage into the subpial space or the substance of the cord produce serious symptoms, usually immediate and serious, unless the amount of hemorrhage is extremely minute.

Death usually results within a few hours; if this does not occur, the later symptoms, the treatment and the prognosis are those of myelitis.

MYELITIS

This term is applied to any inflammatory disease of the spinal cord. Several types of the disease are recognized.

Meningomyelitis is an inflammation involving both meninges and spinal nervous matter; it is generally considered syphilitic.

Poliomyelitis is an inflammation of the gray matter, and may be either anterior, as in infantile paralysis, or posterior, as in certain forms of acute myelitis.

Leucomyelitis affects the white matter; it may be posterior, as in tabes dorsalis, lateral, as in amyotrophic muscular atrophy, or may affect any area, as in pernicious anemia.

Transverse Myelitis involves almost or quite the entire cord, for one or several segments.

Disseminated Myelitis is characterized by its widespread patches of inflammatory foci.

Myelomalacia is probably due to occlusion of an end artery; the area affected undergoes softening, resolution, and, later, absorption. Neurogliar growth fills the area with scar-like tissue, if life persists after the acute process is complete.

Pathology. The nerve cells of the affected area show chromatolysis, vacuolization, swelling of the protoplasm; the nuclei are eccentric or extruded, vacuolated, and present variations in staining. The neuroglia cells may be degenerated, or may show signs of rapid multiplication; the walls of the blood vessels may be almost or quite normal, or they may show inflammatory changes —a proliferative endarteritis is especially frequent, and this is an important cause of the softening found in myelomalacia.

The axons are swollen and bubble-like; granular degeneration is everywhere found.

Etiology. The causes of the different forms are slightly variable; the treatment and prognosis must also be considered for each form.

ACUTE INFECTIOUS MYELITIS

This is an acute infectious disease of the spinal cord, characterized by symptoms referable to the nature of the infectious agent and to the area of the nerve tissue destroyed.

Etiology. Any of the ordinary infectious diseases may affect the cord, though rarely does this occur. It may be difficult to isolate the infectious agent from the lesions, though injection into animals usually gives positive results. The infection may be carried by the blood or the lymph vessels, or may affect the cord through extension, especially in Pott's disease. Whitlow, carbuncle, parturition, may initiate the disease.

Diagnosis. This rests partly upon the symptoms; partly upon the history of the presence of some infectious disease. In tubercular cases the onset may be somewhat gradual, with progressive sensory and motor symptoms, leading, usually, to death within a week or two. In cases due to the ordinary acute infections, the onset is rather sudden, with increased pyrexia, vomiting and nausea —rarely projectile vomiting—and very severe burning pain in the back. Within a few hours flaccid paralysis in the muscles innervated from the segments of the cord affected, with variable sensory symptoms, makes its appearance. Usually the legs and lumbar centers are first affected; bladder and rectal symptoms are serious from the beginning, and the disease progresses rapidly upward until involvement of the respiratory muscles terminates life. When the disease affects other segments of the cord, the location of the symptoms vary; the visceromotor involvement is almost constant. When the upper thoracic cord is affected, flaccid paralysis of both arms with spastic paralysis of both legs may occur. Paralysis of the intercostal muscles compels diaphragmatic breathing; when the cardiac centers, or the phrenic center is involved, death is immediate.

Treatment. This is commonly of little value, after the diagnosis becomes possible. The patient should be given no food, but plenty of water. The position should be left lateral or prone, so that the influence of gravity may lessen the spinal congestion; also, less heat is permitted in the spinal region, and the back is accessible to treatment. The spinal muscles may be very gently examined, and any deep contractions relieved. Unless the correction of vertebral lesions is very easily secured, it is better not to attempt this until after the acute symptoms have subsided. Ice bags may be placed over the spinal column; gentle sponging with moderately cool water is better in most cases. The position of the patient must be changed, after the first few hours, rather frequently, as bed sores are almost inevitable. A water-bed or air-bed should be secured if possible.

Prophylaxis. During the progress of the disease, bladder infection should be carefully avoided; catheterization is often necessary, and the greatest of care is necessary to avoid infection; the resistance of the tissues is greatly lowered, and infection is much more dangerous and less easily avoided than under ordinary conditions.

The disease itself is avoided by care during the progress of the acute infectious diseases, tuberculosis, and other rarer infections—as actinomycosis—and by maintaining at all times as good a circulation of as good blood as is possible. Sick persons ought never be left to lie upon the back, but should be turned from time to time; blood vessels, weakened from fever, may yield to effects of gravity, when the relief given by the change of position may prevent injury.

CHRONIC MYELITIS

True chronic myelitis is probably rather rare. Erb's syphilitic paraplegia is probably the same disease. According to Erb's account the disease is characterized by five qualities. First, it is of syphilitic origin. Second, the reflexes are greatly exaggerated without being associated with any marked muscular rigidity. Third, bladder trouble of insidious onset and with symptoms of varying intensity usually antedate the paralysis. Fourth, paresthesias are present and usually associated with the paralyzed areas. Fifth, the disease has always very gradual development and it may improve under the antisyphilitic remedies. The diagnosis and pathology are such as would be indicated by the above definition of the disease.

Cases of chronic myelitis are reported as following the acute form; probably these are either cases of mistaken diagnosis, or are complicated with other spinal cord lesions.

COMPRESSION OF THE SPINAL CORD

(Compression myelitis)

Etiology. This condition is due to trauma; to neoplasms; or to inflammatory disease of vertebræ, as tuberculosis or syphilis. It is almost always associated with meningeal inflammations and with myelitis.

Diagnosis. This depends upon recognition of the causative factors, plus the symptoms observed. These are due to the area affected.

The first injury is usually to the nerve roots. Pressure upon the posterior roots gives pain, neuralgic in character, and radiating along the various nerves of that area. Paresthesias, formication, pains, are followed by anesthesia. Dissociation of sensations may be noted. Pressure upon the anterior roots causes spasmodic mus-

cular movements, followed by paralysis of the lower neuron type. Pressure upon the white substance produces variable effects; and this is closely followed, or sometimes preceded, by pressure symptoms referable to the gray matter of the cord.

The X-ray is of value; tumors of several kinds, caries, and traumatic causes of the compression are thus quickly and certainly recognized. Stereoscopic views give most accurate information in all but the simplest cases.

Treatment. This is mostly surgical or orthopedic. If the pressure cannot be removed, or after the removal of the pressure, the treatment is that of acute myelitis.

The **prognosis** depends upon the possibility of removing the cause of pressure before degeneration of the nervous tissue has proceeded to any great extent, and the power of recovery left in the injured tissues.

MENINGOMYELITIS

This is a parasyphilitic disease, and is probably never found in its typical form except as the result of gummy inflammation of the meninges, with simultaneous or immediate involvement of the nerve roots and the cord substance.

Pathology. The spinal membranes are thickened, and the subdural and subarachnoid spaces are more or less completely filled with gummy deposit. The meninges around the nerve roots are involved in the process, and present a swollen and "stubby" appearance. From the pia, wedge-like projections of the gummy and proliferative process enter and penetrate the white, then the gray matter, destroying the tissues in turn.

Etiology. In addition to the syphilitic infection, overstrain of the spinal muscles, sexual excesses, and exposure to extremes of heat and cold are given as causes.

Diagnosis. The onset is gradual, with pains resembling neuritis. Backache is usually severe, especially in the lumbar region. Motor symptoms of a neurotic nature follow; then disturbance of reflexes, incoördination, and the disturbances in the bladder, rectum and sexual organs. Impotence and priapism are not unusual. Weakness of the muscles is followed by paralysis, of the lower neuron type in the area of the affected segments, and of the upper neuron type in the muscles innervated from segments below the affected area. When the disease is limited to one side of the cord, as it may be for a short time, a Brown-Sequard paralysis may be present.

When symptoms, first of nerve trunk involvement, then of white and gray spinal involvement appear, with history or evidences of syphilis, the diagnosis is evident.

Treatment. The treatment for syphilis must be given. (q. v.) The patient must avoid overexertion, alcohol, sex indulgence, and

any excitement. His life must be absolutely hygienic; his food nonstimulating and abstemious. Such treatment as facilitates the better drainage from the central nervous system is indicated.

SYRINGOMYELIA

This is a disease of the spinal cord and medulla, characterized by neurogliar overgrowth and cavity-formation, with symptoms referable to the areas destroyed.

Pathology. The pathogenesis is unknown; it seems to depend upon congenital defect in the neural canal, and the relation between nerve cells and glia cells. The disease is most common in the cervical enlargement, next in the lumbar enlargement, and occasionally involves other parts of the cord, the entire length of the cord, or the medulla. It affects the central canal, in some part of its extent, almost invariably, and is thought to originate in the ependyma cells. It involves the posterior horns, preferably, and destroys the lateral, anterior, and white matter less frequently, or later. Hemorrhagic areas are constant; some cases appear to originate in an old hemorrhage into the cord.

Etiology. This is very uncertain. The disease appears about equally in the sexes; is most frequent before thirty, and the first symptoms are often observed soon after puberty. Investigation often brings out earlier symptoms. Syphilis is not directly a cause; this infection may lower the resistance of the blood vessels. History of trauma is not common; the slow and gradual onset would in any case tend to obscure slight injuries.

Diagnosis. This disease may be suspected when the sensations of heat, cold and pain are lost, or noticeably diminished, for any part of the body, with little or no loss of tactile and muscle sense. The diagnosis is made upon the symptoms and course of the disease; no laboratory findings are of value.

The onset is remarkably slow and gradual. Slight variations in the skin sensations, located according to the area of the cord first involved, first appear. The loss of the temperature sense is early, and severe burns may be produced without pain. The nutrition of the skin in the affected area becomes disordered; slight wounds do not heal well; the skin thickens, and skin lesions follow which may resemble almost any of those mentioned in a book on dermatology. Leprosy and skin tuberculosis may give difficulty in the diagnosis. Bed sores are produced with remarkable facility. Vasomotor disturbances are common; blebs and gangrene may suggest Raynaud's disease. Localized areas of hyperidrosis and anhidrosis may occur. The bones break very easily; apparently spontaneously sometimes.

Sensory disturbances vary. The injury of the posterior horns and the gray decussation destroys the conduction paths for heat, cold, and pain; these are usually lost; tactile and muscle sense are often diminished, and are lost if the posterior white tracts are

involved. Coördination is usually diminished. Various paresthesias and hyperesthesias may precede the sensory loss.

Motor disturbances are variable, also, according to the amount of injury to the anterior horns, the lateral descending tracts, and the effects produced by sensory and trophic disturbances. Paralysis is of the lower neuron type, at the level of the cavity and gliosis; and of the upper neuron type below the lesion—from involvement of the pyramidal fibers. Lower neuron paralysis of the arm, with upper neuron paralysis of the leg, is not rare. Variable electrical reactions and reflex disturbances depend upon the tissue destroyed. Muscular tremor, spasm, and twitchings of muscle groups may be variably present. Symptoms of myotonia may occur.

Peculiar overgrowth of one or both hands, or one or both feet may result, probably from the trophic effect of the lesion; possibly from the underlying causative factor of the gliosis. The affected parts become broad, with thick skin and considerable distortion of parts. The paralysis is usually marked, and the deformity may be very considerable.

Morvans' disease is a form of syringomyelia described by Morvan; it is characterized by progressive wasting and paralysis of the upper limbs, sensory loss, and painless whitlows which result in more or less loss of tissues—the fingers may drop off, joint by joint, with little or no sensation. Neuritis may be present at an early stage.

Treatment. Since the disease has a basis of congenital defect, it is evident that recovery is not to be expected. Some relief of the symptoms may follow careful treatment. First, it is necessary to provide good circulation of good blood, with elimination of the waste products of metabolism as rapidly as possible. The correction of structural conditions must be accomplished with great care, remembering the delicacy of the bones of these patients, as well as the fact that very slight pressure often produces evil effects. For the prevention of bed sores, a water-bed, air-bed, or specially constructed mattress may be employed. Let the patient lie in the left lateral position, or otherwise prevent the gravity flow of the blood from adding to the spinal congestion. As long as it is possible, he may remain out of bed; but overexertion must be prevented.

The sensory disturbances may be relieved by hot and cold applications and by very gentle massage. The loss of the heat, cold and pain sensations permit serious injury from hot water bottles, touching hot things, permitting the feet to become too cold; neglecting accidental injuries especially to the feet, with later infection of the injured tissues, and many other factors.

Coördination can be preserved, to some extent, by reëducation of the affected muscle groups; exercises should be specially devised for each patient, with reference to his especial needs.

The various orthopedic appliances useful in infantile paralysis may be used with help, after the paralysis has become fixed.

Occasionally symptoms of syringomyelia are produced by dural tumors; these are operable in certain cases, and a symptomatic recovery may be hoped for. When the pain is very severe, and the hopelessness of the disease is certainly recognized, partial or complete section of the spinal cord above the cavity may give relief; after such an operation the trophic disturbances are usually relieved, doubtless on account of the relief of the pain and the consequent better rest and better nutrition.

Prognosis is always very gloomy for recovery. Life may not be shortened by the disease. Palliative results are all that can be expected from the best of treatment.

CAISSON DISEASE

(Diver's paralysis; the bends)

This is a paralytic disease resulting from sudden change from high to low atmospheric pressure, and characterized by cramps, pain, and varying paralysis which is more or less permanent.

Pathogenesis. Under the high air pressure necessary for work in caissons or within the suits of divers under deep sea pressures, the blood takes up more air than it can hold in solution under normal pressures; also, the blood is driven from the surface of the body into the deeper organs. When the pressure is too speedily diminished, the vessels are unable to accommodate themselves to the sudden change, and stasis and hemorrhages result; also, the air is set free from the blood in the capillaries with almost explosive force. In the soft tissues, this produces pain and cramps of the muscles, but no permanent injury. In the spinal cord, however, and to a somewhat less extent within the skull, the force of this escaping air seriously injures the delicate tissues. The injury is rather greater in the cord, because the small canal receives suddenly the pressure changes, while the rigid wall prevents escape of the pressure. Within the skull, the openings are smaller, proportionately, and decompression is necessarily somewhat delayed. The greater size of the skull also permits greater elasticity of the contents, and the escaping air produces less frequently serious effects than in the spinal cord.

Etiology. Men who are alcoholic, or who are overfat, or are at all subject to arteriosclerosis, are most easily and seriously injured. Young and vigorous men, who remain under pressure not more than two to three hours, are decompressed slowly, and who live hygienic lives, seem able to engage in this work without serious results. The greater the pressure, the shorter the time that is safely endured. Strenuous working, heavy lifting, haste, also increase the danger. At three atmospheres, one hour should be the limit of time at work, with two or three hours intervening rest, at

normal pressure. Half an hour to an hour should be spent in decompression, according to pressure.

Neuralgic pains in the muscles and joints, with giddiness, are the mild symptoms. Headache and tinnitus, with cramping pains in the muscles; then anesthesia, and weakness of the legs, then of the entire body, are noticed. After being a few hours or a day under ordinary pressure, paralysis develops; this is usually paraplegia, and the sphincters may be also involved. Monoplegia and hemiplegia are sometimes found; total motor and sensory paralysis for four limbs and the trunk may be produced.

Treatment. The prophylaxis consists in short hours of work; the forbidding of alcohol for workers; and slow decompression. When the disease appears, the patient may be gradually subjected to a pressure greater than that to which he has previously been submitted for a short time, then be decompressed with extreme slowness—a day or more may be devoted to this process, if the disease resulted from very high pressure. Under the high pressure, the blood again takes up the air bubbles, and under the extremely gradual decompression, this is all breathed out through the lungs, as is to be desired. If this decompression is done immediately, recovery may be absolute; the longer the delay in initiating the treatment, the greater is the tissue destruction.

LANDRY'S PARALYSIS

This is a disease of adults characterized by a very sudden and acute onset, ascending flaccid paralysis of the leg, thigh, abdomen, thorax and arms and neck. It is probably due to some infectious agent.

Etiology. The disease is somewhat more frequent during an epidemic of infantile paralysis, and this together with simultaneous incidence of the two diseases in the same household has led to the inference that it may be due to the same organism. On the other hand no relationship can be established in most cases. The disease appears between the twentieth and fortieth year for the most part. Men are more often affected than women. There is no reason for supposing that the disease is due to any previous infection, though it has been reported in adults who had had infantile paralysis in childhood. In a few cases the symptoms of Landry's paralysis may appear in the terminal stage of typhoid, pneumonia, or other infectious diseases.

Pathology. The spinal cord shows all the usual symptoms of an acute inflammatory process involving particularly the anterior horn cells; this is practically identical with the change in the anterior gray matter in infantile paralysis.

Diagnosis. The disease begins very suddenly. Fever, nausea, sense of weakness, and malaise appear first. Weakness of the legs is speedily followed by paralysis and this weakness and paralysis involve successively muscles of the thigh, the abdomen, the arms and the neck. The foot is rarely affected. The muscles are atonic and the reflexes are lost. No sensory disturbances are present in typical cases. There is no loss of control of the sphincters. The mind remains clear until death. When the paralysis involves the thoracic muscles, respiration is managed through the action of the diaphragm alone; then, when the cervical segments are affected, the paralysis of the diaphragm causes death. The heart's action is not affected and life may be maintained even for hours by the use of artificial respiration. Death usually results in a few days. Rarely the patient may live and the later symptoms produced resemble acute ascending myelitis. Rarely sensory symptoms are present, such as numbness, tingling and cramps in the affected muscles. These are no doubt partly due to the effects produced in the muscles and their action upon the sensory nerve endings in the muscles themselves or it may be that the sensory disturbances are due to the involvement of the posterior gray matter of the spinal cord.

Treatment. Whatever manipulative work is indicated during the acute attack should be carried out with special care. Patients suffering from this disease should be isolated. Since the infectious agent is not known the sick room should be carefully screened from flies and the patient protected from insects. All excretions including those from the nose and throat as well as urine and feces should be thoroughly destroyed. The room should be disinfected after the death or recovery of the patient, just as if he had suffered from any of the ordinary infectious diseases. The fever should be controlled by the ordinary treatment and by gentle baths of tepid water. Great care must be used to prevent having the water cold enough to produce any shock to the sensory nerves. Sponging with water of the temperature of the body reduces the fever and frequent repetitions of this are much better than the use of water which is too cool. Patients should be induced to lie either upon the face or in the right or left lateral position in order that the force of gravity may help to prevent congestion of the spinal cord. The dorsal decubitus should be avoided if possible in this as in all spinal cord disease. Solid food should not be given. Water and fruit juices should make the most of the diet permitted. If the patient recovers from the acute disease the methods of treatment advised for infantile paralysis and for myelitis may be adapted to his particular paralytic symptoms.

Prognosis. This is always grave. Death may be expected within a week or in two weeks at most. Complete recovery is never to be expected and the remaining paralysis is always serious.

SPINAL PROGRESSIVE MUSCULAR ATROPHY

The various diseases which are chiefly characterized by slowly progressing atrophy of skeletal muscles present so many similar features that they may be discussed together, with only various factors of differentiation mentioned in detail. Atrophy of muscles may be due either to disease or degeneration of the muscle itself, as in the muscular dystrophies; or to diseases of the nerve trunk, as in multiple neuritis; or to diseases of the cells in the anterior horns of the spinal cord, as in infantile paralysis or the ordinary form of progressive muscular atrophy; or to diseases of the descending cerebrospinal tracts which in turn produce atrophy of the anterior horn cells, as in amyotrophic lateral sclerosis.

Spinal progressive muscular atrophy includes a class of diseases in which the atrophy of the anterior horn cells of the spinal cord is responsible for atrophy of the nerve fibers and of the muscles supplied by them. The features which distinguish the various diseases included under this generic term depend upon the area of spinal cord first affected and the manner in which the disease extends to other nerve centers.

The most common is the **Duchenne-Aran type.** In this disease the atrophy begins in the anterior horns of the cervical thoracic cord which gives origin to the seventh and eighth cervical and the first thoracic nerves; weakness of the hands is thus one of the first symptoms. The thenar and hypothenar eminences diminish in size. The interossei and lumbricales slowly weaken and disappear. The shoulder muscles are next atrophied and the disease extends very slowly to the upper arm and to the trunk muscles. Death usually occurs from some intercurrent disease but after years of slow progression the atrophy may involve the respiratory muscles thus leading to death.

Duchenne's Subacute Ascending Paralysis. This form begins in the lumbosacral region of the cord and thus the muscles of the feet are first affected. The disease involves next the muscles of the thigh, then of the leg. It ascends to the trunk muscles as the disease ascends through the nerve centers in the cord. Ultimately death results from paralysis of the respiratory muscles, unless some intercurrent disease, usually pulmonary, interferes with the paralytic course of events. The progress of this disease is more rapid than in the case of the Duchenne-Aran type as is indicated by the term "subacute."

A third type (Erb), which is more rare in this country, begins also in the lumbosacral region and affects the peroneal muscles and the anterior tibial, but does not extend to the arms, though it may involve most of the leg and thigh muscles. This disease is extremely slow and may remain stationary for several years. It practically never causes death except indirectly, from accidents due to paralysis.

Progressive Bulbar Palsy may be included in this group also. In this disease the atrophy begins in the motor nuclei of the medulla. There are difficult speech, drooling, dysphagia, which may go on to complete inability to speak, swallow or close the lips. The mentality is not affected. The heart is very weak and rapid and death is due to complete failure of the heart or starvation or suffocation, or all three combined.

A number of other variations in type have been described, with slightly different symptoms. These are all very rare.

Pathology. The pathology of all these diseases is practically identical, a chronic anterior poliomyelitis. The nerve cells in the anterior horns of the spinal cord undergo a very slow atrophy. There is no chromatolysis, no extrusion of the nuclei, no swelling, no pigmentation, and no degeneration in the ordinary sense of the word. The cell-body shows first merely an increased pericellular lymph space. Later the body of the cell diminishes. The nucleus diminishes in size and takes the stain more lightly than under normal conditions. This process goes on until nothing is left of the nerve cell or of the axon which arises from it. The neuroglia proliferates, filling up the space left by the disappearing nerve cell. The spinal cord in an old case may show almost no trace of the nerve tissue in the anterior horns. The columns of Clarke and the posterior horns remain unaffected. The nerve trunks share in the atrophy; there is no recognizable proliferation of the connective tissue cells of the nerve trunk.

Sections of the muscles during the process of atrophy show a granular muscle protoplasm followed by a deposit of fine fatty granules. Occasionally a single hypertrophic muscle fiber may appear. The atrophy of the muscle is very complete and ultimately only a shred of connective tissue may mark its original site. Usually bony lesions are found in close central connection with the spinal area involved, but it is not possible to say whether these are primary or secondary.

Etiology. Practically nothing is known of the real cause of this disease. It begins in middle life, rarely appearing before the age of thirty. It is found in adults who have suffered from infantile paralysis in childhood, but perhaps not more frequently than the law of averages would explain. Occasionally it seems to date from some infectious disease, and sometimes from pregnancy, especially from very frequent pregnancies. Alcoholism and syphilis are not probable factors.

The place of the bony lesion has already been mentioned. The presence of bony and muscular lesions associated with the spinal centers first involved is probably invariable but whether these are localizing factors in the disease or whether they are really important in etiology or whether they are merely some of the effects

of the disease acting through the spinal muscles cannot yet be determined.

The **diagnosis** depends upon the history of a slow and gradually increasing muscular atrophy with no sensory, bladder or erectile symptoms. It is distinguished from progressive muscular dystrophy in the fact that this disease appears in childhood, and is associated with hereditary or family history; has no fibrillary tremors and has usually an individual history and an early hypertrophy.

Laboratory tests and X-radiance throw some light upon the diagnosis. Fibrillary tremors are present during the early stages. There may be pains in the muscles themselves which are apparently due to the fatigue of the weakened muscles being used as if they were normal.

No **treatment** seems to affect the course of the disease in any really efficient way. In correcting the bony lesions great care must be exercised to prevent irritation of the sensory nerves distributed to the skin, muscles and articular surfaces which are in close sensory connection with the trophic area. This work very slowly and gently accomplished seems to delay the progress of the disease in most cases. In a few cases such treatments given once each week or once in two weeks through several years have been associated with relief of the symptoms while the disease seems to progress less rapidly. Perhaps in cases seen very early thorough treatment will be successful.

Rest should be given the affected muscle groups and ordinary good hygienic conditions should be maintained. Some authors advise hot applications to the muscles involved. Mild stimulation by static electricity and frequent gentle massage of the affected muscles are useful in some cases, though these agents appear in other cases to increase the rapidity of the atrophy.

Prognosis. Recovery is probably impossible since the atrophy of the muscles and of the nerve cells innervating them is complete. The disease may remain stationary for some years at almost any time. It is not likely to cause death unless the atrophy involves the trunk muscles or the bulbar centers. When the paralysis involves the legs it may lead to accidents. Pulmonary affections usually give the terminal chapter in the story of this disease.

AMYOTROPHIC LATERAL SCLEROSIS

Amyotrophic lateral sclerosis is a disease of the spinal cord characterized by atrophy of the descending pyramidal tracts, secondary atrophy of the anterior horns of the cord and a slowly progressive muscular paralysis and atrophy.

Pathology. The fact that the disease begins in the lateral tracts in most cases is demonstrated by numerous autopsies, though for a long time it

was supposed that the anterior horn cells were primarily degenerated and that the atrophy of the lateral tracts appeared as a secondary phenomenon. Examination of the spinal cord of the patient who has suffered from this disease shows that the atrophy of the lateral tracts begins at the end of each individual axon, extending upward. The atrophy does not usually pass the medulla, though in a few instances it has been traced to, and involving, the larger pyramidal cells in the precentral convolution.

Etiology. The cause of the disease is not known. It is probably to be included among the abiotrophic diseases. The nerve cells are not reproduced during life so far as our present knowledge goes, and when any given cell is subjected to a greater amount of use or to less than the conditions required for its proper nutrition then progressively increasing inefficiency must result. Such a cell shows senile changes. These may become evident either in the cell body or, as in the case of amyotrophic lateral sclerosis, by a diminution of function followed by an atrophy which most commonly shows disease first in those parts of the protoplasm most distant from the nucleus of the neuron. Atrophy of the lateral tracts may follow anything which separates these fibers from their cell body. Direct injury to the spinal cord such as may be produced by wounds or other trauma, or by tumors, which may exert pressure upon the spinal cord, tumors or abscesses, tubercles or gumma, hemorrhages or any other pathological changes in the brain itself or in any part of the pathway traversed by the pyramidal axon may result in the atrophy of the crossed, or more rarely, the direct pyramidal tracts and thus to the development of the disease referred to. In pernicious anemia and in multiple sclerosis paralysis of this type may be one of the earliest and most conspicuous symptoms.

There is no reason to suppose that syphilis is an important factor. Probably anything which lowers the nutrition of the body as a whole or which interferes especially with the nutrition of the spinal cord may act as an exciting or predisposing factor in the development of this as in any other nervous disease.

The place of heredity in this disease has been very much discussed. There seems no doubt that there is a tendency for the disease to appear among members of the same family and also to be interchangeable in inheritance with several other diseases of the central nervous system.

Diagnosis. The disease usually appears first in the lower part of the body, perhaps because the crossed pyramidal tracts are made up of the longest fibers. There is at first a stiffness in the muscles involved which is associated with an increase in the muscular tone. Reflexes are increased. Paralysis may involve the hands first of all in which case the small muscles are first paralyzed and the disease follows the path of the Duchenne-Aran type. Fibrillary contractions are present. Reflexes are increased; ankle

clonus and Babinski are present. There is a spastic gait. The legs may cross in walking, giving rise to a "scissor's gait," which is rarely very pronounced. The disease is usually bilateral, but one leg may be first involved. There are no bladder or rectal symptoms. As the process of atrophy extends the muscles are progressively involved. When the disease affects the bulbar centers the muscles of the lips, tongue, palate and throat are paralyzed and later atrophied. Fibrillary twitchings are prominent symptoms of the beginning of this atrophy. The patient's speech, deglutition, and mastication become increasingly difficult. The face becomes flat and expressionless after the completion of the paralysis. Rarely the eye muscles are involved. Intelligence is not affected. When the atrophy extends above the medulla there is, as in bulbar palsy, a tendency to excessive emotional expression, so that the patient laughs and cries more extravagantly than is usual among normal individuals. The cause of this excessive emotionalism is not known. It is a source of considerable annoyance to those patients whose knowledge of their own condition is decidedly acute. There is very good reason to believe that the integrity and development of the motor system as a whole is directly associated with those psychological qualities commonly included in the expression "self-control."

Tachycardia is a fairly constant symptom. Death may occur from some intercurrent infection usually of the lungs, or starvation may result from the bulbar effects.

Treatment. The osteopathic treatment must be planned to secure the best circulation through the spinal cord and the muscles as well as to keeping up the general circulation and nutrition of the entire body. Since nervous diseases of this type seem to follow Mendel's law persons from neurotic families should be strongly advised against intermarriage. When one parent comes of a family in which this or other nervous diseases of an abiotrophic type have been present every care should be exercised to guard the children from causes of malnutrition. Their life should be more than usually hygienic. No strain, overwork or excitement should be permitted. Not only while they are children but also when they have reached adult life they must live normally hygienic lives if they are to avoid falling a victim to this or some other serious nervous disease.

After the onset of the symptoms the general health is to be kept up in every way. Stimulation of the muscles by mild massage or by electrical and thermal stimulation seem sometimes to be of some use. When there is difficulty in swallowing it is better to give all food and drink through the stomach tube in order to avoid the danger of aspiration pneumonia.

Prognosis. Probably no recovery is possible. The best that can be hoped for is to somewhat delay the progress of the disease and possibly to cause it to become stationary.

HEREDITARY SPINAL ATAXIA

There is a group of abiotrophic diseases which are variously described by different authors and which present symptoms which vary to a certain extent and have resemblances which are sometimes quite marked. By some authors the term Friedreich's ataxia is applied to the entire group of hereditary ataxias, which are characterized by symptoms referable to degeneration of the posterior funiculi of the cord. The description given, however, seems to apply especially to the spinal cord type of diseases, while the cerebellar form has been best studied by Marie.

The disease is invariably hereditary or familial. The ataxia appears from the third to the ninth year. It involves the legs and the arms about equally. At first, the children appear merely to be awkward; they walk with straddling gait, and the feet are turned in somewhat the position of varo-equines; the child drops things, spills fluids he is trying to carry, knocks things from the table, falls, and appears generally to be awkward and careless; as the condition grows worse, it becomes evident that it is a disease rather than a bad habit from which he suffers; the facial muscles and the respiratory muscles may be involved in the later stages; with constant effort some of these children learn to walk and to handle themselves fairly well. A kyphosis or kypho-scoliosis is almost always present. Nystagmus is bilateral. Stuttering speech is followed by an unintelligible jargon. Intelligence may not be affected.

The pathological changes include degeneration of the posterior funiculi, atrophy of the cells in the dorsal nucleus (Clarke's column), and of some of the cells in the posterior root of the ganglia, and some of the fibers in the peripheral nerves.

Recovery is impossible. The disease does not shorten life, unless some accident should occur as the result of the incoördination. One family was kept under observation for several years in the Pacific College Clinic. Attempts were made to correct the kyphosis but without success. No good results from the treatment were perceptible.

CHAPTER XXXVI

DISEASES OF THE BRAIN

GENERAL DISCUSSION

The diseases to which the brain is subject appear to be due for the most part to variations in its circulation and to the effects of bacteria and of various poisons. There is no reason whatever for supposing that any brain disease is due to overwork except as too long continued devotion to work may lessen the hours of sleep and exercise, or the food necessary to the maintenance of a good circulation of good blood; in other words, in all cases in which overwork is considered a cause of brain disease it is far more probable that it is the poor nutrition, the lack of oxygen or the presence of toxic materials in the blood that is responsible for the brain disease rather than any overactivity of the brain tissue.

The brain is supplied by terminal arteries; vasomotor nerves are known to be supplied to these and to originate for the most part in the superior cervical sympathetic ganglia which in turn receives its control from the first to the fifth thoracic segments of the cord. Bony lesions in this area may thus modify the circulation through the brain to a certain extent. By far the most important factors in controlling the arterial supply to the brain, however, are the conditions which modify the general blood pressure.

Infection of the brain itself extends from meninges in most cases. As in the case of the spinal neurons the extreme irritability which gives the brain its value in function renders it also extremely subject to the effects of poisons in the circulating blood. Poisons as well as bacteria seem to have a selective action upon certain parts of the central nervous system. This is especially noticeable in the effects produced by the syphilitic toxins, lead, mercury, the bacteria responsible for anterior poliomyelitis and the protozoa responsible for rabies.

Circulatory disturbances may be due to poisons, in which case they most frequently occur during middle life; or to birth injuries, in which case the symptoms occur during the first year or the first decade of life; or to senile diseases of the blood vessels, in which case the disease occurs after the sixtieth year. Functional nervous diseases are associated with the time of life during which the nervous relationships and neuronic development are undergoing most pronounced changes and are thus especially frequent during the period of adolescence or during the climacteric period.

CEREBRAL ANEMIA

This condition occurs secondarily in a number of other disorders. It is characterized by nausêa, sometimes vomiting, and dizziness, vertigo, or syncope. Mania or delirium may occur. A mild degree of anemia is present during normal sleep.

General anemia, such as occurs after large hemorrhage anywhere in the body, is associated with cerebral anemia also.

In cachectic diseases, with primary or secondary anemia, the brain shares in the bloodlessness. Varying mental disturbances may result; drowsiness and stupor, even to coma; mania and delirium, or only a diminished interest and ambition, are the results of the starvation and poisoning that are caused by a deficient circulation of poor blood through the brain.

Ordinary fainting, or syncope, is due to overfilling of the splanchnic vessels with blood; under certain emotional disturbances, fright, horror, disgust, rarely anger or delight, the vasomotor centers controlling the liver, intestines, and spleen appear to be paralyzed, and these organs are filled with blood; the muscular wall of the spleen and the muscles of the intestines are also relaxed. The first condition adds to the cerebral ischemia; the second adds to the ischemia and also permits carbondioxid gas to be set free in the intestines in considerable amounts.

Treatment is usually devoted to the underlying cause of the bloodlessness. Anemias must be treated according to the causes of this condition. In ordinary syncope, the head must be lower than the reclining body, and sensory stimulation, sprinkling of cold water, rubbing the hands, smelling salts, etc., are all useful. If the heart is weak, stimulating manipulations around the fourth thoracic spine, and in the left fifth interspace anteriorly, hasten its return to normal.

Another form of cerebral anemia is produced by pressure; overfilling of the meningeal vessels or the venous sinuses, or tumor, or serous meningitis, or cerebral edema, all cause an ischemia of all or part of the brain substance. In these cases, the symptoms produced vary greatly.

CEREBRAL HYPEREMIA

Active hyperemia, aside from the primary stage of inflammatory process, is not certainly known to exist in the brain. The fact that the vasomotor nerves of the brain are comparatively inefficient, and that the circulation is chiefly dependent upon variations in the general blood pressure, seems demonstrated by clinical and by experimental evidence. The rigidity of the skull also prevents the facile variations in circulation, in functional activity, that is found in glands and other active tissues of the body. Transient apoplectiform attacks may be due to the sudden local hyperemia of the brain, and these are best treated by elevating the head, inhib-

iting the splanchnics, and the application of ice bags to the head. The old tendency to consider hyperemia of the brain present when the face is red and congested, is now known to be fallacious.

Passive congestion of the brain may be caused by pressure upon the jugular, the innominate, or the vena cava, or by tricuspid lesion. It is characterized by constant, dull headache, somnolence and sometimes mental torpor. The **treatment** includes removing the pressure upon the veins, in the one case, and inhibition of the splanchnics, correction of muscular and bony lesions which may interfere with the circulation in any way, and, in general, relieving the burden upon the heart, in the other.

CEREBRAL EDEMA

Passive congestion of the brain may cause edema. Nephritis, blood diseases, heart diseases, cause edema of the brain, as of other organs. Angio-neurotic edema may affect the brain, either locally or generally; death may result from this disease. Alcoholism, especially, may cause a form of "wet brain" probably an oversecretion of the cerebrospinal fluid as the result of an inflammatory process (see serous meningitis). Certain other forms of meningitis may be associated with an increase in the cerebrospinal fluid, but these, as hydrocephalus, may not be associated with any increased amount of water either between or within the brain cells.

Edema of the brain cannot be certainly diagnosed ante-mortem, but may be suspected when the symptoms of increased intracranial pressure appear together with any of the etiological factors just mentioned.

The **treatment** is that of the causative factors, plus measures toward equalizing the circulation of the blood, and especially facilitating the drainage from the cranial cavity. Correction of all abnormal structural relations in the cervical region, anteriorly and posteriorly, and such manipulations as may be required to give plenty of room in the thoracic inlet, are the most important factors. The treatment must include good hygiene and frequently some special diet, adapted to the patient's general condition.

APOPLEXY

The term is limited by some authors to cerebral hemorrhage; the impossibility of making ante-mortem diagnosis between hemorrhage, thrombosis, and embolism, and the close relation between these accidents in their pathological, etiological, and clinical significance has led to the broader definition of the word. Apoplexy is a circulatory accident occurring in the brain, and characterized by sudden onset of paralysis, with varying degrees of unconsciousness.

Cerebral hemorrhage includes also meningeal hemorrhage, insofar as this produces cerebral insult and symptoms of apoplexy. At the time of birth, and for a few months after, cerebral hemorrhage is rather frequent; after this period it is rare until the time of arterial degeneration. Hemorrhagic diseases, as scurvy, "black" infectious diseases, etc., may be associated with cerebral hemorrhage; such cases are rare, and may occur at any time of life. After the age of fifty, arteriosclerosis is by far the most common cause of apoplexy. Syphilitic endarteritis is a frequent cause.

By far the greater number of cases are due to rupture of small aneurysms; and this most frequently occurs in the left lenticulostriate artery. This fact is due to the anatomical relations of the branches of the aorta; the most direct path of the cardiac force leads from the left ventricle to this artery. For the same reason, the middle cerebral artery, in some of its branches, is most often the seat of embolism.

Injury to the head, as a blow, may cause rupture of the vessels; this may occur upon the surface of the brain, or within its substance, according to the location and force, and the manner in which the blow falls upon the skull. It must be remembered that the brain, during life, is almost fluid in consistency, and that, like other fluids, it transmits force, undiminished, in every direction. The structural injury produced by a blow with a soft or elastic object is the resultant of many varying lines of force. The destroyed area may be upon the opposite side of the brain, or upon some area in the basal ganglia, where the lines of force meet or cross. Such distant injuries are said to be produced from "contre coup." Disintegration of the brain substance may follow such a shock, and this may result in weakening a blood vessel, which ultimately yields to some slight variation in the blood pressure; this is called "delayed apoplexy."

The term "apoplectic habit" is applied to stout, heavy-set people, usually with thick necks and red faces. When such persons are overfed, alcoholic, and deficient in self-control, they are decidedly prone to apoplexy.

Capillary hemorrhages may result from cerebral congestion, or from any of the infectious diseases; they usually produce no recognizable symptoms, and are merely found after death. Venous hemorrhages may result from injury, from the rupture of varicose veins, or from passive congestion and emotional or muscular stress.

In all cases, some pathological condition of the cerebral vessels must be supposed to be present if any ordinary change in blood pressure initiates hemorrhage. This weakness being present, the rupture may be finally caused by anything which raises the general blood pressure; emotional stress, coition, straining at stool, coughing, muscular effort, as running or lifting, are all causes;

but many cases occur during sleep, and at times of absolute quiet of body and mind.

Diagnosis is directed chiefly to the localization of the injury. Prodromal symptoms are not frequent; there may be headache, increased blood pressure, paresthesias, weakness, vertigo, a vague discomfort, and a tendency toward awkward speech for a few hours or days before the attack. The speech difficulty attracts most attention, and is rather diagnostic of an impending attack, in persons whose vessels are diseased. The voice becomes slightly husky, the words enunciated more slowly but less plainly than usual, and there may be tendency to "forgetfulness" of familiar words—which is really a form of aphasia—in such cases. The symptoms are usually referred by the patient and his family to slight indigestion. The attack begins with unconsciousness of sudden onset. The patient falls; breathes with stertor; the cheeks are relaxed and flap; the pulse is first feeble, then becomes full and strong; the blood pressure is high; the face is flushed, often purple; there may be relaxation of the sphincters. The pupils may be contracted or dilated or normal; the eyes and head may or may not be deviated; the limbs fall flabbily when raised. If the hemorrhage is in the medulla or the fourth ventricle death usually occurs during this time. If the temperature is very high—106° or so—death is usually inevitable within a few hours. If death does not occur this period passes away in a few hours to a day or two. Consciousness returns, more or less completely, and the extent of the paralysis is manifested. Fever, delirium, coma, with spasmodic movements of the muscles of the affected and the sound side, may persist for several days. The temperature on the paralyzed side is higher, perhaps .5° to 2.°, than on the normal side of the body. Reflexes are lost, at first, then are exaggerated. Speech is usually impossible for some days, even when the speech center is not directly affected; mentality seems dulled for some days after the other symptoms have largely disappeared. Within a few days to a few weeks, the effects may have altogether disappeared; or, when the hemorrhage has been great, or the locality affected of considerable importance, there may be permanent paralysis of the side of the body opposite to the injury. The exact extent and locality of the paralysis depends upon the location and the extent of the cerebral injury.

In most cases, the hemorrhage is from the lenticulo-striate artery, and affects the internal capsule, thus, the fibers descending from the motor cortex of the left side. Injury to the deeper cerebral tracts involves the speech mechanism. Lesion of the posterior limb of the capsule causes sensory disturbances; bilateral homonymous hemaniopsia, somatic anesthesias, and partial deafness in

both ears may thus be produced. Sometimes muscle and thermal sensations are lost, but touch, vision, and audition are preserved.

When the hemorrhage involves the pons, the pupils are contracted, the temperature is high, the face is paralyzed upon the side of the lesion, and the arms and legs upon the opposite side. When the injury is in the lower part of the pons, the oculomotor nerves may be paralyzed upon the side of the lesion, and the rest of the face and the limbs upon the opposite side.

Cerebellar hemorrhage is rare, and is hard to recognize. If one lobe is involved, no effects may be produced, beyond the initial insult. Hemorrhage into any of the ventricles is usually quickly fatal. Ingravescent apoplexy is characterized by the slow onset of the coma; it is usually fatal. A second attack, occurring before recovery from the first, is usually fatal. If partial or complete recovery occurs between attacks, a large number of successive attacks may occur, without fatality; that the hemorrhage will ultimately be fatal may be granted in every case; unless death from some other cause occurs speedily.

The paralysis produced is of the upper neuron type; except as hemorrhage into the pons might produce lower neuron paralysis of the facial nerve. Reflexes are exaggerated, but may seem to disappear as the result of the contractions. No reaction of degeneration, or true muscular atrophy occurs, though as the result of disuse, and the steady pull of opposing muscle groups, deformity and atrophy of the muscles may ultimately be noted.

Occasionally, hemorrhages in other parts of the brain, and the effects of the hemorrhage upon the basal centers themselves, cause various choreic and athetoid movements; instability of the emotional states; easy laughter and weeping; stammering and stuttering speech. The mental processes may remain fairly normal, even with these effects, but more frequently mentality deteriorates steadily. Epileptiform attacks are even less frequent, especially in nonsylphilitic cases.

Embolism. The obstruction of an artery by materials carried in the blood stream is called embolism; the matter which is carried is an "embolus," or plug.

Pathology. In the brain, since the arteries are terminal, embolism produces a cone-shaped infarct, whose apex is the point of obstruction and whose area is that of the distribution of the artery interrupted. If there is overlapping of other arteries, or anastomosis in any degree, the after-changes are slower, and recovery may occur. The loss of the circulation may cause recognizable changes in the nerve cells and fibers within a day; the starvation and degeneration of the nerve tissue is very rapid. When there is any hemorrhage into the infarct—and there usually is, especially in gray matter—the process is called "red softening." With progressive digestion of the hemoglobin, and with the occurrence of fatty degeneration, especially in areas which have been hemorrhagic, the "yellow softening" takes place. Infarcts in the scantily-blooded white matter are often colorless—"white softening" then occurs. Softening is the same process, however, in all colors. The nerve cells

and fibers undergo first granular, then fatty metamorphosis, then are digested, and, in time, absorbed. A clear liquid is left, if the softened area is of some size, which may be slightly tinged with red or brownish color. The degenerating material gives stimulus to the connective tissues around the blood vessels, and to the neuroglia of the injured region, so that either or both of these tissues multiply, forming a wall, which surrounds the larger infarcts and contains the liquid remnants (hemorrhagic cyst), or fills the cavities left by the small ones with a scar-like tissue.

Etiology. The emboli are most frequently fragments of clots or vegetations from the aortic valves. Less frequently bacteria or fragments from an atheroma may become emboli. Materials from the lungs sometimes pass through the heart and become emboli, these are often infected, and thus the infarct is also infected by the same disease as that of the lungs.

Thrombosis. Clotting of the blood in a vessel may result in complete obstruction; this process is called "thrombosis"; the clot is called a "thrombus." The same process of infarction follows as in embolism. (q. v.)

Etiology. The coagulability of the blood is increased in pregnancy, in most fevers, after hemorrhage, and in certain of the blood diseases and in cachexia. In the arteries, where the current is usually comparatively rapid, thrombosis usually occurs as the result of atheroma, aneurysm or traumatism. All of these factors are exaggerated by the presence of increased coagulability of the blood. Thrombosis of veins may be due to varicosities, diseased vessel walls, trauma, but is more frequently due to those diseases which increase the coagulability of the blood. Marantic thrombosis, in children with marasmus; cachectic thrombosis, in patients with tuberculosis, carcinoma or chlorosis; and anemic thrombosis, after hemorrhages, or in blood diseases, are not very uncommon causes of apoplectic attacks. The venous sinuses in the brain are so broad, and so irregular in shape, the blood flows more slowly through them and thus coagulation occurs more frequently in them than in other veins. The most common seat is the superior longitudinal sinus. When the coagulation is due to trauma, the location of the injury is that of the thrombus.

Diagnosis. The onset is usually less sudden in thrombosis than in embolism or hemorrhage. In either the motor cortex may be irritated and convulsions occur; this is rare in cerebral hemorrhage in adults; otherwise the symptoms are very much like those of hemorrhage.

Prognosis. Death is less probable in thrombosis and embolism than in hemorrhage. Absorption of the clot; digestion of the embolus, within a few days, may permit almost or quite complete recovery. Softening of the brain may extend beyond the original infarct, however, involving small vessels; this is especially true

in infected emboli; these effects, however, are generally less serious than are the results of the organization of the clot, or the dangers of later attacks, in hemorrhagic apoplexy.

Treatment. When any person is unconscious, or presents evidence of clouded consciousness, if the limbs fall flappily when raised, especially if the head and eye-balls are drawn to one side, it is wisest to treat the case as one of apoplexy. A smell of liquor, or signs of chronic alcoholism, are perfectly in harmony with this diagnosis, and too many unfortunate men have been allowed to die from apoplexy and other diseases with coma, because they were treated as common drunkards. It is a disgraceful fact that it is often considered a joke to give a drunk man care, under a mistaken diagnosis of apoplexy, while death from the lack of care, in apoplexy is merely concealed by those responsible for the death.

The patient should be kept recumbent and quiet; preferably on the side, so that the paralyzed tongue may not interfere with respiration. The clothing must be loosened, if there is any constriction, especially at the neck or the waist. Ice bags to the head and hot applications to the feet facilitate cerebral drainage. Steady, deep pressure in the region of the sixth to the tenth thoracic spines, dilates the splanchnic vessels, and withdraws the blood from the brain; this should lower the blood pressure and cause diminished redness in the face. The patient should not be moved until the breathing and the pulse become fairly regular, if this is possible.

After the coma has passed, general treatment, such as maintains a good circulation of the blood, should be given, at first daily, later at longer intervals, to once each week, until no further improvement is to be found. The food must be mild, preferably liquid, and mostly of fruit and vegetables for some weeks. Stimulating foods and drinks are forbidden. Rest in bed is necessary for several days, in the light cases, and several weeks, in more serious forms.

When the extent of the paralysis is manifest, and no further feverishness or indications of impending hemorrhage are found, reëducation of the patient must be begun. This reëducation is important. The opposite side of the brain seems to have a certain amount of power to control the paralyzed side, especially in the more complicated movements, such as speech or writing. Exercises should be carefully worked out for each individual, beginning with movements which he is barely able to begin even in an incoördinated way, and going on through increasing degrees to the attainment of the greatest possible skill. Dr. Evelyn Bush and Dr. A. A. Gour have published articles giving such exercises in greater detail than is possible here; the underlying principles are included in what has been said—the adaptation of the exercises to

each individual, and a constant working up to the more difficult exercises; the first must be very easy and simple.

DELIRIUM ACUTUM

This is an acute, probably infectious, disease of the brain, occurring in persons previously normal and not necessarily either neurotic or subject to any hereditary taint. The disease comes on suddenly, with a high fever, delirium is very severe and violent. Lucid intervals of a few seconds to half an hour in duration may occur at almost any time. The delirium recurs suddenly after these intervals and often the fever is considerably higher. After death the brain is found full of blood and showing the evidences of very acute inflammatory changes. The infectious agent has not been isolated. No evidence of its being contagious has been reported. The only treatment is symptomatic. Ice bags to the head and cool sponges sometimes seem to give some relief.

Death usually occurs within one or two weeks. The few patients who recover have very slow convalescence, but usually no serious mental after effects.

SENILE DEMENTIA

Just how long people ought to live and how long they should be expected to retain full possession of the mental faculties, is a question which cannot be answered. There is very good authority for supposing that "the years of a man's life shall be 120," but this is not at present the case. The senile changes in the tissues of the body are inevitable. Premature senility is due to overwork, especially to severe muscular exertion associated with exposure to climatic changes; to the vascular diseases associated with alcohol, syphilis, sexual excesses, and overeating; and to inheritance.

The brain, as a whole, undergoes a slight atrophy. The sulci appear broader, the convolutions somewhat smaller; increase in the dural fluid occasionally is found. Upon microscopic examination, the nerve cells are found atrophied; the nuclei may be eccentric, and very large masses of yellow pigment granules occur within the nerve cells. The large multipolar cells of the cerebral cortex and Purkinje cells of the cerebellum show atrophy.

Senility may be considered premature in all cases when it occurs before the age of sixty. In families in whom senility is usually delayed until eighty or ninety, this process should be considered premature in any one person at the age of seventy. In other words, the hereditary character of any individual must be taken into consideration in making a diagnosis of premature senility.

The body may not show senile changes even when the mind is seriously affected; on the other hand, the body may show all of the symptoms of old age to a marked extent and yet the mentality be apparently uninjured.

Treatment. The prophylaxis of senile dementia depends upon the maintenance of a normal blood pressure and the rapid elimina-

tion of the toxins of the body throughout life. The mental aspect is usually important. The man or woman who maintains an interest in the world's progress, who takes up new lines of thought occasionally, who is associated on terms of friendliness with young people, and who lives a wholesome, sane life, is less likely to suffer from premature senility.

After the symptoms are observed, a great deal of help can be given by properly planned treatment and attention to hygiene. People with senile dementia almost invariably have very rigid spinal columns and ribs; they breathe inefficiently and they have either a very high blood pressure or other evidences of arteriosclerosis with cardiac lesions. The treatment must be based upon as much of a relief from these conditions as is possible. Treatments which very gently increase the mobility of the vertebræ and which raise the ribs, increasing the flexibility of the thorax, often cause very satisfactory improvement in the symptoms. If possible, the patient should be taught better habits of breathing and should be made to take an interest in something outside of his recent experiences. If he has been taking alcohol and tobacco, it is probably unwise to deprive him of these things altogether, though in most cases a reduction is advisable. The diet should be light and easily digested. Milk and buttermilk, fresh green vegetables and fruits should make up by far the larger proportion of his food. He needs little or no meat and only a very small amount of starchy food. An increased amount of water should be taken. If he can be induced to drink as much as his heart and kidneys will permit, the increased elimination of toxins will be promoted most satisfactorily.

Prognosis. Naturally, no hope of recovery is possible, but considerable relief from the symptoms in early senile dementia may be expected. Attacks of paralysis and occasionally epileptiform attacks may occur and either of these or some intercurrent malady, especially pneumonia, provide the last injury necessary to death.

HYDROCEPHALUS

This term is applied to any condition in which the amount of cerebrospinal fluid within the skull is greatly increased. It may be either congenital or acquired, or may be either internal or external.

Congenital Hydrocephalus may occur without recognizable cause. Its more frequent occurrence in the children of alcoholic parents suggests the "wet brain" of alcoholism (see serous meningitis). Prenatal infection of the meninges and chronic ependymitis, due to any one of several infectious and toxic agencies, is to be recognized. Congenital hydrocephalus is more frequently

internal. The head may be tremendously enlarged; the fontanels remain open, or are closed by Wormian bones; the sutures spread widely apart, and Wormian bones may be interposed. The cerebral cortex is thinned, sometimes until it contains little or no recognizable gray matter; the white matter may be scarcely perceptible. The basal ganglia are flattened; the lateral and third ventricles, and the cerebral aqueduct (of Sylvius) are greatly dilated; the fourth and fifth ventricles are rarely dilated. The cerebellum may or may not be flattened greatly. Children in whom very slight hydrocephalus is present may attain normal or remarkable mentality; those in whom the hydrocephalus is sufficient to cause noticeable deformity of the skull, with injury to the brain, are mentally defective, and suffer from spastic paralysis, epileptic attacks, and malnutrition. The skull is rounded, rather than square, as in rickets, and the malnutrition of the body is not associated with bony fragility. Congenital hydrocephalus is frequently associated with spina bifida and with club foot, various slight bodily deformities, and stigmata of degeneracy of varying types.

Congenital external hydrocephalus is usually due to deformity of the brain, with normal skull size, or to abnormally large skull with normal brain. In the latter case previously existing internal hydrocephalus is suspected.

Acquired external hydrocephalus may appear at any time of life, but is most frequent during the first few months, or in senility. Wasting of the brain may leave a space, which is filled with liquid (vacuum dropsy), or there may be an increased secretion, probably inflammatory, of the endothelial cells.

Acquired internal hydrocephalus is due to meningitis or to brain tumor. Closure of the veins of Galen or of the foramina of Monro or of Majendie result in an acccumulation of fluid within the ventricles. Softening of the brain, epileptic attacks, various paralyses, and coma lead to death.

Diagnosis. The disease is suspected when enlargement of the head in children, or symptoms of increased intracranial pressure in adults, are associated with any of the etiological factors. Examination of the cerebrospinal fluid, and the use of the X-ray, particularly of the stereoscopic views of the skull, should make the diagnosis clear in most cases.

Treatment. Drainage of the fluid from lumbar puncture gives relief and may lead to recovery; pressure upon the skull, by straps and bandages, may lead to absorption of the fluid, and prevent its greater formation. Drainage of the cisterna, directly, is of doubtful value and of certain danger. Palliative measures include correction of cervical and upper thoracic lesions, and attention to the nutrition of the entire body. Children with heavy heads

should not be encouraged to try to hold the head up, but some support should always be given; the heavy head swinging around on the weak neck leads to various cervical and upper thoracic lesions, which still further embarrass the circulation and drainage of the cranial cavity.

AMAUROTIC FAMILY IDIOCY

Amaurotic family idiocy is a hereditary degenerative disease of the brain, characterized by progressive blindness and loss of mentality. Direct inheritance is, of course, impossible, but the disease attacks several members of the family in each generation. Normal children may be found in the same family, but all the children in one family who suffer from this disease show the first symptoms at about the same time.

Two types of this disease are recognized. One attacks infants and results in complete idiocy and death before the age of three years; in the other the onset is somewhat later, perhaps at about the fifth year, and death may be postponed until the tenth year, or rarely later.

The brain shows no changes on macroscopic examination as a rule, though sometimes irregularities in the convolutions occur. On microscopic examination degenerative processes in the cerebral neurons are observed. Swellings of the cell bodies are especially conspicuous. The granular layer of the retina shows the same changes; atrophy is usually present in retina and optic nerves.

The disease is limited almost exclusively to children of Jewish descent. One very typical case in the P. C. O. clinic had no history of Jewish ancestry.

No treatment is of real value. The case is hopeless from the beginning and the most that can be done is to keep the child comfortable for the remaining months of his life.

CEREBRAL PARALYSES OF CHILDREN

The paralyses which appear first in childhood are characterized by a number of factors which are not present, or are present in different degree, in the paralyses which first appear during adult life, or in old age. The etiology of children's paralyses is greatly different from that of adult paralysis, though both are based upon destruction of nerve cells.

Etiology. Paralysis in children may be due to any one of a large number of factors. Specific infection, as in anterior poliomyelitis, or the infectious agents present in most of the acute infectious diseases of childhood, may destroy the nerve centers in the cord or the brain. Trauma, after birth, at birth, or before birth, may injure the peripheral nerves, as in paralysis of the brachial plexus produced by pressure upon the shoulder in delivery. Long labor or awkwardly used forceps may injure the brain directly; long labor or asphyxia may lead to cerebral or meningeal hemorrhage. Jaundice may poison the nerve cells; marasmus may prevent brain development; the acute fevers may injure by overheat or by bacterial poisons. Premature birth may be associated with malnutrition, or the causes of the prematurity may affect the brain development. Before birth, the

nutrition may be below normal; maternal toxins may injure; direct trauma to the fetus through the mother's abdomen, is not rare; attempts at abortion may injure the fetal head and brain. (See Hemorrhage into Fetal Cord, C. A. Whiting.) Deformities of the nervous system cannot always be explained; these result in variable disturbances of function, often including more or less paralysis. Heredity is important. Syphilis in the parents stands first; next alcoholism is to be considered; neurotic inheritance, especially familial defects of body or mind, is frequently found; these hereditary qualities follow the laws of Mendel. Children of very old parents, and those born last in very large families, are more liable to nerve instability and paralysis. Children of the cachectic, those born during serious ill-health of the parents, are rather often paralytic.

Yet, after all, in very many cases, no efficient cause for the paralysis can be found, even upon the most thorough and careful investigation.

Pathology. Examination of the brains of such children shows various defects. Usually the cerebral and spinal injury is much more serious than would be expected from the symptoms observed. This is, no doubt, due to the fact that in childhood a great deal of compensatory action on the part of other developing nerve centers is possible.

Meningeal hemorrhage at birth is frequent; this may cause no recognizable symptoms in a large majority of cases. Large hemorrhages clot, are digested and ultimately are absorbed; or they become organized with scar-like formation of connective tissue and neuroglia; or these form a wall, within which the digested blood and nerve matter undergo further softening until a cyst filled with a clear and colorless liquid remains. These "hemorrhagic cysts" are often found in the brains of defective children. Thrombosis is not rare at about the time of birth; this causes infarction, perhaps softening, perhaps cyst formation.

Mal-development is indicated by variations in the size and form of the convolutions, by imperfect myelinization of tracts, especially the pyramidal tracts, and by the presence of atypical patches of gray matter scattered through the white matter, especially in the subcortical region. Various atypical relations and defects of the circulation are found.

The effects of hydrocephalus may also be found, in many cases (q. v.). Hereditary syphilis is indicated more vividly in the viscera and skin than in the brain itself; syphilitic lesions of the brain resemble the circulatory defects already mentioned.

Defects in the brain substance are frequent; these may be due to pressure of cysts, hemorrhages, etc., which may have been absorbed before death; or to hydrocephalus; or to abiotrophic changes; or to deformities of unknown cause.

Sclerosis due to neurogliar overgrowth is not uncommon. This may be diffuse or patchy, or may occur in hard, raised, knob-like elevations.

CEREBRAL HEMIPLEGIA OF CHILDREN

This disease is due to destruction of the nerve cells and fibers of the cerebrum, appears early in life, and causes paralysis of arm and leg, rarely face, upon the opposite side.

Etiology. The disease usually has its onset in the first three years of life; occasionally appears to be due to birth injury; and often follows an acute infectious disease, or a series of children's acute infections. Occasionally it seems to be primary. The onset and later history suggest infection, as in infantile paralysis, but no infectious agent has yet been isolated.

Diagnosis. The child shows no prodromal symptoms, save those of the contagious disease, when this is present; has high fever, vomiting, convulsions, and some torpor. As this passes away, one side of the body and face are found to be paralyzed first with relaxed muscles, which soon become spastic. Aphasia is usually present, no matter which side of the body is paralyzed, if the child has begun to talk. Within a few days, the aphasia passes, and the paralysis lessens. Recovery is not complete, and the arm is most seriously affected. Within a few weeks the extent of paralysis is fixed. The muscles which recover motor power show athetoid movements and spasmodic twitchings. Within a year or two, attacks of epilepsy occur; these are mild at first but increase in severity. With these, mental deterioration becomes evident, and this may go on to amentia. In some cases the epilepsy and mental deterioration do not occur, and the after life of the child is affected only very slightly by the paralysis. The growth of the paralyzed limbs is deficient, as in infantile paralysis. In some cases the extent of the paralysis is slight, but the mental effects are profound—paralysis of the intellect.

Treatment. After the paralysis is evident, the treatment should be directed first to securing the best possible circulation of the best possible blood through the brain, cord and muscles; next to the orthopedic correction of the deformities resulting from the paralysis. Special training is necessary for those children in whom epilepsy and mental deterioration are beginning; such children should be taught cleanly habits, and as much of good humor as is possible; but education, in the sense of "book learning," is mostly thrown away. Too urgent efforts toward teaching hasten the ultimate amentia. Sane, wholesome living, with such work as they can do cheerfully and easily, provide the education that is best for them.

Prognosis. The outlook is bad. For two or three years after the paralysis, the imminence of epilepsy must be recognized. The greater the athetoid movements the greater the danger of mental defect. Early cessation of athetoid or choreic movements is a good sign. When the epileptic attacks are frequent the prognosis is worse than when they occur at longer intervals, even though the less frequent attacks may be more severe. Mental deterioration is practically certain when epilepsy follows hemiplegia in children.

Life is rarely interrupted or shortened. After the climacteric the epileptic attacks may disappear; mentality is not usually modified by this.

DOUBLE HEMIPLEGIA

Occasionally this disease affects both sides of the body, and a condition resembling cerebral diplegia results. In double hemiplegia the onset is like that of cerebral hemiplegia, athetoid movements, epileptic attacks, etc., all resemble those of the unilateral type, and the mental deterioration is marked. In some cases, however, it may be quite impossible to distinguish between the paralysis due to disease (hemiplegia) and the paralysis due to defective development (Little's disease).

CHOREIC HEMI-PARESIS

This disease is probably related to cerebral hemiplegia. The paralytic stage is omitted, and choreic and athetoid movements of one half the body occur, as in the post-hemiplegic stage of the disease mentioned. The onset is more insidious, and it usually follows either severe acute infectious disease, or a very severe fright or nervous shock.

CEREBRAL DIPLEGIA

(Little's disease)

Cerebral diplegia is a paralysis due to defective development of the pyramidal tracts; it affects the legs more seriously than the arms, as a rule; and is especially characterized by rigidity of the skeletal muscles. Sometimes paraplegia alone is present, with characteristics of Little's disease.

Etiology. The disease is due to defective development; this, in turn, to asphyxia at birth, difficult labor, or to deficient nutrition before birth. Premature birth is mentioned; this may be the cause of the disease, or both disease and premature birth may be due to some earlier defect in development. Upper cervical lesions seem to be constant findings. These may often be secondary, and often appear to have been caused at birth. In any case, they usually seem to be important in the later history of the patient.

The pyramidal tracts, and sometimes the motor cortex, are found undeveloped; the tracts are nonmedullated or may be absent. This defect leaves the reflex arcs of the spinal cord uncontrolled; whence the rigidity.

Diagnosis. The entire motor system in its lower mechanism seems to be irritable; these children are thrown into spasms by sudden noises or lights. The mothers notice these spasmodic movements, and think the child is "trying so hard to understand, and to learn to control himself." Reflexes are increased; in some cases it is difficult to recognize the reflex response on account of the rigidity. It may be noticed that the child is too stiff, even when it is very small; when it should be holding up the head, this is not done; when it is time for it to sit alone, it is noticed that the

legs do not bend properly, and that the child stiffens iself out, instead of making the usual movements of the legs and arms. Sometimes the condition is not noticed until the child should begin to learn to walk. The rigidity of the legs causes them to be drawn together, so that they may cross; if the child is helped and the legs moved as in walking, or, in lighter cases, if he learns to walk, the legs cross, "scissors gait," and the walking movements are extremely stiff and slow. The muscles may be smaller than normal, or may hypertrophy; there is never any true atrophy, as in other types of children's paralysis.

The development of the tracts may simply be delayed, in which case the symptoms gradually disappear, and the child appears fairly normal at ten or twelve years of age. In most cases, however, the development never reaches the normal, and he is more or less crippled during life.

Mental development is defective, if the paralysis is marked; in this disease the defect in mentality seems to run a fairly parallel course with the paralysis. The vocal muscles are often affected, and this mutism increases the tendency to mental defect. When the face is not involved, the children are often quite intelligent in appearance, with friendly smiles and expressions such as normal children have under different circumstances of joy or grief.

Treatment. In order that every possible opportunity may be given the developmental powers of the nervous system, the constant care of the nurse and the physician is necessary. Treatment must be adapted to conditions as they arise; very important is the repeated correction of the bony lesions which are caused by the muscular tension. Once or twice each week the body must be examined, and very gentle manipulations given, which increase the mobility of the spinal column and the ribs. If these increase the rigidity, there is some error in technique; the result of each treatment should be to slightly diminish the muscular tension. Daily massage of the affected muscles, if gently done, is good; this gives the exercise in as nearly normal a way as is possible. Prolonged warm baths often relieve the rigidity; massage during the bath is especially useful.

Education should be wisely attempted. The child should be taught to rest, first; later very simple and easy movements should be given. At first only those movements which he can make easily should be given; later, these may be increased. It must be remembered that the brain centers are undeveloped, and that very little overuse may result in harmful tiring; constant watchfulness is necessary. The results which have been secured in a few cases as the result of this persistent treatment by osteopathic physicians, with the assistance of good nursing, is most encouraging.

Prognosis. The outlook is always grave, but in all cases of typical Little's disease there is a chance for the later development of the defective tracts.

LENTICULAR DISEASE

Disease of the corpora striata, and especially of the lenticular nucleus, has been described by several authors. The progressive softening associated with cirrhosis of the liver is of interest. The symptoms of lenticular disease include tremor, often intentional; spasticity of the skeletal muscles, usually bilateral and universal; excessive emotionalism; dysphagia; dysarthria but no true aphasia; difficulty in maintaining equilibrium but no true ataxia; and usually no reflex disturbances or sensory peculiarities. Athetoid movements and spasmodic actions may occur.

When associated with cirrhosis of the liver, recovery is not to be expected. Similar cases due to the presence of some peculiar toxin, which can be eliminated from the blood, may disappear with treatment.

Rarely, Babinski's sign, increased reflexes, and mental deterioration are present; these cases are not expected to recover.

BRAIN TUMOR

The term "brain tumor" is applied to any neoplasm or deposit of any kind which affects the brain in any way, either directly or by causing increased intracranial pressure. It is not usually possible to diagnose the variety of tumor until after death or operation.

Varieties. Tumors may originate from the membranes, the blood vessels, the neuroglia, or the connective tissues. They may originate in place, or as metastases.

Membranous tumors include enchondroma or osteoma, from the dura or the skull; psammona, usually from the neighborhood of the pineal body; lipoma, cholesteatoma, fibroma, myxoma, sarcoma, endothelioma, angioma, from the dura and the pia-arachnoid; carcinoma, practically always metastatic; tubercle and gumma, resulting from infections.

From the blood vessels arise aneurysms, tubercles, gummata, and angiomas, perhaps also endotheliomas. From the neuroglia arise gliomas. From the connective tissue grow fibroids and sarcomatous growths.

Tumors of extraneous origin include hydatids, rare in this country; cysterci; and many even more rare varieties. Brain cysts are usually caused by destruction and digestion of brain tissue or of extravasated blood. Hematoma is due to hemorrhage (See apoplexy).

Etiology. Tubercle is more common in children; glioma and sarcoma in youths; gumma in adults; carcinoma in late middle life; and others according to the nature of the infectious cause, or the nature of the neoplasm, or the opportunity for traumatic causes. Men are more frequently affected than women, probably on account of the incidence of gumma and the risk of trauma.

Diagnosis. The diagnosis of brain tumor is usually difficult, especially in the early stages, and when it does not affect the membranes. Symptoms are due partly to the increased intracranial pressure, and partly to the local irritation and destruction of brain tissue.

Headache is present only when the meninges are involved; since the brain substance is devoid of sensory nerves. Uncomfortable sensations may result from local irritation, however. Rarely, pain may be elicited on percussion, near the tumor, but this is subject to so many modifying influences that it is not satisfactory as a means of diagnosis. **Vertigo** is common; it is most marked in cerebellar disease. **Vomiting** is also most frequent and annoying in cerebellar tumor. It may be of the projectile type, or may be of the commoner variety; it bears no relation to the quality or the time of meals. **Choked disk** is almost invariable; interlacing of the limits of the color fields is a frequent ocular finding, considered of great value in more recent writings. **Mental** disturbances usually include dullness and apathy; rarely mania. Emotional or hysteroid attacks are frequent in some cases; increased emotional instability is usually present, especially when the frontal lobe or the basal ganglia are affected. **Constitutional** symptoms include emaciation, adiposity, peculiarities of the pulse and respiration, varying temperatures, most marked in basal ganglia disturbances, and pupillary changes.

The **focal** symptoms vary chiefly according to the fossa involved. Tumors of the anterior frontal region give only vague or elusive symptoms, chiefly mental disturbances of orientation and behavior. Tumors affecting the speech and writing centers, in the second and third frontal convolutions, destroy these powers. Tumors of the precentral area cause first, convulsive movements and epileptoid attacks, or Jacksonian epilepsy, later, paralysis; in the post-central convolution, paresthesias are followed by anesthesias, and these may be associated with convulsive or epileptoid attacks. Tumors of the right anterior frontal and a large part of the parietal and temporal lobes may cause no localizing symptoms. Lesions of the posterior area of the parietal lobe, especially on the left side, cause astereognosis; of the occipital lobes, especially the angular gyrus and cuneus, give varying light flashes during the irritative stage, if present, followed by homonymous bilateral hemianopsia. Lesions of the occipito-temporal region cause auditory and visual aphasia.

When the base of the brain is affected, ocular symptoms are marked; this is due to the effects upon the optic and the oculomotor nerves. The region of the sella turcica is most often affected of all tumors in the supratentorial region, and this leads to blindness, through pressure upon the optic chiasm. The pituitary gland is often concerned, with the symptoms of disease of that ductless gland. (q. v.)

Tumors affecting the **basal ganglia** are often associated with emotional instability, involuntary laughing or crying attacks, athetoid movements of the limbs, especially the fingers and arms, and peculiar hesitating speech. Lesions of any kind in the basal ganglia are apt to involve the internal capsule, with resulting widespread paralysis and usually more or less motor aphasia.

The **cerebellum** and **cerebello-pontine** angle are often the seat of tumors. Subtentorial tumors do not cause symptoms of increased intracranial pressure unless the tumor is of considerable size, or involves the aqueduct, thus leading to internal hydrocephalus. The localizing symptoms depend chiefly upon the effects upon the cranial nerves, unless the middle lobe of the cerebellum is affected. When this occurs, the symptoms progress rapidly. Cerebellar ataxia is marked; the Romberg sign is present; the skeletal muscles, especially those of the legs and back, are weakened and may either be atonic or hypertonic, sometimes variably on the two sides; nystagmus; various symptoms of cranial nerve involvement; strabismus; intense headache; severe vertigo, and often vomiting without digestive disturbance, are present.

Some part of the auditory tract is usually affected in cerebellar or cerebello-pontine tumor; tinnitus and deafness, and the symptoms of Meniere's disease are thus produced.

Tumors of the **pons**, if small, produce varying motor and sensory symptoms, according to the location and the function of the areas affected. Tumors of the peduncles cause cerebellar symptoms. Tumors invading the fourth ventricle, of slow growth, cause diabetes insipidus or mellitus, and sensory and motor, cardiac, respiratory and vasomotor symptoms, according to the areas affected. Death is not long delayed when the visceral symptoms appear.

Treatment. The most satisfactory treatment in most cases is surgical. Inoperable cases are more or less slow in development, according to the nature of the case. Tubercle and gumma are to be treated constitutionally. Decompression operations may prolong comfortable existence for weeks or months; occasionally decompression permits later reparative surgery. In any case of increased intracranial pressure, measures which lower the systemic blood pressure may give temporary relief.

SUPPURATIVE ENCEPHALITIS

(Abscess of the brain)

Etiology. Direct injury is the most frequent cause; pyemia causes multiple small foci; extension of infection from the mastoid cells, the nasal, and other neighboring areas is sometimes causative; rarely circulatory disturbances, due to any of the usual causes, is followed by infection and abscess. More often such conditions are followed by softening by autolytic enzymes than by infection.

Diagnosis. The symptoms are those of tumor, plus those of suppuration—these include chills and fever, leucocytosis, indicanuria, and often the symptoms of the primary infection, as typhoid, pneumonia, or endocarditis. Retinal congestion is usually marked, but choked disk is rare. The symptoms progress by extension along the line of least resistance, rather than by increasing intensity in one place, as in tumor. Multiple abscesses often follow, as in one P. C. O. specimen, in which an unsuspected temporal abscess underlay an old bullet cyst, with multiple abscesses of microscopic size over the cerebral and cerebellar cortices; the apparent cause of death was the rupture of the abscess into the ventricle.

Treatment. When the abscess can be localized, the pus may be evacuated, and recovery follow. When diagnosis is doubtful, and the pus cannot be localized, systemic treatment is to be employed; this includes correction of the cervical and occipital lesions, promotion of nutrition and elimination, liquid diet, with free drinking of water; palliative treatment for the symptoms as they appear, and constant watchfulness in order that surgical interference may be based upon as exact knowledge as possible.

Prognosis. When evacuation of the pus is not surgically secured, the abscess may break into the nasal passage, with immediate relief and recovery; into a venous sinus, with septicemia and ultimately death, or into a cerebral ventricle, with sudden death. In all cases the prognosis for life is serious, and recovery is apt to be slow and incomplete, in the most favorable circumstances.

CHAPTER XXXVII

FUNCTIONAL NEUROSES

HYSTERIA

Hysteria is a functional disease of the nervous system, characterized chiefly by various constant disturbances of sensation and motion and by occasional exacerbations of these disturbances or by convulsive attacks.

Pathogenesis. No morbid anatomy has been described for hysteria, though several investigators have reported finding aberrant masses of gray matter and various slight irregularities in the form of the cerebral convolutions. It is difficult to understand how any single disease-producing factor can possibly be responsible for phenomena so varied and apparently so antagonistic as are the symptoms of hysteria. The most satisfactory theory depends upon a recognition of the nature of the inhibitions under normal conditions. Minimal stimuli repeated at intervals too great to produce summation may so affect the nerve centers as to prolong the refractory periods into a constant inhibitory effect. In hysteria the liminal value of the nerve centers is so greatly increased that stimuli which ordinarily are inhibitory are now of no effect whatever, while stimuli which should ordinarily produce marked effects in consciousness or in bodily activity are now reduced to a level of inhibitory reactions. The specific symptoms in each case depend upon the location of the nerve centers affected; this, in turn, depends upon the etiological factors present in each case, and upon the physiological condition of the different centers at the time of irritation.

Etiology. By far the most important cause of hysteria lies in **heredity.** Hysteria is one of a group of neuroses which are interchangeable in inheritance; that is, if one parent has migraine and another is addicted to the use of drugs or alcohol, the children may be hysteric or neurasthenic or epileptic or subject to any one of several forms of insanity. These interchangeable neuroses follow Mendel's law quite closely.

The next most important factor in causing hysteria is a bad **education.** The only child is especially liable to hysteria, as is the child with brothers and sisters much older or, indeed, any child unduly pampered by the other members of the family. As a result, consideration for others has no place, or only a very small place, in their ideas. Physiologically, it may be said that the neuronic relations of the left frontal lobes are excessively developed at the expense of general cerebral balance. As the result of this inflated egocentric psychology, there is a tendency for every sensory impulse to be immediately transferred into a personal and emotional expression, either word or deed. Given the unbalanced nervous control, exciting causes which would not interfere to any great extent with the health of a normal individual may

have very profound and serious effects. The repressed emotions and wishes so often exaggerated by the followers of Freud occur before puberty, as a general thing.

The stress of modern education is an important factor especially in girls. High school buildings are often badly arranged. Class rooms scattered from the basement to the fourth story and connected by steep and winding stairs, crowded and unpleasant toilet and dressing rooms, together with the need for too much home work, are certainly enough to interfere with the normal development of adolescent nerve centers, but when these are further complicated by the emotional storms incident to fraternity and sorority associations, social affairs, music, art, and dancing, the problem becomes too complex for any ordinary brain to meet in an efficient manner. Girls have the worst of all this; boys usually have outdoor sports, girls may have these but household cares, needle work, and devotion to personal appearance add further complications.

During adolescence, the first love affairs, religious experiences, and more or less freakish ambitions may initiate hysteria. The exciting cause of any attack is usually absurdly trivial. It is indeed "the last straw that breaks the camel's back" in these cases. Recovery from any given attack may also be secured by apparently trivial causes.

Spinal Conditions. The hysterical spine is usually very irregular. No two are alike. Lesions of the occiput, atlas and axis are almost universal. Lesions of the mandible are frequent. Rib lesions are often associated with improper habits of breathing. It is very rare to find a hysterical patient whose respiratory muscles are free and whose respiratory excursion exceeds one-half inch in quiet breathing. Lesions of the coccyx are usually present. Many cases called hysterical coccygodinia are really due to misplaced coccyx and not to the hysteria. Given the hysterical temperament, the symptoms which cause greatest distress are frequently localized through the influence of bony lesions which may be the result either of accident or of reflex muscular contractions.

Diagnosis. True hysteria is rather rare. Hysterical symptoms associated with organic diseases almost anywhere in the body are so common that it is rather rare to find a patient who has been sick for months or years whose symptoms are not somewhat modified by unbalanced neuronic activity. A diagnosis of hysteria alone can only be made when every organic disease has been found to be absent. The symptoms of hysteria are so varied that this fact itself is of a certain value in diagnosis, though it must constantly be remembered that many organic diseases of the nervous system as well as of many visceral organs are associated with hysterical symptoms.

Hysterical phenomena are classified according to the structures chiefly affected.

The **memory** of the hysterical patient shows many lacunæ; doubtless this accounts for the many variations in behavior. Multiple personality depends largely upon this state, as does the existence of split personalities, buried complexes, and the various peculiar antipathies of these patients.

Variations in **consciousness** include trances, twilight states, somnambulism and sleep-like states which may continue for months at a time. Remarkable visions are often associated with these lapses of consciousness.

Hysterical **paralysis** may have a sudden onset following some emotional shock or strain, or it may begin insidiously with gradually increasing weakness of certain muscle groups. The paralysis may be either flaccid or spastic. It may continue for many years until the limbs involved become fixed by contractures and by changes in the articular tissues. The muscles involved do not show atrophy or any marked electrical variations, and reflex action may be increased, diminished or lost. Paralytic muscular contractions disappear under anesthesia and during sleep. It is rare for the contractions to persist through accident, especially if this persistance should be about to result in fracture or other serious injury to the body.

Hysteric paralysis often disappears under the influence of shock. Attempts have been made to cure the condition by providing apparently accidental catastrophes; as, for example, leaving a patient in the house alone and then providing a strong smell of smoke. It is needless to say that attempts of this sort usually result disastrously.

Disturbed **cutaneous sensations** are almost invariably present and are usually rather strictly localized. Modified sensations are more common than total anesthesia. This accounts for the peculiar response which such patients make to tests of sensation; for example, if a patient is blind-folded and is told to tell when she feels a touch upon any part of the body, as the arms, she may respond "Yes" to a touch upon the right arm and "No" to a touch upon the left arm. The fact that the answer "No" is made to a touch upon the left arm proves, of course, that it was perceived in some way. The disturbed cutaneous sensations do not follow the distribution of the nerve trunks, nor of the spinal segments. They do correspond rather closely to the cerebral localization of the sensations affected. Disturbances of touch and pain sensations are the most frequent and most marked. Disturbances of muscle sense, temperature sense and of the peculiar sensations produced by the electrical current are less frequent and the tests for these sensations are decidedly unsatisfactory.

Disturbances of **smell** and **taste** are usually present. Odors which have been previously considered pleasant are likely to become obnoxious. Hyperosmia is not rare. One patient in the P. C. O. clinic was able to tell whether any one of her acquaintances had been in a room within the last hour or more, by the personal odor. She was not able to follow odors in the open air. Anosmia occasionally occurs. Perverted sense of taste is less frequently found and it is usually a disturbance of the olfactory sense, rather than of taste, that is present. When a persistent sweet taste or bitter taste is complained of some visceral disease should be suspeced. Occasionally, however, these taste perversions are present as hysterical phenomena.

Deafness and tinnitus are not frequent. Occasionally hallucinations of hearing occur. **Visual** disturbances are usually present. Quivering lights, flashing alternations of light and darkness, diplopia and other transient phenomena are very frequent. Unilateral blindness and amblyopia occur at intervals. Total blindness of sudden onset may last for minutes or for years, and may disappear suddenly. Contraction of the visual fields and especially of the color fields is so constant a finding that it is of diagnostic value. The retraction of the color fields is usually concentric, though often the blue-yellow field retracts within the red-green field. The visual field may be so retracted that the patients seem to be looking through a tube—"tubular vision." This condition is rarely suspected until the examination is made.

Disturbances of **visceral** sensations are variable. Lack of appetite, voracity, lack of thirst, polydipsia, very severe causeless visceral pain, anesthesia in severe visceral diseases ordinarily painful, sexual frigidity, nymphomania or satyriasis, may be found in different individuals or in the same individual at different times.

The **blood** in hysterical patients is usually good. The hemoglobin, erythrocyte count, and leucocyte count are usually practically normal. The individual cells, however, frequently show the characteristics of immature blood. A few nucleated red cells and immature forms of the white cells are found in those cases in which the hereditary or congenital factor is pronounced.

The examination of the **urine** shows usually a diminished excretion of solids. Phosphates are frequently increased and calcium oxalate crystals are often present. After a hysterical crisis large amounts of urine of low specific gravity are usually voided. This serves to distinguish certain doubtful cases of hysteria from epilepsy.

Crises. The fits or convulsive attacks, called crises, described with so great detail by Charcot and other of the Salpetriere school, are rarely found in this country. These are usually precipitated by some emotion or fatigue but may be self-originating. They

rarely resemble epileptic fits quite closely. They usually last longer than epileptic attacks; the movements have a purposive appearance and usually imitate the expression of some profound emotion or passion. The hysterical patient rarely bites the tongue, injures the body in any way, froths at the mouth or passes urine and feces involuntarily. The hysterical patient is usually bright and appears to feel very well after the attack. Very different conditions are associated with the epileptic fit. (q. v.)

Pseudohydrophobia (Lyssophobia) occurs in neurotic persons after having been bitten by a dog, or even after having been frightened by a dog or any other animal. The use of the term "mad-dog" has caused virulence to be imputed to the saliva of any infuriated animal even though perfectly healthy. The symptoms of hydrophobia are greatly exaggerated in imagination. Some weeks, months, or years after the fright, usually upon some unpleasant occurrence, the patient begins to complain of feeling ill. The site of the wound may become painful, occasionally even reddened slightly. The patient often bites at the old wound, or at the site of an imagined wound, until it becomes decidedly sore, perhaps infected. A horror of water is urgent, and the patient struggles, bites and snaps like a dog, and imitates whatever symptoms of hydrophobia he may have heard of, or imagined. Only under unusual conditions is there difficulty in making a differential diagnosis between this form of hysteria and true hydrophobia.

Pseudomeningitis. Spinal meningitis lends itself readily to imitation by patients with the hysterical love of the spectacular. It is found most often in girls and young women; especially if they consider themselves hopelessly in love. A death from "brain fever" sounds delightfully tragic; and their neurotic symptoms lead them really to believe themselves seriously ill. The hysterical muscular contractions lead easily to opisthotonos; nausea and vomiting are easily encouraged; the fanciful or sportive or maudlin delirium of true meningitis is not unlike the natural expression of the hysterical love-lorn maiden. The hysteria is easily recognized unless the diagnosis of meningitis is accepted by an unsuspicious physician.

Treatment. The essential thing, in treatment, is to provide, first a normal circulation of normal blood through the brain, second, a normal stream of nerve impulses leading to a reëducation of the brain centers. The exact methods to be employed vary for each individual, and must be based upon a physical and mental examination.

The spinal and costal lesions should be corrected by means of movements which do not add to the irritation of the conditions already present. Care must be taken in giving the osteopathic

treatment to avoid securing too great relaxation of the spinal ligaments. It is necessary also to avoid the "treatment habit." Hysterical patients too often find themselves impressed with the need for corrective treatment and go from one doctor to another, constantly seeking heavier or more gentle or more efficient treatments.

Patients who are emaciated, weak or anemic may have the rest cure such as is found beneficial in neurasthenia. (q. v.) Those whose bodies show good nutrition and in whom the blood pressure is low, are best benefited by being sent out doors and by being given exercises which lead to the development of the muscles.

The next most important factor in the treatment of hysterical patients is found in reëducation. It is necessary that the whole trend of thought should be changed from the egocentric to the altruistic. This is not best done by attempting to appeal to the generous emotions, since this leads rather to further self-pity, or to the development of the "martyr" idea. Any sort of fad which leads to out-of-door work may be encouraged. Change of scene is frequently recommended; to be efficient it should be complete. The one who seeks a change of scene, carrying with her her maid, her pet dog and everything which has kept her mind wrapped in cotton wool through all her life long is not likely to find a change of scene in any proper sense, whether she goes to Greenland or to India.

In those cases in whom some emotional shock has been the original cause of the disease, some methods of **psychoanalysis** may be employed. There is no question that some hysterias do rest upon a basis of repressed feelings, either sexual, religious, or other. In such cases the complete exposure and discussion of the varied complexes frequently results in recovery which appears almost unbelievable. On the other hand patients whose condition rests upon some other etiological factor are too often put through a series of unpleasant discussions, whose only effect is to transfer the symptoms into the psycho-sexual sphere. For this and other reasons it is best that these methods should first be employed by the physician in charge of the patient, and without explaining the reason for the various discussions. Much more frankness is secured, from most patients, by asking questions, either directly or indirectly, after the treatment has been given, while the patient still lies upon the table. The variations in pulse rate and in blood pressure are valuable in recognizing significant statements or evasions. Psychoanalytic methods require much time and a certain amount of skill and sympathy. When any physician finds himself unable to give these, or when his best efforts fail, it is much better to send the patient to a professional psychoanalyst for that special line of educational work. The correction of the structural perversions can be done, either before, during, or after the analysis of the mental content. In any case, the structural perversions must

be corrected, else later attacks, though of different symptoms, may be expected.

During a **crisis,** the patient should usually be left alone. When the symptoms are very severe, some treatment may be required; this is palliative and symptomatic. Very slow and strong spinal extension is often useful. A neutral bath, continued for one or several hours, may relieve convulsions. Rest in a dark room is generally the very best thing. No atmosphere of excitement is permissible; nor is any sense of punishment to be manifested. Scoldings may avert an attack, in the very beginning, in mild cases, but these usually exacerbate the fit and intensify the neurosis. Commonly following an emotional storm the patient feels decidedly refreshed.

It is rarely useful to treat symptoms directly, unless there is some organic disease present. Bony lesions may be responsible for various functional diseases of the various organs; correction of the lesions, with or without an explanation of the desired effects, is all that is needed for these conditions. Functional diseases of certain organs, especially gastro-intestinal or genito-urinary, may be due to the hysteria alone; in such cases, the less said about the disease, after one discussion and explanation, the better. Especially in neurotic girls and unhappily married women should the discussion and treatment of the pelvic organs be evaded. When serious organic disease is present the condition should be properly treated; when the pelvic conditions are secondary to the neurosis, local treatment should be postponed until better nervous control is reëstablished.

Sensory disturbances usually need no treatment; when they are severe, massage, counter-irritation, electricity, or sun-baths, may be recommended. Motor disturbances also may be disregarded, unless pain or great inconvenience is caused. Bandages, adhesive straps, electricity, warm baths, may give temporary relief. Orthopedic measures usually do more harm than good; after the neurotic condition has been completely overcome, if structural deformities persist, orthopedic surgery may be necessary.

It is too frequently the case that doctors and nurses, as well as the members of the family, consider hysteria merely another form of malingering. They think that the hysteric patient could be all right if she would, which may be true, but it is also true that she "can't would." No doctor should assume charge of a case of hysteria unless he can deal with the condition in just the same impersonal and scientific and kindly way that he would use in dealing with a patient who suffers from any other disease. The pain in hysteria is as severe and distressing as is the pain in any other disease; the paralysis is as inevitable as it is in poliomyelitis. Unless one is willing to give attention and thought to the disease in a professional and scientific manner he should not attempt to

care for these cases, but should refer them to some other physician who is willing to treat the case properly.

Prognosis. With proper care, the prognosis for recovery is good so far as the symptoms are concerned. Inasmuch as the condition rests upon a constitutional foundation, it is evident that this must persist throughout life, but with ordinary care and good hygienic living these patients should complete long, happy, useful lives.

THE NEURASTHENIC STATES

The term neurasthenia is somewhat vaguely applied to a functional nervous disease which is characterized by symptoms of fatigue of the certain groups of nerve centers.

Pathology. The structural changes are at present extremely doubtful. Chromatolysis of the motor neurons has been described. Functionally there is an increased irritability and increased fatigability of the nerve centers. The muscles are not fatigued as is indicated from the manner in which they react to direct electrical stimulation. The reflexes are first increased but speedily diminished.

Etiology. Both predisposing and exciting causes must be recognized. Of the first by far the most important is **heredity**; it is very rare to find a typical neurasthenic in a family whose ancestors are all nervously sound. Alcoholism, syphilis, tuberculosis, extreme youth, old age, drug addictions, migraine, hysteria often occur in the parents or are characteristic of the ancestry of the neurasthenic patient. When these conditions are variously combined in both parents or grandparents only a normal life can prevent the occurrence of the neurasthenia or other neurosis in the children. Other **predisposing** causes are the use of stimulants, and unhygienic living; overwork has been greatly exaggerated as the cause of neurasthenia. It seems certain that no amount of mental overwork or responsibility causes neurasthenic states in an individual who has proper hours of sleep and of outdoor exercise and whose food is that best adapted to his manner of living; in other words work which does not interfere with hygiene probably is never excessive. The overwork which is associated with an undue sense of responsibility, which causes unrest and worry often leads to the use of stimulants. Work which is never satisfactorily completed causes a troublesome sense of inefficiency, and this in turn often leads to the use of sedatives and stimulating drugs.

"It must not be forgotten that it is not the work which injures; probably persons injured by overwork are rarely found; but it is the lack of sleep, out-of-door exercise, the overeating, either of overly rich food or the food of some faddist, it is the lack of proper hygienic habits which bring the trouble. The person who has good food, good air, good exercise and a body whose parts are properly related, who eats, sleeps, laughs, and plays enough, has no time for overwork and is not apt to overworry."—L. Burns.

"Different cases present different lesions, and no typical lesion may be described for all cases, but certain lesions are common, in the various types; as cervical and upper dorsal in the cerebral; mid-dorsal and ribs, in the gastric; lower dorsal, ribs, and upper lumbar, in the intestinal. Upper cervical and upper dorsal lesions seem to be most constant in the spinal and sympathetic variety. The lesion of third and fourth cervical to the right (reported by Hazzard and also by McConnell and Teall) seems responsible in many of my own cases for the cerebral symptoms, except, possibly, the vertigo which may, and generally does, result from atlas displacement."—C. A. Champlin.

"The neurasthenic complexes are formed by education, and like useful or normal complexes, such as those of motion in piano playing, require time and repetition in formation. The basis of these complexes may be formed without intention, by accident, or by environment. The conditions favoring its formation are repeated frequently and enlarged upon until it is often very hard to recognize the basic causative factor. If this was brought about by education, it requires reëducation to show the patient wherein the misconception of his condition started. If we can trace for a patient from the beginning the successive steps that have led to his present condition, we have gained in the understanding of his case and in starting him on the road to recovery. But we must regard such a case as disease and treat it as such, and see that the family looks upon the patient as sick, not, as is frequently said, suffering from lack of self-control."—C. E. Farnum.

"In run-down neurasthenic patients, anemic or not, the blood pressure is apt to be too high or low. Its level will determine absolutely the dietetic and hygienic treatment; its reaction will determine the osteopathic treatment, frequency, and severity."—L. G. Robb.

Bony Lesions. The characteristic neurasthenic spine is rigid and flat through its entire extent. The various lateral subluxations of individual vertebræ and long slight curves and rotations may be present, but the flatness is characteristic. The irregular cervical spinal column is frequently important in the cerebral or mental types. Coccygeal lesions and innominate lesions are most common in the sexual neurasthenics; depressed lower ribs are practically constant, the eleventh rib stands out and the twelfth rib usually lies within the iliac crest.

Areas of hypersensitiveness are variable in location and in degree. Often the tissues along the spinal column and the angles of the ribs are practically anesthetic at the first examination, becoming increasingly hyperesthetic as the increased mobility, resulting from the treatment, permits the more normal activity of the spinal centers concerned in carrying sensations of heat, cold and pain upward to the brain. There is no question that the spinal condition is an important etiological factor in the neurasthenic states.

Diagnosis. The onset of the disease is usually gradual; it begins with a tendency to fatigue more easily; there is some insomnia and irritability of temper. Very frequently these conditions follow a prodromal period of unusually strenuous living, during which time the patient sleeps less and indulges himself more than is proper in pursuing ambition or pleasure. The fatigability, insomnia and irritability grow worse, a sense of pressure in various

parts of the body is frequent, this gives rise to the sensation called "stocking sensation," "glove sensation," rarely "girdle sensation" and the "lead-cap headache." Sensations geometrically outlined are very apt to be neurasthenic. The sense of fatigue is worse in the morning. During the day, meeting other people and amid the emergencies of work and play, the patient feels more and more able to meet the demands of living. By night he is often very much alive and ready for anything except sleep.

The insomnia is characteristic. The depth of normal sleep is very profound for the first two hours or so of the night, the level then returns almost to the waking line and remains fairly constant until early morning, the depth of sleep increases at this time usually to a point about one half that of the earlier depth and this terminates by awaking. The neurasthenic has only the two "drops" for his sleep period; those hours which a normal person passes in shallow sleep the neurasthenic passes in wakefulness. This daily history is in itself almost pathognomonic. Besides the general symptoms just given neurasthenic patients are subject to various other symptoms, referable to different organs.

In the **gastric type** the patient complains profoundly of digestive disturbances. It is extremely difficult to make a certain diagnosis of this form of neurasthenia because of the difficulty of eliminating organic stomach disease. Gastrectasis is eliminated with difficulty because the neurasthenic has usually relaxed and atonic abdominal and gastric muscular walls. Accumulations of gas within the stomach are quite constant. X-ray pictures taken during the digestion of a contrast meal give the most satisfactory diagnostic information. Reflex muscular contraction, bony lesions and hyperesthetic areas are to be found from the fifth to the tenth thoracic vertebræ and the corresponding costal areas.

Cardiac Type: Vasomotor Type. Neurasthenic disturbances of the circulation are characterized by a weakness in the heart's beat, low blood pressure, slow circulation, cold hands and feet and pallor of the conjunctivæ and mucous membranes. This pallor may be so marked as to suggest profound anemia; the examination of the blood, however, easily eliminates any form of anemia. It is less easy to make a satisfactory diagnosis of the cardiac condition. The weakened muscle walls and the diminished force of systole are probably responsible for the hemic murmurs so frequently found in neurasthenics. The hypersensitiveness and bony lesions are found in the second to the fourth thoracic region and in the upper cervical vertebræ.

Sexual Type. This is one of the most common types of neurasthenia among men. Sexual overactivity and sexual perversions are certainly factors in this form of neurasthenia but their place in etiology has been very greatly overestimated. It is far more

frequently the case that the lack of self-control associated with the neurasthenic state is responsible for the sexual wrongdoing than that these initiate the neurasthenic state. There is no question, however, that sexual perversions and excesses do increase the neurasthenic symptoms most profoundly, especially in men. In women the injury seems to be more often due to unrecognized desire rather than as the result of overindulgence. The evil effects of day-dreaming, romantic literature, and all of the emotionally morbid surroundings to which women of idle lives are often addicted, are extremely injurious to the nerve centers of the lumbo-sacral enlargement. Lesions of the lumbar vertebræ, the innominates and coccyx are almost universal in these cases. The neurasthenic state as well as the bony lesions mentioned exert a harmful effect upon the pelvic organs. Lax muscles and ligaments, congested ovaries, heavy, soft uterus, are usually present in neurasthenic women. When the infantile uterus and poorly developed sexual organs are found in women hysteria is rather more apt to occur than neurasthenia. In men sexual desire is sometimes increased, premature erections and emissions are frequent, satisfactory intercourse is often impossible, and in both sexes intercourse is frequently followed by profound exhaustion and distaste. Marital unhappiness produced by this condition frequently adds to the general nervous malfunction.

Cerebral Type: Psychasthenia. This type sometimes exists with very little sign of neurasthenia as affecting the rest of the body. It is characterized by a mental exhaustion, if the expression may be permitted. The patient realizes his condition but finds himself unable to maintain the interest and the effort necessary to enable him to do the work to which he is accustomed or which he desires to do. The mental state is frequently suggestive of atavistic phenomena. **Kleptomania,** or the passion for annexing and secreting objects for which the patient cannot possibly have any legitimate use is one of these. **Mysophobia** or the horror of contamination is probably not to be included as an atavistic phenomenon. **Agoraphobia,** the fear of open spaces; **claustrophobia,** the fear of closed in places; **zoophilia** or the inordinate love of animals, are among the most common phenomena whose nature suggests more or less vividly the past experience of the race.

Laboratory Findings. These are more helpful in showing the condition of the individual patient than in naming the disease. The amount of **urine** varies; the total excretion of solids is usually low, a retention of uric acid and other purins is very common. Calcium oxalate is present in a great many cases and it indicates the deficient oxidation present occurring in the body. Phosphaturia is frequently present; the examination of urine is of value in

the differential diagnosis between the nervous symptoms due to early diabetes or nephritis and those of neurasthenia.

The **blood** is characteristic. The color index is low; this is due either to an increase in the number of red cells or to a decrease in the hemoglobin percentage. The red cells usually show irregularities in size, shape and staining reactions. Among the white cells the most conspicuous change is the relative increase in the eosinophiles, this is constant and is of value in diagnosis. The blood changes appear to be due to the lack of circulation through the red bone marrow, especially of the ribs. Neurasthenics rarely breathe properly, the respiratory excursion is invariably diminished. Blood examination is useful in making the differential diagnosis between the nervous states of secondary anemia, pernicious anemia, chlorosis, the leukemias, all of which have more or less profound nervous disturbances, and true neurasthenia.

Treatment. The correction of bony lesions as found is important in this disease but this is rarely sufficient to provide all conditions necessary for recovery. Improved nutrition, improved circulation through the spinal cord, improved functional activity of the nerve cells, all of which result from the increased mobility of the spinal column and the raising of the ribs give the best possible conditions for recovery on the part of the inefficient spinal neurons. On the other hand if the etiological factors are constantly throwing greater burdens upon these centers, if the circulating blood is constantly filled with the toxins resulting from bad diet, poor breathing, and insufficient water intake, it is evident that the manipulation alone is not the whole of osteopathic treatment in such cases.

In suitable cases a modified form of the Weir-Mitchell rest cure with a full milk diet works wonders. Rest and increased nutrition of the nerve centers is the whole matter of treatment. Increased proteids, increased fats and increased water intake are extremely important matters. Starches and sugars need not be increased. The raw vegetables in the form of salads should be added freely to the diet.

In many cases it is better to lessen the amount of work but not to take it away altogether. Very frequently the relief from responsibility is all that is necessary. If the work performed is kept within the limit of that which can be satisfactorily completed day by day, leaving time for a few hours of outdoor exercise, most patients do better than when they suffer the humiliation and discomfort of being removed completely from work which has heretofore occupied a very great part of the waking hours.

In other cases a complete change of scene is necessary; this is especially true when the patient has no great amount of interest

in the work which he has been doing. The change of scene must be truly a change. The patient who goes to another climate carrying with him his family or servants, who eats the same food, affects the same dissipation, drinks the same drinks and stays up to the same unholy hours at night, finds no change of scene even though he should travel from the equator to the pole. The great value of a visit to the various hot springs and health resorts comes as much from the fact that change in occupation and thought and a physiological division of the day are insisted upon as from any other factors. The patient must be taught that his mental attitude is an important factor in the insomnia, and, to a certain extent, in the other symptoms.

Prognosis. Neurasthenia rarely terminates life either directly or indirectly; indeed, the disease itself lessens the exposure to the ordinary dangers of living. A recovery from any given attack is certain if the patient will willingly obey directions and receive the proper treatment for his condition. Future attacks are to be avoided only by avoiding the causes as mentioned above. Neurasthenics and those who have suffered from neurasthenic attacks should not marry other neurasthenics. The mildly neurasthenic may safely marry persons with sound and wholesome nervous systems. The children of such marriages are often all that is to be desired in the way of health and usefulness.

TRAUMATIC NEUROSES

The term "traumatic neuroses" is applied to those disturbances of the nervous system following shocks or accidents, but not associated with gross lesions of the nerves, the brain or the spinal cord. The shock is usually associated with some concussion and with very profound fright or excitement. Frequently the symptoms do not appear for some hours or even days after the accident. The symptoms are sometimes very vague and may present an extremely complicated diagnostic picture. Disturbed personality such as characterizes neurasthenic and hysterical states, together with various sensory and motor disturbances, contractures and paralyses may be present. The complicated nature of the effects produced by accidents often leads to a suspicion of bad faith and malingering. There is no question that people who have been in railroad or other accidents often deliberately magnify the results of the injuries in the hope of securing larger amounts of money in payment for the damage they have sustained. Such attempts rarely deceive any earnest investigator. It is necessary that the investigation of these cases shall be pursued in such a way as to protect both the victim and also the company or individual who has the duty of paying for the damages inflicted; in other words, it is the duty of

the physician, in such cases, to seek for the actual truth of the condition. If he makes the examination in a frank and kindly manner, it is usually not difficult to draw the line between even vague and complicated nervous results of accident and the awkward attempts at deceit which are usually made by dishonest persons.

The very fact of litigation causes pronounced nervous disturbances of a functional nature. Railroads and other companies responsible for accidents frequently have the date of trial postponed in the hope that the recovery of the injured persons may lessen the sense of injury. It is true also that with the lapse of time the force of public opinion is considerably diminished. All of this works for harm to the patient. In order that the most speedy recovery may be made from such shocks, the financial aspect of the case should be settled as speedily as possible. The fact that recovery occurs very quickly after the compromise has been effected, or the court procedures have been fully completed, is often held as evidence that the patient was merely a pretender. This is a serious injustice, since it is often the relief from worry that removes the last obstacle to recovery.

Pathology. In many cases in which death has followed some intercurrent disease after such shocks, the examination of the brain and cord shows slight capillary hemorrhages. Concussion certainly causes a tremendous shock to sensory neurons. The effects of these may be very serious but are usually transient. The wrenches of the bony tissues of the body produced by accidents are certainly responsible for a large proportion of the symptoms found in the traumatic neuroses. Lesions thus produced are overlooked by the ordinary doctor of medicine, but are none the less important in etiology. Lesions of the occiput and upper cervical region cause various disturbances in mentality. These may vary from a slight loss of self-control to severe acute confusional insanity. Lesions of the upper thoracic region cause disturbances of the heart action and various vascular disturbances. Lesions of the lower thoracic region affect the abdominal viscera, while lesions of the lower cervical or of the lumbar spine may be responsible for paralysis and for sensory disturbances. Lesions affecting the dorso-lumbar area cause disturbances of the circulation and secretion of the kidneys and suprarenals. Cervical or upper dorsal lesions may cause various disturbances of the eyes and of the ears. The recognition of these lesions as etiological features in functional diseases of the organs named above and their correction should go far toward promoting recovery more speedily in these traumatic neuroses.

Treatment. Bony lesions as found are to be corrected as speedily as is possible under the varying circumstances. After this, the most important factor is termination of litigation. The treatment for neurasthenia is indicated. (q. v.)

MIGRAINE

(Hemicrania; periodic sick headache)

Migraine is a functional disease of the nervous system, characterized by periodical attacks of intense unilateral headache, visual

disturbances, and usually nausea and vomiting. No pathological findings have ever been reported for this disease.

Etiology. Migraine is one of the neuroses which are interchangeable in inheritance. The inheritance of migraine usually follows the female line. The attack may begin as early as the first or second year of life, though the most frequent age of onset is ten to fifteen years. The attacks appear at intervals of a few days to a few months until the climacteric has been passed, when they usually disappear.

Bony lesions, chiefly of the upper cervical region, are important, even in hereditary cases. Lesions involving the splanchnic area are often present. In women in whom the attack occurs at the menstrual period, lesions of the lumbar spine, the innominates or coccyx are frequently found. Often the attacks cease during pregnancy and lactation.

Eye strain, hardened ear wax, adenoids, and other causes of peripheral nerve irritation probably help in promoting the nervous instability. Patients themselves usually consider overwork and the use of improper foods an important factor in precipitating an attack. The relation of migraine to epilepsy has been variously discussed. Since absolutely nothing is known as to the real nature of either migraine or epilepsy, such discussions do not lead to any very useful results.

Diagnosis. Migraine is usually recognized upon the symptoms and history. The attacks begin most frequently with visual disturbances—flickering lights or flashes, floating spots, dim vision, and diplopia. This is followed (sometimes preceded) by various sensations of vertigo, dizziness, nausea, and dull headache. Very sharp pain, usually frontal, practically always unilateral, gives the name "hemicrania" to the disease. This may be so severe as to cause unconsciousness; it is very obstinate to the usual analgesic drugs, and attempts to relieve the paroxysms by these are important causes of drug addictions. The pain is of a neuralgic type; sometimes hot, sometimes cold, applications give relief. After a variable time, usually a few hours, vomiting becomes free; the stomach contents are first vomited, then bile; and usually when much bile has been vomited the pain is relieved, and the patient is left comfortable but very weak and listless. A long sleep, sometimes twenty hours or more, usually terminates the attack and gives the necessary rest. In the intervals the patient is in good health, so far as the migraine is concerned.

Treatment. During an attack it is rarely possible to do more than secure relief. Occasionally extension and careful correction of muscular or bony lesions is possible, and this may give marked relief. This is especially true at the onset of the attack. When manipulations are painful, it is best to postpone corrective work

until the acute attack has passed. The patient may drink freely of hot water, have an enema of rather warm water, and go to bed with a hot water bottle at the feet or over the abdomen, and an ice bag or a hot water bottle at the base of the occiput; this may avert the attack. In patients of sedentary habits, the hot water and the enema may be followed by a walk or some game in the fresh air; by a hot or Turkish bath; or by massage of the entire body. In either case, at least a day of rest is necessary, even if the pain and nausea are averted altogether; otherwise the next attack will occur more quickly. It must be remembered that the usual exciting cause of an attack is fatigue of some of the nerve centers.

During the intervals, treatment must be initiated for the prevention of the attacks.

In any case of migraine a thorough blood examination should be made for the sake of determining the true physiological condition of the patient. The urine analysis serves the same purpose.

Any effective treatment must be persisted in for months. Structural perversions must be corrected and the corrections must be repeated as frequently as may be necessary. The diet must be wholesome, easily digested and planned according to the results of the blood and urine examinations. Once carefully decided upon the diet must be rigidly followed. A study of the habits of the patient is necessary. A careful regime must be worked out and this must be followed religiously. If these directions are persistently followed even the most evident of hereditary cases usually recover completely within a year or two. Recovery is not sudden. In the beginning the condition may seem to be exaggerated, the attacks more severe and the intervals shorter. After one or two apparent exacerbations, the attacks become modified and the intervals longer, until they should finally disappear. The person who suffers from migraine should never marry another who suffers from migraine, nor from any other neurosis.

OCCUPATIONAL NEUROSES

Persons of neurotic temperament whose occupations require the repeated performance of complex movements frequently suffer from a cramp of the muscles concerned which is sometimes associated with considerable pain when their use is attempted. In typical cases pain is not present and the use of the muscles in other movements is perfectly normal. The only **etiological** factor is the occurrence of fatigue of the nerve groups which control the more complicated movements, especially of the hands. In a number of cases reported, bony lesions of the lower cervical and the first and second thoracic vertebræ have been reported. The condition of the shoulder and clavicular joints should be investigated.

Writers' cramp or scrivener's palsy is the most common of these neuroses. Pianists, violinists, telegraphers, seamstresses, barbers, tailors, shoemakers, and cigar wrappers are all subject to these muscular cramps. Dancers, men who walk upon snow shoes, and skaters are sometimes affected by similar cramp, affecting the muscles of the legs.

The diagnosis of writer's cramp is not often difficult. In some cases agraphia, ataxia beginning in the arms, and early paralysis agitans may be confused with this, or some other occupational disease.

The cramp which results from the habitual and improper use of skeletal muscles in maintaining equilibrium probably belongs in this group. Lesions affecting the pelvic girdle cause cramp of the muscles of leg; lesions affecting the shoulder girdle cause cramp of the muscles of the forearm; lesions of the occiput cause cramp of the muscles of the neck; lesions of the mandible cause cramp of the muscles of mastication, and lesions of the thoracic vertebræ may cause cramp of the intercostal muscles and the diaphragm. These, and other muscular cramps, are sometimes confused with neuritis, as in cases of sciatica and lumbago, now generally recognized by orthopedic surgeons and other physicians as being due to lumbo-sacral strains, or to subluxations of lumbar vertebræ.

Treatment. Rest is essential. The correction of the bony lesions as found facilitates recovery. The left hand may be used instead of the right, but the cramp soon affects this hand also. For writers' cramp the typewriter may be advised. A bracelet which holds the pen and is moved by the forearm muscles can be used. Cramp is, however, apt to attack these muscles in the course of time. In general, a change of occupation ultimately becomes necessary.

CHAPTER XXXVIII

NEUROSES WITH MOTOR SYMPTOMS

EPILEPSY

(Falling disease; seizure; morbus sacer)

A satisfactory definition of epilepsy is very difficult. Certainly it is a disease of the brain, of unknown cause, and characterized by attacks associated with loss of consciousness and with more or less pronounced motor phenomena. There is a tendency on the part of some authors to limit the term epilepsy to the idiopathic form, others apply the term to all typical seizures in which the epileptic sequence of events is present.

Grand Mal is the term applied to the ordinary epileptic fit which shall be described hereafter.

Petit Mal has little or no muscular action and is characterized only by a loss of consciousness, usually very short.

Jacksonian Epilepsy is due to a localized cortical lesion. The attacks always begin in a certain limited area of the body and spread to neighboring muscle groups until the whole body is concerned in the convulsion.

Epileptic Equivalent or psychic epilepsy is rather rare. In this form the place of the fit is taken by what may be called a mental convulsion. It is really an attack of more or less violent insanity.

"Running Epilepsy," epilepsia cursoria, or procursiva epilepsia, are terms applied to a condition in which the place of the ordinary convulsion is taken by a sudden attack of violent running until the patient is exhausted. All epileptic equivalents are dementing.

Myoclonic Epilepsy is a form in which the muscles are in a state of increased tone during the intervals of the attacks.

Status Epilepticus is a state in which the fits follow one another rapidly; consciousness may not be regained in the intervals, and it may be impossible to count the fits. Occasionally death occurs speedily from exhaustion; occasionally the patient lives longer than seems in any way possible in this state, and may even recover his usual health after days of apparently constant subjection to the epileptic attacks.

Nocturnal epilepsy occurs during the night only. **Diurnal** epilepsy occurs only during the day time.

Symptomatic epilepsy is a symptom of recognizable disease anywhere in the body, but usually involving the brain.

It is very evident that all epilepsies are truly symptomatic. Only because we do not know the true cause of what is ordinarily called idiopathic epilepsy, do we apply that term to it.

Pathology. Epilepsy is preëminently a degenerative disease of the cerebral cortex, though the true nature of this disease is as yet unknown. Gliosis of the horn of Ammon has been described. Various degenerations and atrophies especially affecting the external layer of the cortex have been described. Small hemorrhagic areas have frequently been found in the basal ganglia.

Abnormally small aorta, deficient cerebral blood vessels and the congenital absence of certain branches of the circle of Willis have been considered responsible for the condition through the defective circulation through the brain thus produced.

Most epileptics suffer from gastro-intestinal disorders. In most cases the time required for the passage of food through the alimentary tube is considerably increased. The colon is frequently dilated. It is supposed that the toxic materials absorbed as the result of this slow peristalsis may be in part responsible for the attacks. The gastro-intestinal disorder is probably due to the underlying neurosis which manifests itself also in the epileptic attacks.

Etiology. Heredity is a very important factor. While epilepsy itself is not often found in the parents, it is very rare to find a case of epilepsy occurring in a family in which no other neurosis appears. Hysteria, migraine, drug addiction, the alcoholic habit and other neuroses in the parents are very frequently associated with epilepsy in the children. As had been stated elsewhere, the inheritance of neuroses in general follows Mendel's law.

Alcohol is certainly one of the important factors. The old idea that the child resulting from conception occurring during an alcoholic spree and especially during the drunkenness of the father is predisposed to epilepsy is certainly based upon truth. This is supported by a study of the children born at a time corresponding to various feast-days in certain localities in which drunkenness is usually limited to such holiday periods, and by the finding of a number of individuals in whom a single drunken intercourse resulted in conception. Alcohol given children is one cause of epilepsy. Fortunately the indiscriminate use of medicines containing alcohol or the opium derivatives is not at present permissible.

Epilepsies occurring during early life may be due to injury at birth or to the acute diseases. The cerebral hemorrhage produced at birth has long been recognized as a cause of epilepsy occurring sometimes rather late in childhood. The fact that injury to the cervical spinal column may be produced by abnormal birth processes, or by improper obstetric procedures, is recognized by osteopaths as being an important factor in birth palsies as well as in epilepsy.

Falls and various mental and physical shocks occurring during the first few years of life are probably responsible in some cases. Here again the presence of the spinal injuries must not be forgotten.

Bony Lesions. It is very rare to find a case of idiopathic epilepsy in which there is not a lesion of the occiput or the atlas. Lesions of the other cervical vertebræ, the second to the fourth thoracic, and of the ribs are also described in this connection.

Diagnosis. It is usually not difficult to make a diagnosis of grand mal. The typical grand mal presents the following history:

The patient may have prodromal symptoms for a few hours to a few days before the attack. These usually include vague uncomfortable sensations, some indigestion, sometimes headache and very often a marked irritability of temper; rarely a tendency to somnolence is observed. The **aura** precedes the attack but a few seconds or a few minutes. This may be either sensory or motor. The sensory auræ include olfactory sensations, e. g., a smell of burnt feathers or of violets; gustatory, e. g., a sweetish taste; auditory, e. g., ringing or crackling noises; visual, e. g., a brilliant red light, a sensation as of flames or floating bright specks; visceral, e. g., nausea or hunger. More commonly the aura consists of a vague, indescribable sensation of impending catastrophe. The aura may be sufficiently prolonged to permit the epileptic to lie down and thus lessen the danger of injury.

The **convulsion** begins with a tonic phase, during which all the muscles of the body are contracted and tense; the face is at first pale, later red and then purplish. The sudden contraction of the respiratory muscles produces the typical "epileptic cry." The tonic phase is followed by the clonic, in which the muscles alternately contract and relax in a violent and often disastrous manner. The movements grow progressively less marked and finally cease. The patient regains consciousness within a few minutes or passes into a deep sleep, which may be from a few minutes to several hours in duration.

During the fit the tongue is often bitten so that the blood mixes with the saliva. The forced respirations churn the saliva into a froth which is, of course, sometimes bloody. Urine and feces may be voided, less commonly semen is expelled. When these attacks occur at night the patient may not be aware of his condition. The soiled or wet bed clothing may be the only indication of an attack. In children in whom persistent bed-wetting occurs the possibility of nocturnal epilepsy should be borne in mind.

In grand mal the attacks may come at intervals of six months or even a year, or they may recur so often that there is no interval of consciousness between them. In this condition the term **"status epilepticus"** is used.

The blood of epileptic patients is characterized by high viscidity, diminished coagulation time, and usually an increase in the eosinophile percentage. The hemoglobin and the red and white cell counts are usually normal or slightly above. The water in the blood always seems deficient.

In the intervals between the attacks the urine may be normal. Just before the attack the solids may be considerably diminished. After the attack a small amount of urine, highly colored, usually offensive in odor, with high specific gravity and heavily charged with urates is voided. Blood and albumin are often present, and the

phosphates may be increased at this time. Occasionally there is no change from the normal in the urine.

The blood pressure is usually above normal at all times and increases 10 to 30 millimeters before an attack.

Petit Mal. The occurrence of short attacks of unconsciousness is not infrequently associated with grand mal, though petit mal may exist alone. The attacks may occur at rather long intervals as several days apart, or they may come rather frequently. In one patient in the P. C. O. clinic the attacks came every two minutes for some days. The length of the attacks varies from a second or even less to several minutes. The patient is usually unaware that anything has happened. He may stop in the middle of a word and at the termination of the attack complete the word with no idea that his speech has been at all interrupted. Occasionally a slight sense of dizziness tells him that he has "been away" or "had a spell." The relation between petit mal and the epileptic equivalent must not be forgotten.

Psychic Epilepsy. The cases of psychic epilepsy include some of the most peculiar, and some of the most horrible of all of the crimes in history. From those cases in which the patient, after a slight period of unconsciousness, such as that of petit mal, performs some clownish or illogical act, such as partially undressing himself, or whirling in a circle while he spits very rapidly in every direction, to terrible murders of the Jack the Ripper type, these patients display many absurd and freakish phenomena. One P. C. O. patient had visions in which she visited the home of the Katzenjammer Kids. Occasionally the patient who has seemed to be perfectly harmless during the attacks may suddenly develop a destructive mania. These patients are very dangerous if they become angered or frightened.

Jacksonian Epilepsy. This form is invariably symptomatic. The area of the brain affected can be rather strictly localized by noticing the character of the movements which begin the attack. In this form one certain muscle group, as, for example, the flexor of the index finger, first undergoes tonic contraction, this is followed by flexion of the other fingers and the forearm, etc., until the whole body is in tonic convulsions. This is followed by the clonic convulsions as in the case of the grand mal attack. Sometimes these attacks are abortive, and consciousness may not be lost at any time.

Treatment. The treatment of epilepsy must vary according to the conditions as found upon examination. In idiopathic epilepsy the upper cervical and occiput lesions must be corrected. The diet should be almost exclusively vegetarian, with the addition of milk and the milk products and eggs. Meat, alcohol, tobacco, tea,

coffee, rich pastry, are to be refused absolutely. Excess of starch and of sugar should be avoided. Some epileptics are unable to manage more than a very small amount of fats. Fresh fruits and vegetables, especially raw vegetables, are to be eaten very freely. An increase in the amount of water intake is almost always necessary. A few weeks upon the exclusive milk diet is sometimes advantageous in patients in whom there is pronounced weakness and emaciation.

The condition of the gastrointestinal tract, especially the colon, is important. Dilatation of the stomach, constipation, visceroptosis must be treated vigorously. (q. v.) H. W. Conklin considers the ascending colon and the sigmoid especially important. X-ray examination should be used for determining the true condition of the intestinal tract; this gives foundation for rational treatment. Enemas and manipulations directed to restoring the correct structural relations are indicated in most epileptics.

Every effort should be made to remove possible sources of nervous irritation. The presence of eye strain has been discussed pro and con. There is no doubt that epileptic as well as all other persons should be fitted with glasses when the condition of the eyes renders such a course advisable. Adenoids, hardened ear wax, scar tissue in any part of the body, intestinal parasites, anal abnormalities, adherent prepuce or clitoris, and any other sources of peripheral nerve irritation should be completely corrected. Children should be especially guarded from excitement. They should not be sent to the ordinary schools, but should receive teaching under circumstances that preclude the possibility of their being associated with other children at the time of an attack. Drugs are to be avoided. It is true that certain drugs (bromides) commonly used diminish the force and the frequency of the fits, but these invariably increase the mental deterioration and they usually cause more or less of gastro-intestinal and other disturbances.

The patient who suffers from petit mal should be carefully guarded lest some of the psychic phenomena appear suddenly. The patient with psychic epilepsy should usually be placed in some institution where he can be guarded from injury to himself or to others. In Jacksonian epilepsy and also in certain other types of epilepsy in which a history of injury to the skull is secured, surgical procedures are often most helpful. It is necessary to make a careful study of each case in order to decide upon the location and the nature of the operation to be performed. The help to be secured from surgical interference depends greatly upon this procedure being initiated at an early stage. It seems that the recurrence of these attacks for a considerable period of time brings about a degeneration which is more or less widely spread throughout the brain centers.

In symptomatic epilepsy, the treatment depends upon the true cause of the condition. Brain tumors are sometimes operable. The prognosis and treatment in these cases is always that of the underlying cause.

The epileptic character. When epilepsy begins early in childhood, especially after the attacks are frequently repeated, the mental deterioration is speedy and marked. This is probably due to the fact that injury to the cerebral nerve cells is more profound when it acts upon them during the stage of their most rapid development than it is if it acts upon them after the development has reached a fairly stable degree.

When the first attacks begin during late childhood or during puberty, the effect upon mentality is somewhat less marked. In these cases and also in milder cases of very early onset, we have developed a peculiar personality which may be due to the effects produced upon the brain or may be due, at least in part, to the effects of the treatment which epileptics receive from other children and from the grown people with whom they are associated.

The epileptic is almost universally gloomy, pessimistic, egotistical and suspicious. He may love intensely and even with great self-sacrifice. With this he rarely trusts even those whom he loves and is almost invariably subject to furious jealousy. Not rarely the ingenuity and the powers ordinarily called purely mental are excellently developed in epileptics. This is evident in the fact that so great a number of epileptic men and women have been powerful in modifying the course of a history of the world.

"Epilepsy, affecting centuries ago the greatest of the Cæsars, has been present as a human affliction during all the span of human existence. It is recognized as a condition resulting from effects upon the central nervous system, due to abnormally constructed brain elements, the sequence of alcoholic or syphilitic parentage, to fright, injury to the head or a sunstroke, to peripheral nervous irritants such as adenoids, enlarged tonsils, adherent prepuce or lumbricoids or to auto-intoxication of a severe type.

"In this case I have assigned its causes under two heads, accidental and predisposing, and I have chosen to call the accidental as follows:

"1. Forceps delivery, in which no deformity was produced at the time, but which was undoubtedly the cause of a severe lesion, occipito-atloid.

"2. Fright early in her sixth year, due to a narrow escape, while with parents, from being crushed by a train.

"3. A fall from a small cart drawn by a boy, striking violently on her head on a cement walk, no appreciable damage to skull, this occurring two or three months before the appearance of any trouble.

"The predisposing causes were an oversensitive nervous system, reacting to all environal changes, even the most minor and a tendency to gastro-enteritis, with its concomitant nervous influences.

"The lesions presented are an occipito-atloid, previously referred to, in which the occiput on the left is tightly jammed down upon the lateral mass of the atlas and a compensating lateral axis. With these as primary lesions the secondary lesions are alternating lateral conditions at the cervico-dorsal junction and lesions in the lower thoracic and sacro-iliac regions."—C. H. Phinney.

Prognosis. When epilepsy begins early in life, it is usually incurable and dementing. When it begins late in childhood, it may be outgrown by about the age of twenty. When its onset shortly precedes the puberty changes, it may disappear within a few years after the puberty changes are completed. Grand mal has the better outlook; petit mal is more frequently dementing, while the psychic type is almost always dementing. Combinations of types have the more gloomy prognosis; occasionally, however, a petit mal will be followed by grand mal for a few attacks, and this be followed by cessation of the attacks. The sequence is sometimes reversed.

Life is not shortened by epilepsy, until status epilepticus leads to death from exhaustion. The fits prevent patients from engaging in much hard work; rarely they may cause death by accident. Epileptics are usually so egotistical and so selfish that they care for themselves better than normal persons usually do; they may outlive their generation.

Recovery may be expected when some removable cause can be found for the condition, provided suitable treatment is begun at an early date, before brain injury has supervened.

ACUTE CHOREA

(Infectious chorea; Sydenham's chorea; St. Vitus dance; St. Anthony's dance)

Acute chorea is an infectious disease of the nervous system, characterized by the occurrence of awkward, spasmodic movements, especially of the face and hands, and occurring chiefly in children from 5 to 15 years of age.

Pathology. Very little is known of the brain changes in chorea. Degenerations in the lenticular nucleus have been reported. The heart is almost invariably affected. Vegetations are found mostly upon the mitral valve. Cerebral embolism affecting the smaller arteries may occur.

Etiology. The disease occurs most frequently in children after they begin to go to school and before puberty. It is rather rare before the age of 7 or after 20, though cases do occur in very young children and among old people. Although it is probably an infectious disease, hereditary neurotic taint is very common. It would seem that children who descend from neurotic parents have nerve cells less resistent to the action of infectious or toxic agents than those of normal ancestry.

The ordinary infectious diseases of childhood appear to be responsible for a few cases. It is more frequently associated with rheumatism or with tonsillitis than with any other disease. Heart lesions are very common. The diagnosis of chorea is denied by some authors in the absence of evidence of cardiac injury. The place of reflex nerve irritations as a causative factor has probably been overestimated. The infectious agent has not been isolated.

There is some reason to believe that it may be identical with that which produces acute articular rheumatism.

Diagnosis. The disease may follow any other contagious disease, rheumatism or tonsillitis. There is a prodromal period during which the child is extremely irritable and hard to manage, sleep is disturbed, bad dreams are frequent, and night terrors may occur. After a few days, it is noticed that he is very awkward in his movements; he drops things which he has in his hands, may knock the dishes off the table while he is eating and behaves generally in an unusually awkward manner. If he is punished, as is too often the case, the condition grows more rapidly worse, the involuntary and spasmodic character of the movements then becomes evident. Silly grimaces, twitchings of the facial muscles and of the muscles around the eyes and eyelids are usually associated with more or less of a shrug of the shoulders. The hands and feet and sometimes the whole body take part in these spasms. The child may be so seriously affected as to die from exhaustion. He may be unable to swallow and respiratory movements may be irregular. The movements cease during sleep, but they may prevent his being able to go to sleep. In most cases the symptoms are less severe and recovery occurs in one or two months. Those cases in which the fever is high, perhaps 104°, have a worse prognosis. The diagnosis is made upon the symptoms as observed.

Treatment. The treatment of chorea depends upon securing and maintaining the best possible circulation of the best possible blood through the central nervous system. It is equally advisable to pay no attention to the spasmodic movements during the acute stage of the disease. The child should be treated as gently and kindly as possible during the period of his greatest irritability. After the disease has terminated, the movements may persist as habit spasms. In this case the condition should be treated as are other habit spasms or tics.

"The prognosis of simple chorea is good, nearly all cases get well under osteopathic treatment. Some few cannot be cured but can be materially benefited. In those cases where grave nervous diseases are traceable in the ancestry, the prognosis is never so good for an absolute cure."—A. H. Zealy.

CHRONIC PROGRESSIVE CHOREA

(Hereditary chorea; degenerative chorea; Huntington's chorea)

As the name indicates, this is a degenerative disease of the brain, characterized by gradually progressive choreiform movements of the voluntary muscles, by a progressive dementia and by its hereditary nature. It is rare in the United States.

Etiology. Heredity seems to be by far the most important cause of the disease. In "choreic families" normal individuals may occur. The children of these are usually free from the disease, but children who are free from the chorea and the descendants of these are very apt to suffer from epilepsy, hys-

teria, idiocy, the adolescent insanities, or paranoia. Its onset in middle life (rarely before thirty or after forty-five years of age) permits the transmission of the disease in direct heredity, though not usually to many children in one family.

Pathology. A diffuse meningitis which involves both the dura and the pia-arachnoid is usually present. Capillary hemorrhages, which seem to be most marked in the corpora striata, are usually found. Associated with these are various degenerations and atrophies of the cerebral neurones.

Diagnosis. The symptoms and history give the diagnosis. The disease begins in middle life with a change of character, the patient becoming irritable and unstable. Peculiar movements, jerky respirations, changes in speech, appear at first to be the expression of whimsy or eccentricity. The involuntary nature of these movements soon becomes evident. The movements do not often become so severe as to cause injury, as is the case in infectious chorea, but they may interfere with the patient's ability to earn a living. He walks with his legs wide apart, the arms hanging dangling in a jerky way, and the whole gait and habit are often clownish. Indeed it is not improbable that clownishness originated with such patients. The movements disappear in sleep and can be voluntarily inhibited for a short time. Usually after voluntary inhibition, they recur with increased violence. After a few months or a few years, the mentality becomes recognizably diminished and finally complete dementia supervenes. Life does not seem to be shortened by the disease and the patient may remain helpless and demented for twenty years or more, unless some intercurrent malady terminates his pitiable existence.

Treatment. The treatment must be symptomatic. A child born into a family in which this disease has occurred should be kept in as nearly as possible a normal environment, with wholesome surroundings, good food and preferably outdoor life. After the onset of the disease, it is doubtful if anything can be done to prevent the ultimate degeneration. Intercurrent maladies should receive appropriate attention. As soon as the dementia reaches a noticeable degree, the patient should be sent to some institution where he can be made comfortable and kept harmless. Marriage should be prevented, or if members of these families are married, they should remain childless.

The **prognosis** is hopeless after the disease has become evident.

INFANTILE CONVULSIONS

(Eclampsia infantilis)

The occurrence of convulsions resembling those of eclampsia and sometimes those of epilepsy in children during the first or second year of life is not at all unusual, especially in children of neurotic inheritance.

Etiology. Convulsions occurring in children may be due to a great many different factors. These are always either of nervous or toxic origin, or both. Perhaps the most common causes are intestinal disturbances occurring during the eruption of the first teeth. Convulsions due to this condition usually leave no serious after-effects. The presence of worms in the intestinal tract is also a frequent source of infantile eclampsia. No doubt both the nervous irritation due to the presence of the worms and the absorption of the toxic substances produced by their metabolism are concerned in producing the convulsions.

Acute nephritis in children may produce uremic convulsions. Rachitis is frequently associated with convulsions, which in this case are probably toxic in origin.

Not rarely the meninges become inflamed in the course of the acute infectious diseases of childhood, in which convulsions resembling those of ordinary meningitis are likely to occur. High fever associated with the acute infectious diseases, or with gastro-intestinal diseases, may produce convulsions.

Emotional storms in neurotic children frequently cause extremely severe convulsions, and these may be associated with slight capillary hemorrhages into the brain substance. Children who suffer from convulsions upon apparently trivial excitement or emotional shocks are very likely to grow up into hysterical or neurasthenic adults.

Sometimes the fits which appear to be infantile convulsions recur through childhood as true epilepsy. In such cases it is probable that what appeared to be infantile convulsions due to gastro-intestinal disorders, was merely epilepsy occurring at that time.

Convulsive attacks in children may be due to organic brain lesion, brain tumors, rarely hydrocephalus, brain tuberculosis, inherited syphilis; or the postponed effects of cerebral hemorrhages caused at birth may be responsible for one or several convulsions occurring during the first few years of life. All of these extremely varied etiological factors indicate that infantile convulsions, as well as epilepsy, must be considered a symptom of some underlying disease.

Pathology. The pathology differs according to the various causative factors. Various degenerations of the motor cortex and the basal ganglia have been reported. Capillary hemorrhages in the meninges and in the brain are sometimes present. The lesions of rickets may be found. The convulsions themselves are probably responsible for minute hemorrhages in the brain and meninges and for the chromatolysis and vacuolization of the motor neurons of the brain and cord.

Diagnosis. The diagnosis of infantile convulsions is easy, for the very fact of the convulsive spasm is rather typical. A child which has been more or less ailing for a few days becomes pale, seems to lose consciousness, the muscles undergo sudden stiffness, and the legs, arms and back become straight; the respiratory muscles are contracted, the breath is held; lips are blue, face is very pale, and this terrifying appearance remains for a few seconds or a few minutes, the breath is caught, face flushes, the child screams, the muscles relax, and the attack is over, or it may be immediately succeeded by another similar attack. Occasionally the muscles remain contracted, respiration is difficult, and pallor is marked for some hours.

The diagnosis of the underlying cause of the convulsion is sometimes very difficult. When the spasm is due to gastro-intes-

tinal symptoms, the history of previous gastro-intestinal disease or of the eating of improper foods may help in the diagnosis. The stomach tube or enema may bring absolute proof of the cause of the disturbance. The recognition of worms (q. v.) in the intestine is sometimes difficult. Kidney disease should be suspected when there has been edema, or when there is a urinary odor about the child. Urinalysis is always indicated. Diagnosis of the acute infectious diseases, rickets or meningitis can be made by applying the tests usual in these conditions. Usually there is something in the symptoms which suggests these diseases. The organic brain lesion may present considerable difficulty in diagnosis. Examination of the fundus of the eye should never be neglected in children who are subject to convulsions without recognizable cause. Blood examination may indicate the correct diagnosis.

Treatment. The treatment of the convulsion itself is rarely difficult. The old-fashioned process of putting the child into warm mustard water is probably the best thing the mother can do. Gentle and prolonged extension of the spine is good. Raising the ribs in the movements of artificial respiration frequently brings the convulsion to a sudden termination. If gastric disturbances are present, it may be necessary to use the stomach tube as soon as the relaxation of the muscles permits. Dilatation of the anal sphincter may terminate the attack, and is indicated when there is reason to believe that worms or other anal irritations are present. When the convulsion is caused by adherent prepuce or clitoris, the relief of this tension may relieve the spasm. In uremic convulsions the treatment as outlined for uremia in general should be employed.

The convulsions due to brain lesion usually do not yield to any ordinary therapeutic methods. In these cases or in severe convulsions due to any other cause, the inhalation of chloroform may be necessary. A very few drops sprinkled upon a handkerchief and held in front of the child's nose is usually sufficient. It is not advisable to permit chloroform to be given by any member of the family as a general thing. Not only is there danger of sudden disastrous results from overuse or improper use of the chloroform, but this poison itself sometimes has a very serious effect upon the liver.

For the treatment of the cause of the convulsion it is necessary to consider the etiology. The underlying neurosis is usually best met by securing increased nutrition and better hygiene and education for the child. Emotional disturbances and especially ill-judged attempts at discipline by nervous and erratic parents must be carefully avoided. Education must be secured by the use of firm and yet gentle measures, always avoiding emotional storms. Organic brain lesions and certain bodily conditions may

best be removed by suitable surgical measures. Proper diet, proper habits of living, the correction of bony lesions as found upon examination, together with the treatment adapted to such other abnormalities as may be found on examination should result in recovery in by far the greater number of these cases.

Prognosis. In any case of infantile convulsions, a somewhat guarded prognosis should be given. While it is true that by far the larger number of these cases recover completely with no ill after-effects, yet it is impossible in any given child to say definitely whether this will be true in his case. The possibility that the convulsion may be the first of a series of epileptic attacks, or that it may be the symptom of some unrecognized nervous disease, must never be forgotten.

TICS

(Habit chorea; habit spasm; motor tic; palmus)

A tic is an involuntary movement occurring in neurotic individuals as the result of some voluntary movement first performed under the influence of a morbid physical or mental condition.

Etiology. Probably a neurotic constitution is necessary to the formation of any tic. Morbid physical conditions affecting the activity of the nervous system in pain certainly act as predisposing factors. Tics may originate from habit as in the case of a limp which persists after a painful injury to the foot or it may originate from some violent emotional state, as the blinking of the eyes after the sight of some terrifying object, or it may represent some of the repressed emotions, such as have been so strongly emphasized by the Freudian school, as in certain forms of tremor of the right hand.

Children are especially apt to have tics develop as the result of imitation of other children, a child with chorea, for example, may set the example of choreiform movements to his playmates.

Diagnosis. It is sometimes rather difficult to decide whether any given movement is a tic or spasm. The tic can usually be imitated exactly, the spasm cannot. The tic disappears with education, either with or without some psychoanalysis. The origin of the tic is in some volitional movement, spasm makes its own appearance. The tic may involve almost any of the voluntary muscles. Functional wry-neck, blepharospasm, grimaces, peculiar movements of the tongue and the mouth, shrugging of the shoulders and many awkward movements of the hands and fingers are forms of tic. Certain types of stuttering and stammering speech are tics.

Treatment. Reëducation is by far the most important factor in treatment. In order to secure cure, the patient must imitate

his involuntary movement until he can perform it voluntarily. If the tic includes speech disturbance, he must imitate his stuttering performance, or imitate very carefully whatever sounds he may have been making. One can refrain from doing only those acts which he is capable of performing. As soon as he has learned to perform the action represented by his tic exactly, he is able to refrain from performing that action. Usually the very learning to do the act results in its inhibition. When there is reason to suppose that an emotional shock or some repressed complexes are concerned in the etiology of any particular tic, some modification of the methods of psychoanalysis may be employed.

GENERAL TIC

General tic is a disease which is characterized by the occurrence of extremely complicated movements, with or without speech disturbances. The mentality is not affected, though imperative ideas and obsessions are not rare.

Etiology. No cause is known for the occurrence of the disease, other than that it is most apt to occur in neurotic individuals or in those of neurotic heredity. It occurs in late childhood, frequently just before the onset of adolescence, and both sexes are about equally affected. The disease generally begins in some of the eye muscles, especially orbicularis palpebræ. Uncontrollable winking is the most frequent first symptom. Various facial spasms follow and then other muscles take part in the convulsive reaction. Various cries, sometimes imitating the crow of the cock, or the bark of a dog, or the sudden and explosive speaking of certain words (coprolalia, echolalia, etc.) may occur. Echokinesis or the tendency to imitate any movement which he sees others perform may result in considerable mental disturbance. The mentality is not affected and the patient usually feels his lack of self-control most keenly.

Diagnosis. The diagnosis rests upon the symptoms as enumerated and is usually not difficult.

Treatment. The treatment is based upon securing the best possible circulation of the best possible blood through the brain and cord. Rest and ordinary good hygiene are important.

Prognosis. The prognosis is very bad for recovery. The most that can be hoped for is to delay the further progress of the disease to some extent. Life is not shortened by the disease. Indeed such patients are apt to live longer than normal people, because they are, by their infirmity, protected from the ordinary infections and accidents of normal life.

PARAMYOCLONUS MULTIPLEX

(Myoclonus multiplex; including also myokymia fibrillary; chorea of Mouvine; myoclonus fibrillaris; multiplex of Kny; electric chorea of Dubini; Bergeron, and Henoch)

This is a disease of unknown etiology and pathology, characterized by sudden spasmodic contractions of muscles which are rapid and do not produce movements of the limbs or body.

Etiology. The disease occurs in families in which the hereditary neuroses occasionally appear. Emotional disturbances, especially fright, are frequently given as the cause by the patient or his family. Similar spasmodic contractions are sometimes associated with idiocy.

Diagnosis. This rests upon the lightning-like character of the contractions, which greatly resemble the effects produced upon a muscle by stimulation with the electric current. The movements may be very frequently up to 100 each minute. There are no symptoms of organic nervous disease and no changes in mentality, the tendon reflexes are increased but slightly and no changes in the electrical reactions have been reported. The disease may be confused with hysteria or with infectious chorea.

Electric Chorea of Dubini is endemic in Northern Italy, and is found in this country only among immigrants. This form begins with pain in the neck. The muscular contractions are marked and may be painful. In a few days to a few months coma appears and death results. It is almost invariably fatal within a few months. In this form meningeal congestion is found, and it is often associated with inflammatory diseases in the lungs or sometimes in other viscera.

Henoch's Chorea may be merely a subtype of infectious chorea. It appears in infants or children and becomes chronic rather than self-limited, as is usual in the infectious type. It may disappear at puberty.

Bergeron's Chorea appears in poorly nourished and anemic children. The progress is about that of Henoch's Chorea.

Tetanic Chorea is a peculiar form of chorea in which the movements are made slowly and somewhat strenuously, as is evidenced by the expression tetanic. This disease is rather rare and is associated with cirrhosis of the liver in every case so far reported.

Nodding Spasms of Infants. This is rarely found in this country. A few hours or a few days after birth nodding movements occur. These disappear during sleep and do not seem to cause the infant any particular discomfort. They may persist for a few months or they may last until the child begins to walk when they gradually disappear.

Treatment. In all of these cases the treatment must be planned towards securing the best possible circulation of the best possible blood through the entire central nervous system. The **prognosis** is implied in the description of the disease already given.

PARALYSIS AGITANS

(Parkinson's disease; jerking palsy)

Paralysis agitans is a disease of late middle life which is characterized by a trembling of the muscles, increase in muscular tone and progressive weakness.

Etiology. Almost nothing is known of the cause of the disease. There is no reason to suspect any hereditary taint. The disease is likely to occur after a fall, hard work, fright or excitement, or after the occurrence of some infectious disease. Since all of these factors are very common, while the disease itself is rather rare, it is evident that these factors alone are not sufficient to account for its etiology. Bony lesions of the cervical and upper thoracic region are almost universally present.

Pathology. Almost nothing is known of the pathology of paralysis agitans. Atrophy of the cells in the motor cortex, gliosis in the spinal centers, and overgrowth of neuroglia around the spinal arteries have been described. All these changes are found in senility, whether paralysis agitans had been present or not. Similar conditions have been induced in animals by the removal of the parathyroid glands, and there is a certain amount of evidence looking to the thyroids and the parathyroids as being concerned in this disease.

Diagnosis. The diagnosis is made upon the symptoms. The disease begins in the fingers, then extends to the muscles of the arms, the neck and other groups. The increased tone of the muscles gives a certain stiffness to all voluntary movements. This increase in tone is not limited to the trembling muscles, but usually involves practically the entire body. The face assumes a set mask-like expression; all ordinary movements are performed in a stiff and awkward manner; the gait is characteristic—the patient finds difficulty in getting started to walking and his shoulders bend forward, his arms hang stiffly and he walks as if he were being pushed from behind. This effect is emphasized by the fact that the change in the center of gravity of the body makes it necessary for him to walk more briskly. The gait resembles a sort of slow trot.

The mental processes usually are delayed, increased reaction time sometimes is evident even without the use of any particular tests. The simplest question may have its answer delayed for some seconds or minutes. The mentality is usually unaffected, although a recognition of his condition usually causes him to be more or less depressed. One patient in the P. C. O. clinic was so affected in this way that he committed suicide. Usually, however, the depression is much less marked.

Treatment. The correction of the bony lesions as found or the use of movements which increase the mobility of the spinal column in a general way usually lessen the tremor or cause it to disappear altogether for some hours. The progress of the disease is somewhat diminished, apparently, by such treatment. A few cases in the incipient stage have been reported cured.

The affected muscles should be kept at rest. Passive movements and massage are somewhat beneficial. Cold usually increases the stiffness and the tremor. Therefore, patients should

be sent to a warm climate if possible. A long continued warm or neutral bath frequently relieves the trembling for some hours. Rest of body and mind are very important. Members of the family must be warned against any display of impatience when the patient is slow in answering questions, or when he fails to understand as readily as had been his custom.

The neck must receive careful attention. Contracted muscles may interfere with cerebral drainage, or with the circulation or innervation of the thyroid or parathyroid glands. This treatment relieves the melancholy tendencies in many cases. The lower thoracic region, especially the eleventh and twelfth thoracic vertebræ and ribs usually require correction. Normal condition of the liver and kidneys may prevent adverse toxic influences upon the muscles.

Prognosis. The disease does not apparently interfere with the general health, except as the stiffness may be responsible for some accident. Recovery is not to be expected in typical, well-developed cases.

CHAPTER XXXIX

DISEASES OF THE PERIPHERAL NERVES

GENERAL DISCUSSION

The symptoms produced by abnormal states affecting the peripheral nerves depends upon the structure of the nerve trunks and their central and sympathetic relations. The nerve fibers which make up a nerve trunk are three in origin and function. The motor nerve fibers arise from the nerve cells in the anterior horns of the spinal cord; the sensory fibers arise from the cells in the sensory ganglia in the intervertebral foramina, and related cerebral ganglia; the vasomotor and secretory and visceromotor fibers arise in sympathetic ganglia, situated in various parts of the body. The motor and sensory nerve fibers (except the olfactory) are enclosed in a fatty sheath, called the medullary sheath, or white substance of Schwann; this is structureless and its existence depends in some way upon the functioning of the nerve fiber. Around the medullary sheath is a very delicate membrane of connective tissue, the neurilemma. The sympathetic fibers, which lack the medullary sheath, are surrounded by the neurilemma, as are the cerebrospinal fibers. These various fibers are bound into bundles, which are loosely supported and permeated by connective tissues. Blood and lymph vessels for the nutrition of the nerve trunks are carried in the connective tissues; these receive nerves for their control. Sensory nerves also are distributed to the nerve trunks.

Abnormal conditions which affect the vasomotor and sensory nerves of the nerve trunks, nervi nervorum—may cause severe pain in the nerves themselves, without causing any interference with the structures innervated by the nerves affected (neuralgia); irritating substances in the circulating blood may affect either the nerve fibers within the nerve trunks (toxic neuritis); or may affect more seriously the nervi nervorum (toxic neuralgia); pathogenic bacteria affecting the nerve trunk usually affect all its structure (neuritis due to the infectious diseases); abnormal structural relations, tumors, fragments of bone, callus, gummata, etc., affect first the nervi nervorum, causing what is usually called neuralgia, later, affect the ultimate fibers, and pressure neuritis results.

It seems fairly evident that the vasomotor control of the nerve trunks is a function of the spinal vasomotor centers in the lateral horns of the cord, and thus is subject to reflex disturbances, as are other tissues of the body. The neuralgia which results from cold, or from visceral disease, is thus explained. Bony lesions

may cause disturbed vasomotor control of the nerve trunk in this way; the slight congestion may persist and ultimately a true neuritis result.

It is probable that much of the pain caused by visceral disease is due to neuralgia of the somatic nerves, most closely related to the affected viscera in the spinal or lower cerebral centers. Abnormal irritability in any sensory center is apt to be referred in consciousness to those peripheral areas most frequently the origin of stimulation; for this reason sensations arising from visceral disease are often referred to the skin, joints and muscles, innervated from the same segments (referred or reflex neuralgia). Here again the effect of bony lesions may be found; the disturbed sensory impulses due to the tension on the articular nerve endings may be referred to the peripheral areas. The hypersensitiveness associated with bony lesions is often of this type.

The peripheral effects produced by neuritis and neuralgia, such as paresthesias, anesthesias, lesions of the skin, disturbances of secretion and of the growth of hair, paralysis, muscular hypertension and muscular atony, are undoubtedly due, in some cases to the structural injury to the nerve trunk itself, and in some cases to the disturbed action of the spinal or lower cerebral nerve centers; this may in turn be due directly to the neuralgia itself, or, more frequently, due to the same underlying conditions which cause the neuralgia.

From what has been said it is evident that a diseased condition which is, at first a neuralgia, may so affect the circulation through the nerve trunk and perhaps the trophic relations of the fibers, that a true neuritis is produced.

NEURALGIA

Neuralgia is a painful disease of the nerve trunks or their distribution, characterized by varying intensity and location, and by the absence of any constant recognizable anatomic changes.

Etiology. It is due to variations in the circulation through the nerve trunk, or to the presence of irritant toxins in the circulating blood, acting upon the sensory nerve endings. The first factor may be reflex, as in the facial neuralgia due to decayed teeth, eye strain, etc., the sciatica due to rectal or other pelvic disease, and to those neuralgias due to bony lesions anywhere. The second factor may be due to autointoxication of any kind, including copremia; to inorganic poisons, as lead, arsenic, or mercury, or to organic poisons taken as drugs or with foods, as alcohol, tobacco, tea, coffee, or meat used excessively. Generally, poor nutrition causes neuralgia, both from the lack of efficient circulation, and from the products of metabolism, which are usually retained more or less extensively in starvation. Early stages of pressure neuritis

are often wrongly diagnosed as neuralgia. Any cause of neuralgia, persisting, may ultimately cause a structural change in the nerve trunk, often inflammatory, and thus terminate in a true neuritis.

Diagnosis. The symptoms are fairly pathognomonic. The attacks begin as paresthesias which become sharply painful; sensations of heat, cold, boring, cutting, grinding, pricking, stabbing, are variously described. Twitchings, like those produced by electricity, are frequent. Muscular contractions, most pronounced in the deep spinal layers of the segment of origin of the affected nerve, are constant. Vasomotor changes—pallor or flushing—of the area of distribution of the affected nerve may occur. Trophic disorders may include dermatitis and eczematous eruption, urticaria, and others less frequently.

Neuralgia may be confused with neuritis; diseases of the spinal cord, especially myelitis and tabes; meningitis, rheumatism; and disease of the brain.

Treatment. The treatment includes the recognition and removal of the cause in each individual. In any case the pain itself initiates reflex contraction of the muscles innervated by the same spinal segment or medullary center. These muscular contractions tend to cause slight congestion of the nerve trunk and to increase the neuralgic pain; the relief of these muscular contractions, and of whatever structural perversions these may have caused, is an important factor in the treatment of any case of neuralgia however produced. Neuralgia is almost always associated with poor nutrition; though the patient may be obese.

Prognosis. With such modifications in the diet and hygiene as may be indicated in each case and the relief of structural perversions nearly any case of neuralgia will disappear. Those cases due to pressure, by tumors, broken bones, scar tissue, usually require surgical relief; in the case of the callus around a broken bone, it may be necessary to use merely palliative measures until the bone is healed, when the pain disappears; or persists until surgical relief is compelled. In such cases, massage, hot and cold applications may give relief.

Facial Neuralgia. (Tic doloreaux, frontal neuralgia.) Neuralgia often affects some of the branches of the fifth cranial nerve, with perhaps greater suffering than in any other location. The pain is often of a twitching nature, superimposed upon a dull, unendurable aching.

Lesions of the upper cervical and upper thoracic vertebræ, and of the mandible, are important factors in etiology and in treatment. Diseases of the teeth, especially at the roots, antrum disease; nasal polyps; middle ear disease; and probably eye-strain, are the most frequent causes of the milder forms; these usually

yield to the treatment already mentioned. Occasionally the neuralgia is due to a degenerative process occurring in the Gasserion ganglion, which in turn may be the result of a syphilitic pachymeningitis, or of arteriosclerosis, and relief is secured with great difficulty. A lowering of the blood pressure (see arteriosclerosis) may afford relief in some cases. Surgical extirpation of the ganglion is a serious operation, but may be inevitable. Destruction of the affected nerve trunk—except the ophthalmic—may be secured by injections of alcohol into the nerve itself. Surgery is only to be recommended after other means fail, and the pain remains unendurably severe. Sometimes it recurs after surgery. Not rarely nothing gives relief until the death of the patient.

Occipital Neuralgia is usually due to carrying heavy loads upon the head or shoulders, or to falls; it is practically always associated with lesions of the occiput, atlas or axis. Rarely spondylitis is found as high as this area. In most cases, correction of the lesions as found gives speedy relief; the pain may recur when the lesions recur, but persistent treatment should result in permanent relief. If the conditions persist, the hair may fall or turn gray upon the affected side.

Intercostal Neuralgia may affect one or more of the intercostal nerves. It may be difficult to distinguish between this disease and pleurisy, especially when the pleuritic adhesions follow the nerve distribution. The lesions of the ribs and the related vertebræ are usually easily found, easily corrected, and the relief of the pain is usually immediate.

Mastodynia is neuralgia of the breast; it is often associated with slight edema, often localized. This arouses fear of malignancy; which increases the pain and tenderness. Vomiting may be associated with the paroxysms. The scar left from an old mastitis may add to the difficulty in diagnosis. The condition is usually associated with rib or clavicular lesions; the correction of these relieves the pain; this and the explanation, relieve the fears of the patient.

Cervico-brachial Neuralgia may affect any of the branches of the cervical or the brachial plexuses. When the pain is bilateral, the trouble may be due to spinal cord disease, or disease of the vertebræ. Neuralgia due to bony lesions of the vertebræ usually affects one arm more than the other; rarely, these may also be bilateral. Lesions of the lower cervical or upper thoracic vertebræ may be responsible; usually rib and clavicle lesions are associated with these. Contraction of the scaleni may raise the ribs, so that direct pressure is exerted upon the brachial plexus. The clavicles may be too low; anterior curve of the neck—"ewe-neck," "bicycle neck"—with tensions upon the anterior cervical muscle group,

may also exert direct pressure upon nerve plexuses. Surgical cases include tumors, scars, and calluses. The treatment is indicated in the etiology.

Sciatica (Neuralgia of the sciatic nerve) is a very severe form, and may be confused with spinal cord disease, tumors of the cauda equina, the pain of tabes and neuritis. It may be due to pressure, as in childbirth, or long sitting in an awkward position; or to severe constipation; or to trauma, in addition to the usual causes of neuralgia. Lesions of the innominates or of the fourth and fifth lumbar vertebræ, or the coccyx are constantly present. Ovarian disease, hip disease, pelvic diseases of various kinds, may cause a reflex pain, with muscular contractions. The treatment is that of the causative factors; rest, with the leg wrapped in cotton, gives relief; the leg should be very gently stretched and rotated, avoiding undue pain in the manipulation. The manipulation of the tissues around Poupart's ligament, as well as those around the sciatic notch and along the course of the nerve, facilitate better circulation and promote recovery. The relief will not be apt to be permanent until the bony lesions mentioned have been corrected.

Coccygodynia, neuralgia of the coccygeal nerves, is most frequently found in women. It may occur in either sex as the result of trauma or of anal diseases. In men it is caused by stone, or by prostatic disease; in women, by ovarian or uterine disease. The coccyx is often dislocated; in recent injuries it may be easily replaced, working with a finger in the rectum and a thumb on the outside, over the coccyx; sometimes old cases are easily corrected; more often the treatment must be repeated for some weeks, until the tissues either become sufficiently relaxed to adapt themselves to the abnormal state, or better, until the bone remains fixed in its normal position. If the joint is flexible, with normal tone of the surrounding tissues, the fact of its malposition is of comparatively small importance. The pelvic lesions should receive proper treatment, and the usual ordinary systemic treatment for neuralgia is indicated in most cases.

Visceral Neuralgia, lumbo-abdominal neuralgia, femoral, obturator and genito-crural neuralgias, affect the nerves mentioned; the examination of each patient reveals the specific causes in each case, and indicates the treatment. These neuralgias are rare, and are often associated with other diseases.

NEURITIS

Inflammation of a nerve trunk, however produced, is called neuritis. When the inflammation is limited to a single nerve trunk it is called "local"; when many nerves, or all the nerves, are

involved the condition is called "polyneuritis" or "multiple neuritis."

Pathology. The inflammatory process may chiefly involve the connective tissue coverings of the nerve trunks; "interstitial neuritis," or it may affect chiefly the axis cylinders, "parenchymatous neuritis." The changes in the nerve fiber, in either case, may resemble those of Wallerian degeneration, or of simple atrophy, or of fatty degeneration.

Etiology. Local neuritis is usually due to local causes—chilling or trauma; lesions associated with arteriosclerosis; extension of inflammation from neighboring diseased tissues; septic foci, bony lesions, and any disturbances of the circulation of the nerve trunks. Multiple neuritis has usually some poison as its cause; alcohol, lead, arsenic or mercury, extrinsic poisons, or the toxic effects of other diseases, as typhoid, syphilis, malaria, influenza, beri-beri—and many others, or the poisons arising from disturbances of metabolism, as in diabetes, gout, pregnancy, arteriosclerosis, pernicious and severe secondary anemias, and the cachexias generally. "Idiopathic neuritis" is that in which no cause of the trouble can be found; such cases are not rare.

Alcoholic Neuritis is usually multiple, and is associated with more or less profound mental disturbances. Other symptoms of chronic alcoholism are usually present. (q. v.) When the mental deterioration includes delirium or hallucinations, with progressive dementia, the condition is called "Korsakow's psychosis" or "syndrome."

Arsenic neuritis is not usually associated with mental changes.

Workers in rubber and silk manufacturers may suffer from neuritis due to carbon disulphide. Frontal headache and giddiness, with the symptoms of a multiple neuritis, should lead to change of occupation.

Saturnine, or lead, neuritis, affects chiefly the muscles, and has little or no pain. "Wrist drop" and "foot drop" are almost pathognomonic; the "blue line" upon the gums, "lead colic," sometimes delirium, "lead encephalopathy," and rarely optic neuritis, may be associated with the neuritis.

Beri-beri is a specific neuritis (see acute infectious diseases).

Senile neuritis occurs in old age, and is probably due to arteriosclerosis.

Diagnosis. The symptoms of neuritis include motor, sensory and trophic phenomena. Pain varies; it may be very severe, especially is this true in local neuritis. The nerve is sensitive to pressure, usually along its entire course. Its area of distribution is hypersensitive, and the tissues around the vertebræ of the segment of origin of the nerve trunk are hypersensitive; this is true whether any bony lesion is present or not. Tactile sensation may be lowered, while the hypersensitiveness to pain and to temperature

changes becomes extremely exaggerated. Anesthesia and analgesia may follow, or may be present from the beginning of the disorder. Motor disturbances include convulsive movements and twitching, which may or may not occasion pain; paralysis may follow the convulsions, or may be present from the beginning of the disorder. Trophic changes include a peculiar shining appearance of the skin, which is usually reddened; thickening of the nails, and dropping or whitening of the hair, or rarely an overgrowth of coarse hair may be found in the area of distribution of the affected nerves. Vasomotor changes include variable pallor and reddenings, and sometimes edema.

Treatment. The treatment depends absolutely upon the etiological factors present in each case, plus such palliative measures as may give relief. In painful cases, the affected area should be well protected from temperature changes, usually by wrapping in warm cotton wool, and by complete rest. Local neuritis must always be treated with complete rest of the affected part, if possible. Very gentle manipulations or none are to be given; massage is to be omitted during the painful stages of any neuritis. It is best to postpone correction of lesions closely related to the affected areas until after the acute pain has disappeared. Just absolute rest and protection is the best thing during the acute stage of neuritis.

After the acute stage has subsided, and in those cases in which the pain is not severe, the course of the nerve trunk from the periphery to the spinal origin should be investigated, and all structural perversions corrected as gently as possible. Corrections of bony lesions must be made in such a manner as to avoid irritation to the sensory nerves, rarely it may be necessary to postpone corrective work until the pain has disappeared completely. The corrections should then be made, in order to prevent later attacks. Complete rest of the affected limbs is important during the entire course of the disease.

The motor changes require especial care. During the convulsive stages the affected parts are to be kept quiet, and every sensory irritation of the entire body avoided. The affected muscles should be well protected from chill, usually by wrappings and cotton wool; these should extend well beyond the affected areas. When paralysis is present, during the acute stage there must be absolute rest. With the subsidence of the symptoms of acute inflammation, passive movements, then active movements and massage should be begun. The muscles are weakened, and overwork is to be avoided. The patient may be able to perform movements while he is in a warm or neutral bath, which would be impossible out of the water; this is an excellent exercise.

Trophic disorders may lead to bed sores; these are avoided by the usual good nursing and baths. A water or air bed is useful in severe cases.

Neuritis which is due to arteriosclerosis should receive treatment for that condition (see arteriosclerosis). Special attention should be given to any possible septic focus. Whatever poison is active should be removed as speedily as possible; alcoholics may have to be brought rather gradually to abstinence; other poisons are to be removed at once, and even alcohol can sometimes be suddenly stopped. Occupational causes must be met by change of occupation. Elimination of poisons is facilitated by thorough correction of the lesions and the reflex rigidities usually present; especially in the lower thoracic and dorso-lumbar region. Baths, enemas, special exercises, are sometimes indicated, according to individual needs.

The following treatment is illustrative:

"As the general system was much run down I advised the use of a nutritious diet, including raw eggs and milk and a liberal allowance of open air. The specific treatment consisted in, first relaxing the contracted cervical muscles followed by gently stretching the shoulder muscles and those of arm and forearm and the ligaments of the shoulder and elbow joints. This was preceded by deep inhibition all along the roots and trunks of the affected nerves, thus permitting deeper adjustive work. The nerves were gently stretched wherever possible. The subluxated cervical vertebræ were adjusted during the second month of treatment. The upper ribs were adjusted."—W. B. Keene.

Prognosis. In favorable cases, the recovery may be complete. When the nerves have been seriously damaged, some anesthesias or paralyses may remain permanently. The death of the axis cylinders may lead to atrophy or degeneration of the nerve cells of the anterior horn of the cord and the sensory ganglia; later ill effects may follow from these changes. When the inflammatory changes affect the muscles of respiration, death may occur from asphyxia. In the infectious cases, death may occur from heart failure, thrombosis, or exhaustion.

Prophylaxis. The use of arsenic and mercury in the treatment of diseases is much less frequent than formerly; alcoholism is decreasing; modern knowledge of nerve surgery prevents many traumatic cases; the use of poisonous substances in the trades is being constantly more closely supervised and controlled by law; all of these factors should lead to great diminution of the number of cases of neuritis.

NEUROMATA

Nerve tumors are rare; they cause varying degrees of pain and inconvenience.

Amputation Neuromata (stump neuromata) follow amputation of a limb, or section of a nerve. The nerve fibers grow out into the tissues, and often

form bulbous masses, or coiled fibers. They are usually avoided by modern methods of amputation; the treatment consists of surgical excision.

Nerve-Trunk Neuromata are tumors upon the nerves; they may be true or false, and may be extremely numerous. They may occasion no symptoms, or may be painful. The only treatment is surgical; and that is not often satisfactory, on account of the number of the tumors.

True Neuromata include nerve fibers, rarely nerve cells, with connective tissues for support, as in normal nervous tissues.

False Neuromata are connective tissue tumors growing upon nerve trunks.

REGENERATION OF NERVE FIBERS

When a nerve trunk has been cut, or its continuity severed in any way, the fibers degenerate peripherally to their endings, and centrally for one or several nodes. The fibers and the nerve cells of origin undergo certain changes, but do not necessarily die. If the cut ends are brought together, or if the pressure be removed, or other causes of injury be removed, regeneration may occur. This means that the ends of the nerve fibers above the degenerative processes begin to send out fibrils, which ultimately grow into the peripheral remnant of the nerve trunk, and reach the original field of distribution. Function is thus restored with varying degrees of completeness.

In surgical cases, the nerve ends should be sutured. Regeneration begins within a few days, and the fibers grow at an average rate of about one millimeter each day—this is subject to great variation. In cases in which the nerve is injured by pressure, as by tumors, exostoses, and other structural factors, the removal of the pressure may be followed by regeneration only slowly, if at all, and regeneration is less complete than in surgical cases.

Regeneration may be facilitated by correct treatment. The field of distribution of the injured nerve trunk must be kept in normal condition by bathing, massage, and sometimes by electrical stimulation of the muscles left without the normal nerve stimulation. Volitional attempts to move the paralyzed muscles seems to exert a helpful influence upon the motor nerve cells.

CHAPTER XL

DISEASES OF THE CRANIAL NERVES

The **Olfactory Nerves** are peculiar in having no medullary sheaths. They are not often diseased. Inflammations of the nasal membranes may destroy the olfactory nerve endings, in which case olfactory anesthesia or anosmia results—loss of the sense of smell. Injury to the nerve trunks or the olfactory bulbs or tracts may be due to fractures of the skull, brain tumors, or meningitis. Anosmia results from abnormal dryness of the nasal membranes, as in early acute rhinitis, or in disease of the nasal branches of the fifth cranial nerve.

Hyperosmia, olfactory hyperesthesia, may be present in hysteria or insanity, or as a congenital peculiarity. Delicacy of smell comparable to that of wild animals or dogs may be present occasionally in such individuals. Parosmia is often due to partial loss of smell; occasionally as a hysterical symptom it may be pronounced. Olfactory hallucinations are often present as epileptic auræ; they may be present in insanity and in hysteria.

In testing for olfactory variations, it is necessary to employ odorous substances which do not affect the common sensations; ammonia, smelling salts, pepper, act upon the fifth nerve and are useless. Aromatic oils are most useful for such tests. It is necessary to avoid too great stimulation with these, and very small quantities give most accurate results.

Treatment of the olfactory nerve disturbances is usually very unsatisfactory. Hysteria which shows olfactory symptoms is usually obstinate. (q. v). Nasal diseases may be treated; this may relieve the olfactory disturbance to a certain extent. The temporary loss of smell due to acute rhinitis disappears completely, in most cases. In all other olfactory disturbances the prognosis is bad for recovery. Fortunately, olfactory sensations are not essential to life or to comfortable living.

The **Optic Nerves.** Normal vision depends upon the activity of many nerves—the retina and the optic nerves; the sympathetic nerves which control the circulation, nutrition, and the action of the intrinsic eye muscles; and the motor nerves which control the extrinsic muscles. The activities of several brain centers are also essential to normal vision.

The **Retina** is subject to disturbances of several types.

Toxic Amblyopia is most often due to tobacco or alcohol; less often to lead or other poisoning. Central scotoma, especially

affecting the red-green fibers, is usually the first symptom; this is followed by progressive loss of vision. The disturbance may be a retro-bulbar neuritis.

Hemeralopia (day blindness) is characterized by inability to see clearly in a bright light, but vision is very clear in dim light. It may be due to abnormal dilation of the pupil; to albinism, cataract or it may not be possible to find the cause after careful examination.

Nyctalopia (night blindness) is most often due to syphilitic retinitis; less often to abnormal constriction of the pupils, and to retinal fatigue. In this disturbance vision is practically normal in a bright light, but fails completely in dim light.

Retinitis is characterized by progressive failure in vision, and its diagnosis is based upon the retinal examination; this shows the disease before the vision is affected, and should be made as a routine procedure in cases in which the condition is suspected. Albuminuric retinitis may be the first symptom of chronic interstitial nephritis; it may occur in any nephritis. The retina shares and sometimes precedes edema of other parts of the body. Retinal hemorrhages are frequent and may be serious.

Syphilitic Retinitis is usually associated with choroiditis; it occurs late in the disease. There are whitish or opalescent patches upon the retina.

Detachment of the Retina is present in wasting diseases; it is due to diminished intra-ocular pressure or to exudates back of the retina. Heavy falls, blows upon the head, and suddenly produced cervical lesions are causative.

Pigmentary Retinitis is chronic, usually attacks young adults with hereditary syphilis or some wasting disease; it is associated with progressively increasing pigmentary deposits upon the retina, with gradual loss of vision to blindness.

Retinal Hemorrhages occur in many systemic diseases; nephritis, leukemia, purpura; scurvy; pernicious anemia; arteriosclerosis; under conditions associated with high blood pressure, during parturition or muscular strain; it is recognized by the retinal examination. The blood may be absorbed and vision restored, or the injury may be permanent.

Choked Disk (papilledema) is a condition in which swelling or edema of the portion of the retina occupied by the optic nerve in transit causes it to project forward. It is recognized by the retinal examination, and is present in nephritis and in all conditions associated with increased intracranial pressure. It is an important factor in the diagnosis of brain tumor and certain other diseases.

Optic Neuritis is due to the causes of neuritis elsewhere (q. v.) especially to syphilis, alcoholism, and nephritis; mild cases may be due to refractive errors. If the process continues, optic nerve atrophy results. The treatment is indicated by the etiology. Vision may not be changed at first; the diagnosis can usually be made by the retinal examination. Pain is not present.

Optic Nerve Atrophy occurs in multiple sclerosis, and in the parasyphilitic diseases, tabes dorsalis, paretic dementia and taboparalysis; it may be the first symptom observed in these diseases. It may result from optic neuritis, may be hereditary, and is present in amaurotic family idiocy. It is characterized by variations in the color sense, followed by gradual loss of vision to blindness.

Destructive Lesion of either optic nerve causes blindness in the corresponding eye, with almost total loss of light-reflex in that eye. Inflammatory conditions of either eye may affect the other eye; for this reason surgical removal of an injured eye is often required in order to preserve the normal eye from injury.

The **Optic Chiasm** is injured by tumors of the pituitary body or by basal meningitis. The decussating fibers are chiefly affected, in most cases, and the result is blindness of the nasal halves of both retinæ; so that the patient seems to be looking forward into a tunnel. The macula retains its vision, and its field occupies part of the outermost limit of vision, in both eyes.

The **Optic Tracts** are also affected by tumors and basal meningitis; lesions anterior to the quadrigemina usually affect the motor nerves of the eyeball and sometimes other cranial nerves; a ray of light thrown upon the blind half of the retina in such cases may initiate pupillary contraction. This reaction is not always present. Lesion of either optic tract causes blindness upon the same side in both retinæ—bilateral homonymous hemianopsia.

The **Visual Cortex**—i. e., the region of the cuneus and the calcarine fissure—may be injured by blows, fragments of skull or osteomata; thickenings of the dura, however produced; tumors of various kinds, hemorrhage, or softening. When one side is destroyed, bilateral homonymous hemianopsia is produced; when both sides are destroyed, blindness may result, or the macula may escape, leaving fairly good vision for direct fixation. When the neighboring cortical tissue is destroyed, memory for the significance of things seen may be lost; such a patient sees fairly clearly, but without understanding; he cannot read, nor recognize persons; the condition is called "mind blindness." When an irritative lesion, as a throbbing aneurysm, affects the cuneus or calcarine fissure, flickering lights, vague visual sensations of several kinds, are present; when the neighboring or overflow areas are so affected, memories appear as visions, and various visions of angels, dead friends,

etc., may be described very clearly and in great detail. Such occurrences are not rare in old persons, and in early cerebral degenerations.

The **motor nerves of the eyeball** include both somatic and visceromotor fibers. The visceromotor fibers include those which govern the blood vessels, the pupils, the lens, and the nonstriated fibers of the levator palpebræ and the capsule of Tenon. The centers of the third nerve send fibers which terminate in the ciliary ganglion (sympathetic), and these innervate the circular fibers of the iris and the ciliary muscle. From this and related centers, fibers pass to the region of the upper thoracic spinal segments; fibers from the gray matter of that area pass to the superior cervical sympathetic ganglion, whence the gray fibers pass to the radiating muscle fibers of the iris, to the capsule of Tenon, the nonstriated fibers of the levator palpebræ, and the blood vessels of the orbit. The fibers which reach the eye by way of the upper thoracic spinal segments may be affected by lesions of the upper thoracic vertebræ, and indirectly by lesions of the cervical vertebræ. Thus, functional disorders of circulation of the conjunctivæ and the eyeball; ptosis, unequal and irregular pupils, may be caused; these may lead to later and more serious disturbances of the orbital tissues. Correction of the lesions as found is the only treatment required at an early time, but if the disturbance has been active for months or years, the tissue changes may be so marked that considerable time, and perhaps other treatment, are necessary.

Disease of the nerve centers or of the meninges along which the nerves pass may cause disturbed function of the nerves, either irritative or destructive, as the case may be.

Iridoplegia is paralysis of the muscles of the iris; several forms are described.

Myosis, or contraction of the pupil, may be produced by an irritative lesion affecting the third nerve fibers, or by paralysis of the sympathetic fibers from the upper thoracic segments. It is present in locomotor ataxia; sometimes in tabo-paralysis and paretic dementia. In these cases it may affect the pupils unequally. Tumors, etc., pressing upon the cervical sympathetic ganglion or the cervical sympathetic cord may produce unequal pupils. In any case, the fibers in either pupil may be unequally contracted, so that a "comma pupil" or "feline pupil" is produced.

Mydriasis, or dilatation of the pupil, may arise from irritative lesion of the sympathetics or from paralysis of the third nerve fibers or visceromotor center of the third nerve. Lesions of the upper thoracic vertebræ are most often followed by slight mydriasis; this may be unequal.

Cycloplegia is paralysis of the ciliary muscle; vision is unchanged for distant, but accommodation for near objects is lost.

Accommodation Iridoplegia is characterized by absence of the contraction of the pupils on near vision. The pupils may contract when the lids are closed, or such motion is attempted. It is most often found in paretic dementia.

Ophthalmoplegia Interna is characterized by loss of the pupillary reflexes, both for light and for distance.

Argyll-Robertson Pupil is one in which the pupils change normally to distance variations, but not to light, "light reflex iridoplegia."

The somatic motor nerves are distributed to the extrinsic eye muscles. These may be irritated and thus spasm is produced; or destroyed, when paralysis follows. Functional variations may occur also; usually either twitchings of the muscles or weakness of one or more of the eye muscles is produced.

Lesions of the extrinsic motor nerves occur as the result of syphilitic or alcoholic meningitis, brain tumors, fracture of the base of the skull, and in other less well recognized conditions. The sixth nerve has the longer course upon the meninges, and is most often affected.

These nerves innervate special muscles—the sixth, the abducens; the fourth, the superior oblique; the third, all the others—but the nuclei of these are so intimately related, especially in the control of antagonistic muscles, and in so many cases fibers arising in one nucleus are distributed with the fibers of another nerve, of the same or of opposite sides, that the special symptoms observed do not always indicate the exact anatomical lesions.

Nystagmus is a rapid motion of the eyeballs, due to alternating contractions of muscle opponents. It is rarely a congenital neurosis; and is a symptom in Friedrich's ataxia, insular sclerosis, Meniere's disease, meningitis, and other diseases of incoördination. It may appear temporarily on voluntary movement in eyes with a weakened or partially paralyzed muscle. It is usually present in albinism.

Strabismus occurs when the weakness of any muscle prevents correspondence of the axes. The deviation of the paralyzed eye is called the "primary deviation"; when this eye is fixed, the normal eye suffers from overaction of the corresponding muscle; this is termed "secondary deviation." It does not occur in strabismus due to spasm, and its presence indicates paralysis.

Ocular Vertigo is due to the effect produced in consciousness by imperfect vision, whereby the objects appear farther away than normal, on account of the increased effort required for fixation in partially paralyzed eyes; the incongruousness between the eye

efforts and the information of other senses gives a peculiar and distressing sense of dizziness.

Diplopia, or double vision, may result from strabismus. It occurs also as a neurosis, and in wasting diseases.

The **Fifth, Trigeminal, or Trifacial,** nerve has such a wide area of nuclei of origin and insertion, and its fibers so intricately interlace with the fibers of other nerves, that it is difficult to decide, in any given patient, whether the fifth nerve alone is involved or whether other nerves also are involved. Its broad and long nuclear relations render it very improbable that a nuclear disease affecting the fifth nerve does not also affect other nerves. The nerve trunk may be variously diseased, rarely as a whole, but frequently as one of its branches. (See neuralgia.)

Sensory disturbances of the fifth nerve are varied. Irritative lesions cause various neuralgic pains and paresthesias in the area of distribution. Abnormalities of taste vary in individuals, apparently. Parageusias are recorded in irritative fifth nerve lesions; in other cases with apparently identical pathology, the sense of taste remains unaffected.

Destructive lesions cause anesthesia in the area of distribution of the nerve, or of its injured branches. Variations in the sense of taste are sometimes reported. Injury of the sensory portion of the nerve causes various trophic changes also. Dryness of the olfactory membrane may cause anosmia; dryness of the conjunctivæ may result in injury to the orbital tissues. Corneal ulcers are frequent. Herpes and increased liability to infection result from the loss of sensory impulses, or of trophic control of the tissues.

Irritative motor lesions cause either tonic or clonic spasm of the muscles of mastication. Tonic spasm is most common in tetanus, tetany, and hysteria. The clonic spasms most often occur with other muscular disturbances, as in paralysis agitans, chorea, and general convulsions.

Destructive lesions cause paralysis of the muscles of mastication. The fibers of the fifth which supply the mylohyoid, digastric, and tensor tympani, do not show symptoms when the nerve is paralyzed. It must be remembered that the area supplied by the fifth nerve is overlapped greatly by other nerves, and that there is also much overlapping of the right and left areas of innervation.

The **Seventh, or facial** nerve is the nerve which controls the muscles of expression. It is frequently subject to functional and structural diseases; and the diagnosis of its various affections is usually rather easy.

Sensory disturbances are not marked. Common sensation in the skin and mucous membranes of the lower part of the face

and the mouth is sometimes slightly changed, in lesions of the nerve trunk. Loss of taste is variable, and occurs when the nerve lesion lies between the geniculate ganglion and the beginning of the chorda tympani nerve. Disturbance in the buccal secretion occurs when the lesion lies centrally to the beginning of the chorda tympani; this is usually a diminution of the salivary flow.

Motor disturbances may be irritative or paralytic; paralysis may be of the upper or the lower neuron type.

Spasms of the facial muscles may be either functional or associated with gross lesions. Functional disturbances include the various tics, choreic movements, and hysterical neuroses. (q. v.) Athetoid movements may appear in lesions of the basal ganglia, especially the striata, whether congenital or due to tumors, etc., and are often associated with other localizing symptoms. Variable spasms may be due to injury to the facial nerve as the result of middle ear disease.

The bony partition between the middle ear and the facial nerve is thin, often defective, and is easily penetrated in diseased conditions. The nerve is thus left with only membranes; these may even be destroyed; and only the nerve sheaths are left for protection. Anything which causes variation in the circulation through the ear membranes, such as cold, nervous disturbances, bony lesions of the cervical and upper thoracic vertebræ, extension of infection from the throat or other tissues may thus bring pressure upon the nerve trunk, or permit the infection of the surrounding tissues.

When the pressure thus produced is variable, the spasms vary in intensity and in location. Increasing pressure causes first spasm, then paralysis of muscle fibers or muscle groups, in turn; sensory disturbances and secretory disturbances may also vary, according as the pressure irritates, inhibits, or destroys the nerve. In many cases supposed to be due directly to cold, the middle ear disease is the intermediate factor in the etiological series of events.

Paralysis of any branch of the facial nerve is usually due to injury of that trunk alone; a comparison of the symptoms with the anatomical distribution of the nerve branches will usually give the location of the injury within narrow limits.

Bell's Palsy is lower neuron paralysis of the seventh nerve. It is characterized by flaccidity of muscles, which do not react to reflex stimulation nor to emotional states; and which give the reaction of degeneration to the electric tests. The lips and eyelids drop, and the saliva and tears flow freely, as a rule. After some weeks or months, the paralyzed muscles shrink, causing various deformities of the face. Many of these cases can be completely relieved by early attention to the upper cervical region.

The muscles of expression have little or no bony attachments; and antagonistic muscles are lacking or are not exactly antagonistic; thus the effects of this later shrinking of the muscles is not like that produced in paralyses of the other parts of the body, where the contractions resulting from paralysis are associated with hypertension of the muscle antagonists. The shrinking of the facial muscles produces a mask-like drawing of the face, so that at first the normal side appears to be the paralyzed one. Attempts at whistling or blowing show the true condition.

Hyperacusia is said to be due to paralysis of the stapedius, in facial nerve paralysis. Probably the effects produced upon hearing in Bell's palsy are more often due to the fact that disease of the middle ear is a common cause of the paralysis rather than that the paralysis itself exerts any marked effect on the intrinsic muscles of the ear; occasionally, no doubt, the latter factor is of some importance.

Upper neuron paralysis of the facial nerve is characterized by increased tension of the affected muscles; the mouth is drawn upwards, the wrinkles are deepened; saliva and tears are normal; reflexes are exaggerated or normal; taste is normal or very slightly affected; emotional states may cause movements of the affected muscles in variable degree or the paralysis may involve the emotional reactions also. Rarely the face alone is involved in upper neuron paralysis; usually the limbs are also included.

Lesion of the pons, taking in the decussation of the nerves, may cause bilateral facial paralysis. Two lesions, affecting the facial centers, may cause bilateral paralysis; occasionally an upper neuron paralysis on one side may be associated with a lower neuron paralysis of the opposite side. Various complicating factors may occur under such conditions.

Treatment. In addition to the usual features of treatment of cranial nerve diseases, the facial nerve should receive attention from the standpoint of the ear affection. Relief of the diseased condition of the middle ear may prevent further development of the paralysis of the seventh nerve, and may permit a return to normal function on the part of fibers which have been affected by the pressure but have not been destroyed.

In cases which do not yield to conservative measures, the surgery of the nerve trunks may be useful. The hypoglossal may be sectioned, and its central end sutured to the peripheral end of the facial; paralysis of the hypoglossal is less serious and annoying; much reëducation is necessary before restoration of normal condition of the facial muscles.

The **Eighth** cranial nerve is composed of two physiologically distinct parts. The **Auditory** portion is stimulated by sound

waves, and is important in conveying these impulses to the brain. Higher cerebral activities depend in great measure upon the sounds received by the auditory apparatus and the nervous effects of these in the central nervous system.

The **Vestibular** portion is stimulated by varying pressure conditions within the semicircular canals, and is important in the effects produced upon the coördinating apparatus. Little information is conveyed by this apparatus, but equilibrium is maintained and the efficiency of certain body movements is secured through the vestibular apparatus.

Deafness may be due to any one or more of a great number of causes. The membrane of the middle ear is continuous with that of the Eustachian canal and the pharynx; infections of this region are readily carried to the ear, with varying subsequent effects upon the otoliths, tympanum, and lining membranes. Injury to the internal ear, affecting the cochlea with the membrane, organ of Corti, and nerve endings may follow middle ear disease, or may arise independently, from hemorrhage, infections, the effects of alcoholism or syphilis. Deafness may be due to involvement of the auditory nerve itself, either within the canal in the petrous portion of the temporal bone, or within the skull. Cerebellopontine tumors often cause deafness; this may be a very early symptom.

Atrophy of the auditory nerve may occur in tabes. Injury to the auditory cortex may interfere with hearing, but rarely causes deafness.

Mind or word deafness is due to injury of the cerebral cortex in the auditory overflow areas—the psychic auditory areas. In this condition hearing is reasonably acute, but the significance of things heard is lost—words are heard, as if they were in a foreign language. A certain degree of aphasia is usually associated with this condition.

Hyperacusis is a condition in which all sounds are intensified. Paralysis of the stapedius muscle allows low tones to be heard with especial distinctness. Neurasthenic individuals are affected uncomfortably by sounds, and complain of their loudness; rarely is audition more efficient in neurasthenia. In hysteria, there is often increased hearing; sounds may be heard and interpreted with greater facility than in normal individuals.

Dysacusis is difficult hearing. It is sometimes applied to partial deafness. It may be due either to middle ear or to labyrinthine disease. When due to middle ear disease, bone conduction is better than air conduction; the tuning fork placed upon the skull can be heard longer than when held near the ear, in the air. When

the labyrinth or the nerve itself is at fault, bone conduction and air conduction are about equally diminished.

Tinnitus Aurium, or ringing in the ears, may be due to a number of very different causes. In anemic or neurotic individuals they appear to be due to an abnormal appreciation of the sounds produced by the circulation of the blood—these are synchronous with the pulse. Noises which are unnoticed by normal individuals may arouse unpleasant sensations; this can be determined by closing the ears, and noticing the cessation of the sounds supposed to be tinnitus.

Hardened ear wax causes varying crackling, ringing, buzzing noises. Other causes of tinnitus, which affect the middle ear or the labyrinth include the effects of poisons, as quinine, alcohol, or certain diseases, otitis media, arteriosclerosis, brain tumor, or aneurysm. Irritation of the cervical sympathetic chain, and bony lesions affecting the cervical and upper thoracic spinal centers, may cause tinnitus, through varying the circulation through the ears or the general blood pressure. Attacks of migraine and epilepsy may be preceded by tinnitus.

Noises due to involvement of the nerve trunk are less common. Irritative injuries to the auditory cortex cause sounds which are usually complex; words, and even long speeches, often associated with visual hallucinations, may be reported by the patient with great detail; he is usually very certain that these have an extrasomatic origin.

Meniere's Disease. Disease of the labyrinth, associated with vertigo and disturbances in equilibrium, usually with tinnitus and partial deafness, sometimes with vomiting, is called Meniere's Disease. It is most common in men past thirty, and is due chiefly to syphilis, alcoholism, gout, senility, or hemorrhage into the vestibule or the semicircular canals. It may be precipitated by gastric disturbance, emotional shock, or blows or falls. A single attack may persist, or a series of attacks may occur; unless the cause is removed, the vertigo, nausea, and other vestibular symptoms persist until the destruction of the affected nervous elements; then the centers for equilibrium and coördination become adapted to the conditions, and no further symptoms are noticed. The deafness becomes permanent.

Similar symptoms may be produced by tumor in the cerebellopontal region, or by basal meningitis. Other symptoms of meningitis or of tumor should make the diagnosis fairly easy.

Treatment must be based upon the cause of the attack, and upon its severity. The recumbent position may give relief. Thorough treatment to the cervical spinal column, with correction of whatever lesions may be found, may relieve, probably through relieving the congestion in the vestibule. Counterirritants to the mas-

toid may be helpful. If the patient can be kept fairly comfortable until the death of the neurons concerned, his later life is not affected.

The **Glosso-pharyngeal** nerves are so closely related to the other basal nerves, and their areas of distribution are so thoroughly overlapped by the distribution of neighboring nerves, that almost nothing is known of their diseases. Disturbances in taste are due to involvement of these nerves, but individual variations are common, and the fact of disturbed gustatory sense is not conclusive. Its disease is probably always associated with disease of the vagus.

The **Tenth, Vagus, or Pneumogastric** nerve has such intimate relationships with the ninth, eleventh and twelfth cranial nerves that it is practically impossible to make exact ante-mortem diagnosis in cases in which many branches of the vagus, or any complicating factor whatever, are present.

The vagus is subject to the usual causes of basal meningitis and increased intracranial pressure, such as syphilis, chronic meningitis, toxic influences, and tumors. During its course through the neck its proximity to the pulsating carotid modifies the symptoms produced by direct pressure upon the common sheath. In passing through the superior thoracic inlet the nerve trunk may be subjected to pressure by goiter, tumors, aneurysm, anterior curve of the cervical spinal column, and other less frequent factors. Hypertension of the scaleni and other muscles of the anterior cervical group may raise ribs and clavicles and may also, by their swellings, diminish the size of the thoracic inlet and exert more or less serious pressure upon the vagus-carotid-jugular sheath. Wounds and surgical operations in the neck may injure or sever the vagus. Neuritis, especially diphtheritic and toxic, may affect it also.

The vagus is peculiar in being only very indirectly and feebly subject to volitional control, yet it is, in all its branches, very urgently affected in emotional, and still more, hysterical, control. It may almost be called the "hysterical nerve."

The **Pharyngeal** branches are intimately associated with the branches of the glossopharyngeal nerves, in the pharyngeal plexus. Spasm of the pharyngeal muscles is usually hysterical, "globus hystericus." Paralysis of these muscles causes various disturbances in deglutition; when the soft palate is paralyzed also, the food passes into the nose. This disease is usually part of a glosso-labio-pharyngeal paralysis, and is usually nuclear. (See bulbar paralysis.)

The **Laryngeal** branches are both sensory and motor. The winding of the left recurrent laryngeal around the arch of the aorta and of the right around the subclavian artery, subjects these nerves to the effects of aneurysm of these vessels; the left nerve

is also affected by pressure from the dilated auricle in mitral stenosis.

Laryngeal spasm is most frequent in children (see laryngismus stridulus). It may occur in adults as part of a general neurosis of various types; in hysteria; as an equivalent for migraine; as a crisis in locomotor ataxia, and under other even more rare conditions. It causes dyspnea, which reaches apparently a severe stage; the accumulation of carbon dioxide finally so affects the respiratory center as to produce relaxation. Death never occurs from asphyxia due to this alone, though in organic diseases of the nervous system or the heart, death may be precipitated by such an attack.

Laryngeal paralysis is usually bulbar and is generally bilateral. Rarely cerebral lesion may occasion upper neuron type of laryngeal paralysis; this is practically never limited to the area of the laryngeal muscles. The weakness of the laryngeal muscles that comes from overuse, as in "clergyman's sore throat" or as part of general weakness, must not be confused with true paralysis of the muscles. In complete bilateral paralysis phonation and coughing are impossible; respiration is unimpeded, though there may be some harshness of the respiratory sounds, due to the relaxation of the cords, perhaps also to some swelling of the mucous membranes. In unilateral complete paralysis the symptoms are variously modified.

Paralysis of the abductors permits the approximation of the cords by the unopposed adductors. Various whistling and stridulous sounds are caused by the long, sometimes difficult respiratory movements. Bilateral paralysis of the abductors may at any time become very serious from swelling of the membranes; asphyxia may be fatal. In unilateral paralysis the voice is hoarse and low; ultimately contractures result in about the same condition as in bilateral paralysis.

Paralysis of the adductors leads to loss of phonation; coughing is normal; there is no stridulent tone, and no dyspnea. This is usually hysterical; overuse of the voice may result in fatigue that may be practically identical with paralysis. Usually recovery is to be expected upon relief of the etiological factors.

Sensory paralysis of the laryngeal nerves may allow the food to enter the larynx and trachea; aspiration pneumonia or immediate suffocation may result.

The **Cardiac** branches. Irritation of the vagus produces slower heart beat. When the irritation is long continued, the action of the heart reflexes, with the variations in blood pressure thus produced, result very often in an irregular beat. The proximity of the pulsating carotid prevents pressure inhibition of the

vagus, such as may occur in most nerve trunks of the body. Cervical lesions affect the vagus both through muscular contraction and through the reflex action of the centers in the upper cervical cord and the medulla. When both right and left nerves are destroyed the outlook is grave; the condition is rarely recognized ante-mortem, on account of the speediness of death. Most affections of the cardiac branches are functional. (See cardiac neuroses.)

The **Pulmonary** branches are related with sympathetic branches and are distributed to the blood vessels and to the nonstriated muscle fibers of the bronchioles; trophic fibers are not certainly proved. Sensory fibers controlling the action of the respiratory centers are present. Irritation of these nerves causes attacks of asthma. Respiratory movements are influenced by many factors, and are thus of variable value in diagnosis.

The **Gastric, Intestinal, and Esophageal** branches of the vagus have such intimate relations with the sympathetic and the splanchnic nerves, and the symptoms referable to these various nerve trunks and their related centers are so complex, that it is difficult to distinguish between organic and functional disturbances of the different groups. Nausea is caused by irritation of the vagus or its center; vomiting may occur. Bilateral lesion of the vagus causes persistent disturbance of digestion, usually with vomiting of bile, sometimes of feces, with other symptoms of acute intestinal obstruction.

Vagotony is the term applied to a symptom complex supposed to be due to increased action of the vagus. It includes constipation of the spastic type, with attacks of diarrhea and mucous colitis, which are precipitated by slight nervous disturbances, or by drinking hot or cold water. Pain in the colon, very severe just after defecation, is often a prominent symptom. The heart is slow, asthma is often present; variations in perspiration and in vasomotor control are frequent. The treatment is chiefly symptomatic. Reflex causes of disturbed function must be sought and relieved.

The **Spinal Accessory, or eleventh** nerve is composed of two distinct parts; one joins the vagus, and is probably properly considered as a part of that nerve, since it is derived from the vagal nucleus. The other part represents a spinal nerve trunk, and is derived from the upper cervical spinal segments. It is distributed efficiently only to the trapezius and the cleido-mastoid muscles.

Spasm of the second of these, with or without associated spasm of the first, causes wry neck. This is often a temporary condition, as in the wry neck caused by rheumatism or cold. It is then either due to disturbed nerve action, or, more frequently, to a

muscular rheumatism affecting the cleido-mastoid, with other neighboring muscles. Spasms may be present in chorea or in tics. Congenital wry neck is properly a deformity, a shortening of the muscle of the affected side. It may begin primarily as a muscle spasm, with ultimate shortening of the contractured muscle. (See Torticollis.)

Paralysis of these muscles causes the shoulder to droop, and those arm movements which depend upon shoulder-girdle fixation become impossible or difficult. This type of paralysis is always of the lower neuron type; upper neuron lesions affecting these muscles include other muscles also.

The most common causes of organic disease of the eleventh nerve are caries of the upper cervical spinal column, meningitis, or the usual causes of neuritis. The most common causes of functional disturbance are rheumatic, toxic, or the result of emotional disturbances.

The **Hypoglossus, or twelfth** cranial nerve, is rarely affected alone. Irritating lesions cause spasm of the tongue; this is most commonly caused by hysteria, and not by organic lesion.

Destructive lesions result in paralysis; the tongue is drawn to the affected side, by unopposed action of the normal muscles. Nuclear and infra-nuclear lesions cause paralysis with loss of reflexes and muscular atrophy; supra-nuclear lesions cause loss of voluntary movement, but not of reflexes, and without atrophy. Little evil effect follows this paralysis.