Secondary degeneration may follow transverse myelitis or compression-paraplegia; or it may be due to central disease, and the symptoms proper to those affections will also be present. Secondary degenerations are believed to be permanently incurable.

Spastic Spinal Paralysis—Spastic Paraplegia—Simple Primary Sclerosis of the Lateral Columns.—The sclerosis affects the posterior part of the lateral columns of the cord symmetrically. It is a *rare* primary disease of the cord, affecting adults between the ages of thirty and fifty. It occurs also as a congenital condition, and it is believed to be sometimes due to injuries at birth.* The *causes* of primary lateral sclerosis occurring in adults are quite unknown. It affects males more than females. The *symptoms* are very like those of the secondary degenerations, which affect the same tract of fibres in the cord, although in primary lateral sclerosis the disease does not extend in the lateral columns quite so far posteriorly. There are spasms, rigidity, and contraction, with motor weakness, and subsequently paralysis of the lower limbs. The deep reflexes are exaggerated, the knee-jerk and *ankle-clonus* being marked; and the gait is characteristic. The patient is said to walk like a "Highland piper." The legs get soon wearied, and the back may be stiffly arched when taking exercise. The bladder and bowels are unaffected; and there is no pain nor impairment of sensibility. The disease may run a course of ten or fifteen years. The patient may die of phthisis or some intercurrent malady. The *treatment* of primary lateral sclerosis is not satisfactory. Galvanism seems to be the most suitable treatment. Nitrate of silver is often prescribed, and Fagge advocates the use of the extract of calabar bean.

The Combined Sclerosis.—(1) A form associated with pernicious anæmia and other cachectic states, is known as "Putnam's type." There is rapid progression with paræsthesia, and sometimes anæsthesia, ending in the paraplegia, generally *spastic* in character. The sclerosis is in the posterior and lateral columns.

(2) *Hereditary spinal ataxia* (*Friedreich*), including two other forms—viz., hereditary cerebellar ataxia, and hereditary ataxic paraplegia. In Friedreich's ataxia, the sclerosis is in the posterior and lateral columns and clinically the ataxia ascends and gradually involves the upper limbs and speech. In *cerebellar ataxia*, the cerebellum is atrophied. It is a disease of early life (10 to 30 years). The gait is that of a drunken man. There is general inco-ordination and jerking. The speech is ataxic. There is mental deficiency. In *hereditary ataxic paraplegia* the gait has more of the spastic character. The mind is not affected.

Alcoholic, Syphilitic, Hysterical, and Reflex Paraplegia.—This group may be usefully alluded to here, as the striking symptom of all is the paraplegia, which often requires differentiation. The alcoholic form has been referred to under neuritis, the syphilitic under myelitis, &c., and the hysterical under hysteria. A *senile* paraplegia has been described, and the Caisson disease (Diver's paralysis) is of the nature of a paraplegia. Many of the so-called

* Little's disease.

cases of *reflex paraplegia* have been shown to be really cases of myelitis; and some authors consider this form, occurring in cases of chronic visceral disease, to be caused by *extension* of inflammation to the cord rather than by a reflex *inhibitory* action. The *symptoms* in all forms consist of more or less complete paralysis of the lower limbs, with impairment of sensibility. In the *alcoholic* form it is more frequently incomplete (paresis). Constitutional *syphilis* does not affect the spinal cord so frequently as the brain. The functional condition alluded to here does not include compression-paraplegia from syphilitic tumours or growths. It improves with anti-syphilitic remedies. The *hysterical paraplegia* is generally complete when attempts are made at walking. The patient, however, can generally raise the leg when in the recumbent position. There is impaired sensibility, and anæsthesia and hyperæsthesia are often marked. *Reflex paraplegia* may be caused by tight stricture of the urethra; disease of the urethra and bladder; chronic kidney disease; dysentery; intestinal worms; or a displaced uterus, &c. Many other causes are conceivable. When due, reflexly or otherwise, to severe organic disease which cannot be cured or alleviated, this form of paraplegia is not so favourable as the other three members of the group—in all of which a complete cure is possible. The *diagnosis* is chiefly inferred from the history and surroundings, and by exclusion of the other, and more serious members of the paraplegic group. There is no tendency to the formation of bed-sores, no rapid muscle waste, and the bladder and bowels are not paralysed. The *treatment* is considered elsewhere.

Spinal Weakness and Irritation. Spinal Anæmia, and Congestion (?).—There is still a difference of opinion with regard to this group—some authors describing all four conditions as separate and distinct affections, while others are inclined to attribute the symptoms of spinal weakness or irritation to anæmia or congestion of the cord. They all seem to be closely associated.

Spinal Weakness is characterised by muscular fatigue with pains in the legs. The patient complains of feeling easily tired, and occasionally he has feelings of numbness or formication in the feet. "Irritable weakness" of the generative organs is often present. The *causes* are over-fatigue of body and mind, and sexual excesses. The *diagnosis* can only be made after exclusion of all grave organic disease of the cord. The condition is often associated with the hysterical; and the same may be said of spinal irritation.

Spinal irritation or "*rachialgia*." This affection is often superadded to the preceding one, if it may not be considered a more violent form of it. The additional characteristic symptom is the great pain and tenderness on pressure upon certain regions of the spine. The cervical and dorsal regions are the usual seats of the disorder. It is believed to be of the nature of a neuralgia affecting the posterior spinal roots, and neuralgic affections are often present along with the spinal irritation. The pain is always increased by movement, and a hot sponge passed down the spine may produce intense burning sensations. Some cases are related in which pres-

sure upon the vertebræ has set up such agony as to render the patient insensible. The older writers believed the cause to be congestion; Hammond and others believe it to be associated with *anæmia* of the cord. Uterine disease is a common cause; and certainly females are affected with rachialgia more than males. Railway accidents, and shocks to the spine of any kind, are also causes of this disease.

Anæmia of the Cord.—This affection is believed by many to be the true pathological condition giving rise to the symptoms of the two preceding disorders. It is still doubtful. Paralysis in animals may be produced experimentally by cutting off the blood supply to the cord. Embolism may produce a like result, theoretically; and severe hæmorrhage might give rise to anæmia of the cord. The *symptoms* consist of numbness and formication in the feet, paresis, and sometimes paralysis, with the signs of spinal irritation and weakness.

Congestion of the cord is said sometimes to produce similar symptoms to *anæmia*; and a paralysis due to one or other of these conditions is said to be differentiated by the fact, that in congestion the patient is benefited or relieved by the prone or upright positions, and is much worse if allowed to lie upon his back; while in *anæmia* of the cord the reverse is true, and the patient should be kept as much as possible in the recumbent position. Authors do not agree that this differentiation can always be made. *Concussion* of the spine may produce a congestion of the cord and paraplegia; but this subject is more properly considered in surgical works.

In the *treatment* of the neurasthenic group the severe cases may require absolute rest. The prone position may be adopted in the *congestive* cases, if such be found beneficial. In milder cases the patient is allowed to go about, unless movement excites irritation. A generous diet should be allowed, and wine may be taken in moderation. Moderate exercise and a change of air are to be recommended. Quinine, iron, strychnine, and cod-liver oil are all useful. During the early stages, leeches, or cupping over the spine, are highly beneficial; or blisters may be applied. Morphia may be injected subcutaneously in cases with severe pain. Galvanism of the spinal cord is often useful.

Landry's Acute Ascending Paralysis.—This is a rare disease of the spinal cord, believed to be due to a poison of microbic origin. Abnormal sensations may at first be complained of, as numbness, formication, and great muscular weakness; but the characteristic symptom is the paresis, then paralysis, of the feet and lower limbs, gradually extending to the body and arms. Swallowing becomes difficult, and death may result in a few hours or days, from asphyxia—the medulla being invaded. A case may extend to ten or twelve days or even longer. Some few cases recover. The bladder and bowels are not paralysed, and bed-sores do not form. There are no symptoms of sensory disturbance, the disease being confined to the motor tracts. There are no spasms in the muscles, and the limbs lie flaccid, although the electrical contractility is always present. The reflexes are present at first, but after a few days they are lost. There is no treatment known to be of any benefit.

Locomotor Ataxia.—The histological changes are a degeneration of the posterior root fibres within the cord, followed by an increase of the connective tissue. The *sclerosis* of the posterior columns chiefly affects the dorsal and upper lumbar regions of the cord, but occasionally it extends as high as the cervical regions, and may involve the sensory cranial nerves.

In the lumbar region the *external* divisions of the posterior column are most affected; while higher, and especially if found in the cervical region, the *inner* divisions, or columns of Goll, suffer most. The posterior spinal ganglia are sometimes affected; and the peripheral nerves (neuritis). Grey degeneration sometimes affects the optic nerves, and sometimes the third, fifth, and sixth nerves. The joints are sometimes disorganised.

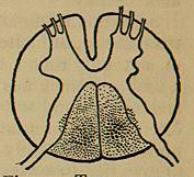

Fig. 37.—Transverse section of cord in locomotor ataxia.

The **causes** of locomotor ataxia are:—Severe chills, sleeping on damp ground, sexual excesses, syphilis, over-fatigue, injuries and concussion, and hereditary influences of a neurotic character: syphilis being the most important single factor. It is said, sometimes, to follow acute fevers, diphtheria, acute rheumatism, and pneumonia. It is more frequent in males, and the cases are most numerous between the ages of thirty-five and fifty years.

The **Symptoms.**—It is important to keep the *early* symptoms of locomotor ataxia well in view, as mistakes are very readily made during the slow and gradual development of the disease. (1) Pains in the lower limbs—frequently ascribed to rheumatism—are very common, and they usher in the more characteristic "lightning" or "toothache-like" pains so often present later. They occur in paroxysms and the patient may be free of them for days, or weeks, at a time. Changes in the weather, over-fatigue, and strong mental emotions are said to excite them, and they are not always confined to the legs, but sometimes they spread to the trunk and more rarely to the arms. (2) "Gastric crises" are common, consisting of periodic attacks of stomach disorder, *i.e.*, flatulence, vomiting, and lightning pains in the abdomen, with giddiness, palpitation and general *malaise*. Other "crises" are *laryngial*, cardiac, &c. (3) Diplopia, strabismus, nystagmus, inequality of the pupils, impairment of vision, or colour-blindness—are all early symptoms. Optic atrophy, with the characteristic ophthalmoscopic changes, is frequent. The paralysis producing diplopia and strabismus, &c., is often transitory. "Spinal myosis" (extreme contraction of the pupils) is a very common condition, both early and late. The "Argyle-Robertson symptom" consists of insensibility of the pupils to *light*, the contraction with accommodation for near objects, still being present. It may occur

as an early symptom and then it generally persists throughout the course of the disease. (4) *Joint affections* may occur early (Charcot's disease). The knee is commonly the first joint affected, and there is no pain, redness, or heat, and no history of an injury.

In a developed case of locomotor ataxia the gait is very characteristic. There is a more or less complete loss of co-ordination. The foot is lifted high, thrown out, and brought to the ground with a stamp. It is most marked when the patient *begins* to walk, and then he looks as if balancing himself, and he requires to keep his eyes open. The gait is made more obvious when the patient is directed to walk along a line—the course taken being very irregular and "shaky."

Sensation is impaired and numbness, formication, anæsthesia, and paræsthesia, are often present. "Girdle sensation" is complained of, not only round the waist, but sometimes felt as a band round a limb. Sometimes it is present as an early symptom. The patient feels the ground soft under his feet or like "walking upon clouds," &c. *Painful* sensation is often absent when simple *tactile* impressions are readily noticed; and sometimes a gentle touch appears to give rise to severe pain, when the limb is quite analgesic. The impressions are often retarded in their course to the brain. Cold and hot test tubes are generally discriminated, even after common sensation is lost. Sometimes this sense is also impaired. A very striking symptom, and often an early one, is the swaying and inability to balance the body steadily, whenever the eyes are closed. Although this symptom is common in locomotor ataxia it is not pathognomonic, as it occurs in other spinal diseases. The condition is usually present when there is defective sensation in the lower limbs, whether due to locomotor ataxia or to other spinal disease. There is a loss of the muscular sense, and this may be demonstrable in other ways, as when the patient is asked to point to his toe after the leg has been moved—when he often indicates a point far from it. The muscular sense may also be tested by weights. The sense of resistance is reduced or abolished. The contractile power of the muscles is generally normal. Incontinence of urine, and sometimes unconscious passage of the bowels, especially in cases where anæsthesia is marked; and impairment of the sexual functions with nocturnal emissions—are late symptoms. During the early stages the sexual functions are excited—the genital organs being irritable. The knee-jerk (tendon reflex) is almost invariably absent; and this, in typical cases, is an *early* symptom. The muscles do not rapidly atrophy, and their electrical re-actions are normal. The subsequent end of locomotor ataxia is often a state of complete paralysis with atrophy of the muscles, bed-sores, cystitis and paralysis of the bladder. Sometimes the disease spreads to the upper extremities, and then after premonitory symptoms of numbness and pain in the fingers, there is loss of co-ordination and inability to use a knife and fork, or to button the clothes, &c. In rare cases the upper extremities are attacked first. Cerebral disease may ultimately develop; but many cases seem to become quiescent for years. Periods of six, twelve, and even twenty or thirty years are quoted as the *duration* of locomotor ataxia. Phthisis is a common intercurrent disease.

The **treatment** recommended by Erb is galvanism of the spine. Absolute *rest* is indicated. Nitrate of silver is believed to be a useful remedy, given with the usual precautions against *argyria*. Iodide of potassium should be given in suitable cases. *Cold-water* douches and packs are also recommended by Erb. The application of mustard or the use of blisters, sometimes relieves. Morphia is often required for pain. The general condition may be improved by cod-liver oil and phosphates. Antisyphilitic remedies will be used in proper cases, but sometimes mercurials appear to be hurtful. Good effects have been recorded as to the benefits of *suspension* of the patient by the chin and occiput. The clothing should be warm, flannel being always worn. The diet should be generous, but coffee, tea, and alcohol should be given up. Tobacco is hurtful.

The **diagnosis** of locomotor ataxia is often very obscure during the early stages, and care must be taken that *neuralgic* or *rheumatic* pains in the limbs are ascribed to their true cause. The eye symptoms, loss of the knee-jerk, and the inability to stand with the eyes closed and the feet together, should suggest a thorough examination. *Joint affections* may be mistaken for surgical diseases. In a developed case the diagnosis is usually easy. *Disease of the cerebellum* gives rise to no spinal symptoms, and the gait is staggering, with giddiness, vomiting, and other head symptoms. A *chronic spinal sclerosis*—especially when the posterior columns of the cord are much affected—may simulate a case of locomotor ataxia; but the absence of lightning pains, eye symptoms, &c., and the presence of the knee-jerk—which is usually exaggerated in sclerosis—the gait, and the presence (in multiple sclerosis) of headache, giddiness, tremors, and defective speech, &c., may serve to differentiate these diseases. *Multiple neuritis* (*pseudo-tabetic form*), *hereditary ataxia*, and *neurasthenia* have to be noted. *General paralysis* of the insane is excluded by the prominent cerebral symptoms, and the gait.

Spinal Meningitis—*Acute and Chronic.**—The membranes, in the *acute* form, become hyperæmic. There are minute extravasations, and the surrounding tissues are infiltrated with serum. The exudation becomes fibrinous and partly purulent. The roots of the spinal nerves become thickly coated, and they are swollen and softened. The cord participates more or less in the inflammation. In the *chronic* form the membranes become thickened and adherent; and the cord sclerotic, with atrophy of the nerve elements. If the posterior roots be affected, degeneration of the posterior columns may follow (*Rosenthal*).

The **causes** are:—exposure to cold and damp; injuries and diseases of the vertebræ; bad hygiene in scrofulous subjects; and extension of disease from the brain, or from neighbouring tissues. Spinal meningitis affects males more than females; and it is a

* Pathologically, sometimes, divided into (*a*) meningitis, external and internal, (*b*) Leptomeningitis, (*c*) hypertrophic meningitis.

disease, mainly, of youth. Alcohol and syphilis are exciting causes.

The **symptoms** begin with a rise of temperature, headache and nausea, with general *malaise*. The most characteristic symptom is the intense pain in the back shooting up the neck, and into the loins and lower limbs. The body is kept *rigid*, as the slightest movement aggravates the pain. The lower limbs are in a state of spasmodic contraction, and there is often retention of urine. These muscular contractions are increased by attempts at movement; but not by irritating the skin. This symptom helps to differentiate meningitis from tetanus (Jaccoud). There is hyperæsthesia and hyperalgesia in the parts of the skin supplied by the nerves which pass into the affected spinal area. "Girdle sensation" is often present. Later, partial paralysis supervenes, and anæsthesia of the parts which were at first hyperæsthetic. Reflex movements are not abolished. The electro-contractility may be weakened, or it may be normal. The urine is acid throughout. If the meningitis affect the cervical regions, there is dyspnœa, dysphagia, and slowing of the pulse, with weak action of the heart—the respiratory and cardiac functions being disturbed. Death may result within a few hours or days from asphyxia. If such a case affect the medulla there will also be delirium, vomiting, and ocular derangements, &c. In other cases there is extension of the paralysis, and death from exhaustion in two to four weeks. Some cases recover, and many pass into the chronic form.

In *chronic* spinal meningitis the symptoms are somewhat similar, but the development is slow. The pain is not so acute, and the sensory derangements, ridigity, and spasms, occur in slighter forms and at irregular intervals. Ultimately, there is weakness and fatigue of the muscles; numbness, tingling, and anæsthesia, become more constant; and lastly there is paraplegia. Partial recovery is possible, but, most frequently, permanent lameness results.

The **prognosis** is almost always unfavourable.

The **differential diagnosis** of myelitis and spinal meningitis may be tabulated thus (Bramwell):—

Myelitis.	Meningitis.
Pain in the back not prominent.	Pain in the back, increased by movement, is generally very marked.
Shooting pains and hyperæsthesia are seldom prominent. Anæsthesia quickly appears, and is generally well marked.	Shooting pains in the limbs or trunk, and hyperæsthesia are generally prominent features. Anæsthesia occurs later, and is relatively slight.
Paralysis appears early, and is much more prominent than cramps and spasms.	Muscular cramps, spasms, and rigidity of the limbs, and stiffness of the back are more marked than motor paralysis, which is late in appearing.
The sphincters are often paralysed; the urine is often ammoniacal.	The sphincters are not paralysed, the urine is not ammoniacal.
Trophic disturbances of the skin are common.	Trophic disturbances of the skin are rare.
Fever is sometimes considerable, but may be absent.	Fever generally well marked.

The **treatment** of spinal meningitis. In acute cases the local treatment is the same as in acute myelitis. Hypodermic injections of morphia may be necessary for the pain. In the less severe cases, opium may be prescribed, or opium, aconite, and ergot, may be used, as recommended by Bartholow (two minims of the tincture of aconite, five to ten minims of the tincture of opium, with fifteen to thirty minims of the fluid extract of ergot—every two hours during the early stages). Quinine is also useful, in large doses; galvanism and massage are necessary for the paralysed muscles during the stage of convalescence. Iodide of potassium is useful in the late stages; and it is also indicated in the chronic forms of meningitis.

Poliomyelitis Anterior Acuta—Infantile Paralysis. Acute Atrophic Paralysis. (1) *In infants;* (2) *In adults.* This disease has long been known as *infantile paralysis*; but as it is found to occur (very rarely) in adults, the first term is more appropriate, and is the one most generally adopted. [Polio-myelitis from πολίος, *grey*, and μυελος, *marrow.*]

The **pathological condition** found is that of an acute exudative inflammation. An inch or more in length of this part of the cord may be affected. The *post-mortem* examination reveals no naked eye appearances which can be deemed characteristic, unless the disease have existed for many years. In such cases a shrinking of the anterior part of the cord, on one side, or both, may be obvious when a section is made. Microscopically, delicate connective tissue, corpora amylacea, and a few shrunken cells are found in the affected areas. In the more recent cases, there is *inflammatory softening*, and the multipolar cells have almost entirely disappeared, while free nuclei and granules are present in large numbers. The nerve fibres, in the affected parts, are entirely destroyed.

The **causes** are not well known. It occurs most commonly in children between one and four years of age; but cases are recorded earlier, and later. Teething and chills are sometimes blamed. It often arises during convalescence from fevers, or severe illness; and this is the case, also, in the form occurring in adults.

The **symptoms**, in the child, begin with fever and *malaise*, as a rule, although spasmodic twitchings or convulsions may be the earliest indications. The fever may only last a day or two, and the convulsions may be repeated during the same time. The child is

then apparently well, and the attack attributed to teething or other infantile disorder. It is noticed shortly afterwards that the child does not use a limb, or that perhaps even more than one limb is powerless. The limb affected is relaxed and the reflex movements are lost. The faradic re-action is diminished very soon, and it may be absent in a fortnight; and the galvanic current soon yields the "re-action of degeneration." The limbs are often attacked in succession; but the paralysis is always *complete* within a few hours, and there is no progressive advance, nor tendency to bladder complications or formation of bed-sores. The sensory functions are normal, and the paralysed limbs are pale and cold. Recovery may be complete; but this is rare. Partial recovery is the rule, and for some weeks the case looks hopeful. Eventually, however, the improvement ceases, and the affected arm or leg—for in the majority of cases only one limb is paralysed—remains shortened, and undeveloped either in bone or muscle. Contractions and deformities often result, and "club-foot" is a common sequel to infantile paralysis.

In *adults*, the history of a case of poliomyelitis anterior acuta, is very similar, only it is never ushered in by convulsions, and the limbs are not shortened, although a certain amount of contraction and deformity may follow. The nuclei of the cranial nerves may be involved: "polio-encephalitis superior" when eye-muscle nuclei, and "polio-encephalitis inferior" when the cranial nerve nuclei lower down are affected. In the diagnosis *peripheral paralysis from pressure* need only be mentioned. The prognosis must always be guarded.

The treatment consists of rest, with the ordinary general and local treatment of an acute inflammation in the early stages: later the use of electricity—both faradic and galvanic. Great improvement sometimes follows the *long-continued* use of galvanism. A generous diet, with cod-liver oil and iron tonics, is indicated; and the deformities may be, in a measure, prevented by the use of bandages and splints.

Poliomyelitis Anterior Subacuta vel Chronica.—Subacute or chronic inflammation of the anterior horns of the grey matter in the spinal cord produces a thickening of the connective tissue, destruction of the multipolar cells, and degenerative atrophy of the cell elements, as in the acute disease. The causes are unknown. It occurs most frequently between the ages of thirty and fifty. Exposure, lead-poisoning and syphilis are possible causes.

The symptoms begin with weakness in the lower limbs—both being generally affected. Actual paralysis develops within a few days or weeks. The reflexes are absent. The muscles are relaxed and they waste very rapidly. The electrical reaction is abnormal—the anoidal closing contraction being more marked than the cathodal closing contraction, even at the beginning. The faradic re-action is lost. There is little or no anæsthesia, and no bladder complications, nor tendency to the formation of bed sores. The disease may

extend to the arms (and in some exceptional cases it begins with the arms), and a fatal termination may occasionally result from the still further extension of the disease upwards. There is then interference with the respiration and deglutition. It is far more frequent that the case ends in complete, or almost complete recovery. The improvement does not commence, however, for about six weeks, and it may be months before the patient regains the perfect use of his limbs. The gradual return of the normal galvanic re-action serves to estimate the rate of recovery.

The prognosis should be guarded; but it is hopeful, and recovery is the rule.

In the diagnosis, *lead paralysis* and *multiple neuritis* sometimes resemble it, and should be noted.

The treatment consists of galvanism. Iodide of potassium is useful sometimes.

Progressive Muscular Atrophy.—The chief pathological change consists of atrophy of the multipolar cells in the anterior horns of grey matter in the spinal cord. The neuroglia becomes thickened, and the nerve elements are destroyed. The muscles, supplied by the nerves from the affected area within the cord, undergo rapid atrophy, and the intestines between the atrophied muscles become filled with adipose and connective tissue. The muscles lose their normal red colour and become much paler. In extreme cases they are little more than "bands of white fibrous tissue."

The causes are acute infectious diseases, lead poisoning, and overuse of the muscles with extreme fatigue. It occurs more frequently in males; and the average age is between thirty and forty years.

The characteristic symptom is the weakness in certain muscles, with generally obvious muscular atrophy. Certain groups of muscles are affected, especially the muscles of the ball of the thumb; and the atrophy gradually extends to the other muscles of the hand and arm. Soon afterwards the disease appears in the other hand, but it does not always progress in a symmetrical manner. The lower limbs are the last, as a rule, to be affected. There is great weakness in the affected muscles, increased by fatigue and cold, and, in the case of the hand, the prehensile power is much diminished. The hand may present the "griffin" or "bird-claw" like deformity. The loss of muscle is sometimes obvious, but not always so, as the development of fat may conceal it. The testing the muscles with the faradic current will prove the absence or presence of muscular tissue; or a small piece of the muscle may be removed with a "harpoon" for microscopical examination. When the lumbar or abdominal muscles are involved there is much hollowing of the back when the patient attempts to stand. Transient "fibrillary twitchings" of the muscles are generally present in progressive muscular atrophy. The sensory functions are normal, and there is no pain or numbness. The rate of progression varies from two years to ten or even twenty. The disease may remain stationary for two or three years. The cases which progress rapidly are more dangerous, as the

atrophy frequently extends to the trunk and destroys life by interfering with the respiratory movements. Bronchitis, pneumonia, and phthisis are common intercurrent diseases, which are very fatal when the intercostal muscles or diaphragm are involved. Some cases end in "bulbar paralysis." Syringomyelia—a hollow space occupying the centre of the cord—is a pathological condition which sometimes gives rise to symptoms identical with progressive muscular atrophy. Extension of the sclerosis to the anterior horns of grey matter in locomotor ataxia, or multiple sclerosis, &c., produces muscular wasting, in addition to the already existing symptoms proper to these diseases.

Charcot has described two additional forms of progressive muscular atrophy. The one he calls *sclérose latérale amyotrophique* and the other *pachyméningite cervicale hypertrophique*. The former consists of a sclerosis of the lateral columns which spreads to the anterior horns of grey matter chiefly in the cervical region. There is combined paralysis and rigidity of the lower limbs, with atrophy of the muscles of the upper limbs. Byrom Bramwell agrees with Charcot in describing amyotrophic lateral sclerosis as a distinct affection; and as it appears to be more rapid in its progress, and invariably fatal by extension to the medulla (bulbar paralysis), it is important to discriminate this form from the simple progressive, and from the second form mentioned below. The *treatment* is unsatisfactory.

The second form described by Charcot consists of a chronic thickening of the dura mater, which adheres firmly to the cord, and presses upon the nerve roots coming off in the cervical region. There is progressive wasting of the muscles of the upper limbs, and rigidity of the lower limbs. The ulnar and median nerves are especially involved, and the wrist is *extended* instead of flexed.

In this form there is sensory disturbance; and anæsthesia, numbness, and tingling, with pain and rigidity of the neck, generally precede, and continue along with the atrophy. Sometimes "bullous eruptions" arise upon the arms. Charcot considers this form to be sometimes curable; but in the later stages, there is often paralysis of the bladder and rectum, and the formation of bed-sores. This form does not extend to the medulla.

A progressive hereditary muscular atrophy of the leg type (*Charcot-Marie*) is described.

In the **diagnosis** of progressive muscular atrophy—besides the differentiation of Charcot's two forms, and the exclusion of the indiscriminate (transverse) or secondary lesions—the *peripheral paralyses*, *lead paralysis*, and *chronic rheumatism* have to be noted. A knowledge of the anatomical distribution of the nerves will serve to distinguish the first, and the use of electricity will confirm the diagnosis. In peripheral paralysis the reactions are markedly abnormal. The same tests, *plus* the symptoms of lead-poisoning will exclude lead paralysis (neuritis).

The *treatment* of progressive muscular atrophy is often unsatisfactory. Faradic and galvanic electricity are both recommended; and in some cases, favourable results have been recorded.

Pseudo-hypertrophic Paralysis.—In this disease the muscles become atrophied, and their substance is replaced by large quantities of adipose and fibrous tissue—sometimes only the latter. The abnormal muscles are red-yellow in appearance, and the fibres may still be recognised. It is a primary degeneration of the muscles (dystrophy). *Hereditary*, *infantile*, and *juvenile* types are described.

The **causes** are obscure. Heredity seems to play a part in its production, as cases are reported in which several members of a family were affected. It is a disease of childhood, and it affects males more than females.

The **symptoms** begin with motor weakness. After a few months, or even a year, of gradually increasing paralysis, the change in the size of the muscles becomes apparent. The calves generally suffer first, and then the *erector spinæ* in the loins, and the *glutei* muscles. Sometimes the whole of the trunk, and all parts of the limbs are affected. The muscles seem to be enormously increased in size, and the child has an excessively over-developed appearance. When standing he widens the legs, and projects the abdomen, and the hollow of the back is increased. He frequently falls, and he cannot rise without using his hands, and with apparent effort he "climbs up his knees." The electro-contractility is diminished. There are no tremors, as a rule, in the muscles, and the sensory functions are normal. The mental condition is sometimes defective. The bladder and rectum are not affected. Sometimes the knees are kept flexed, and the *tendo-Achillis* may be contracted (club-foot). The disease sometimes does not advance for a few years; but at last the arms become involved, and complete paralysis is the ultimate result. Death may take place from exhaustion; or from phthisis, which is a common intercurrent disease.

The **prognosis** is always unfavourable.

The **treatment** is not satisfactory. Faradic electricity may be tried; and mechanical appliances may be used to lend support to the weak limbs.

Glosso-labio-laryngeal Paralysis—Progressive Bulbar Paralysis.—In this disease the medulla does not appear much altered, to the naked eye. Sometimes it appears unsymmetrical, or shrunken. There is extreme atrophy of the nerve roots, especially those of the hypoglossal and facial nerves; but sometimes the eighth, sixth, and part of the fifth (motor) are affected. In specially prepared sections, the multipolar cells of the nuclei are found to have undergone degenerative changes, and the nuclei present the appearance of a myelitis in the late stages (sclerosis).

The **causes** are unknown. It is a disease of advanced life, as a rule; and it may be the result of extension of spinal disease upwards, as in progressive muscular atrophy, with which it is occasionally associated.

The **symptoms** begin very insidiously. Headache and giddiness are complained of, and the patient notices that he is apt to "choke" upon his food. Difficulty in utterance soon follows, and the voice acquires a nasal tone from the paralysis of the palate. The *labial* consonants present the greatest difficulty to him. The tongue gradually wastes, and the saliva dribbles from the mouth, which trembles visibly whenever speech is attempted. The mental faculties are, however, quite clear. The voice becomes husky, and it is ultimately lost when the disease extends to the nucleus of the vagus; and there is also, then, occasional suffocative attacks, dyspnœa, irregular action of the heart, and great oppression. The sensibility is not impaired, except in the palate. Taste may be impaired, or absent. The muscles affected present the "re-action of degeneration," and, if far advanced, electro-contractility may be lost. The disease may run a course of from one to five years. Death may result from sudden syncope, starvation, or intercurrent disease, especially pneumonia. The symptoms of bulbar paralysis sometimes resemble those associated with effusions of blood into the pons, acute inflammation, or a tumour. The two former diseases occur suddenly. The latter develops slowly, but it sets up irritative symptoms from the first, while, in bulbar paralysis, the onset is slow, gradual, and without irritation. Tumours also give rise to other symptoms, as swelling of the eyelids, &c.; and to ophthalmoscopic appearances (choked disc, &c.). General paralysis of the insane is easy to exclude. In the early stage of bulbar paralysis, the trembling of the lips resembles that which occurs in general paralysis, but the mind is not affected in the former disease.

The **treatment** is unsatisfactory. Faradic and galvanic electricity may be tried. Nitrate of silver is sometimes prescribed. Feeding by means of the stomach tube, or by nutrient enemata, may become necessary.

An *acute form of bulbar paralysis* is described, which is generally fatal within a few days. It is caused by inflammation of the medulla, and changes take place similar to myelitis, within, and limited to the bulb. Cases have recovered. The treatment is the same as in myelitis.

Multiple or Disseminated Sclerosis.—This is a disease which affects the brain as well as the spinal cord. It consists of the formation of irregular or rounded patches of sclerosed tissue, which feel hard, and are of a yellow-grey colour. Microscopically, the patches present an appearance similar to those changes found in chronic myelitis. In the brain they are found scattered through the white matter of the hemispheres, while in the cord any part may be affected. The chief cause known is *infection—e.g.*, enteric and eruptive fevers, malaria, diphtheria, &c. It occurs with most frequency between the ages of twenty and thirty years.

The **symptoms** are very variable, and depend upon the seat of the sclerosis. The characteristic symptoms of a case in which it is general are *tremors; slow, accented, and "scanning" speech;* and *nystagmus*. The tremors are only present when movements are attempted, and they are especially marked when the patient is asked to lift a glass of water to his lips. The hand trembles violently, and the water is generally spilled. The nystagmus is only marked when the patient is directed to look at different objects in rotation. It is not ceaseless, and it may not be observed when the eyes are at rest. The mental faculties are not bright, and subjects suffering from this disease are liable to attacks of *stupor*—sometimes followed by transitory hemiplegia. The patient is inclined to be emotional, and this aggravates the trembling movements. Constipation is the rule, and headaches and vertigo are almost invariably complained of. The other symptoms depend upon the seat of the sclerosis in the spinal cord. Subjective sensations (numbness, tingling, &c.) are often present. Anæsthesia, or impairment of tactile sensation, is not usually present. The reflexes are increased when the lateral columns alone are affected, and the limbs then become rigid and the gait *spastic*. The symptoms peculiar to locomotor ataxia will be present when the posterior columns are sclerosed, but there is no loss of sexual power. The eye symptoms, described as occurring in locomotor ataxia, may be present. The muscles, in exceptional cases, may become atrophied. The *course* is slow and irregular—five to ten years being the usual duration, and death generally results from some intercurrent affection, as pneumonia or phthisis.

The **treatment** is not satisfactory. Electricity may be tried. Strychnine, arsenic, or nitrate of silver may be prescribed.

In the **diagnosis**, *mercurial poisoning* may give rise to tremors not unlike those which occur in multiple sclerosis; but the history will exclude this disease. Locomotor ataxia must be noted (see Diagnosis, page 231). The tremors in *paralysis agitans* can sometimes be diminished or arrested at will—the movement being otherwise *continuous*. *Hysterical* affections may sometimes simulate a case of multiple sclerosis in some of its symptoms. When the tremors and cephalic symptoms are absent, the other diseases of the spinal cord must be differentiated by careful examination of the motor and sensory tracts, &c. In the early stages it sometimes is impossible to make a confident diagnosis.

Paralysis agitans, or shaking palsy, is a nervous disease which consists of oscillatory movements affecting chiefly the limbs, but sometimes also the neck and tongue. It generally begins in the arm, and gradually spreads to the leg, and then to the limbs of the other side. The head often shakes, but there is no continuous movement of the eyeballs (nystagmus). At first it occurs in paroxysms, and the movements may be controlled. The limbs are quiet during sleep, but later the palsy is constant. The movements are much aggravated by emotion or excitement of any kind. The electro-contractility is unimpaired, except that the muscles get quickly exhausted. The speech is *jerky* and slow. There is often pain in the limbs. The head is carried forward, and the gait is hurried as if to prevent a fall. Sometimes the patient when starting to walk finds himself impelled to run backwards. In the later stages there

is rigidity of the muscles, with paralysis. The duration is very chronic, and paralysis agitans is essentially a disease of the old, but sometimes (rarely) it affects the young. Pathological changes have been found in the spinal cord and medulla, of the nature of congestion in the grey matter, increase of interstitial tissue, and atrophy of cells.

The **treatment** is not satisfactory. Prolonged rest of the limbs during the early stages should be tried. Chloride of barium (in one grain doses) is recommended; and arsenic sometimes produces improvement.*

* It may be of use to the reader, and an aid to the memory in the examination of a case of spinal cord disease, to note the order and classification of the foregoing diseases. The "paraplegic" group comprises *myelitis, compression paraplegia, secondary degeneration, the sclerosis* group, *functional paraplegias*, and the group *neurasthenia*; then follow three *special* diseases, viz.:—*Landry's acute ascending paralysis, locomotor ataxia*, and *meningitis*; the group associated with *muscular atrophy*; and lastly, three diseases associated with *trembling* of the muscles.

CHAPTER XI.

DISEASES OF THE NERVOUS SYSTEM.—Section II.

Contents. — Cerebral hæmorrhage; Apoplexy; Occlusion of the cerebral vessels—*i.e., thrombosis, embolism*, and *syphilitic disease*; Hemiplegia; Aphasia—Intra-cranial tumours—Cerebral abscess—Meningitis; acute, chronic, and tubercular; Pachymeningitis—Cerebral congestion and anæmia—Sunstroke—Chronic hydrocephalus—Epilepsy—Hysteria; Catalepsy; and Hypochondriasis—Chorea—Writer's cramp; Athetosis; Singultus or hiccough—Spasmodic tics—Meniere's disease—Migraine—Tetanus—Hydrophobia—**The differential diagnosis of brain diseases.**

Cerebral Hæmorrhage; Apoplexy; Occlusion of the Cerebral Vessels—Thrombosis, Embolism, and Syphilitic Disease. — These diseases may with clinical advantage be considered together, as they form a group in which the symptoms are very similar, and the points of difference may afterwards be stated. In *cerebral hæmorrhage* there is rupture of a blood-vessel in the brain—the most frequent site being the internal capsule, corpus striatum, lenticular nucleus, or optic thalamus. More rarely, it occurs in the cerebellum, pons, or medulla. The symptoms arise from the damage done to these, and the neighbouring parts. The clot may vary in size, from a pea to a large walnut, but in extensive ruptures the fluid blood often diffuses into the ventricles. The clot undergoes changes, and there may be secondary inflammation of the surrounding cerebral tissue. Ultimately a spongy connective tissue is developed, which encloses the clot—in those cases which survive. Small clots may become absorbed, and large clots—instead of becoming enclosed and thus promoting a favourable termination—often light up general inflammation. In chronic cases when the tissues have been much broken up, "atrophic degenerative" changes take place in the descending fibres of the nerve tissue, through the *crus* and *pons* to the spinal cord (*secondary degeneration*).

The commonest *cause* of cerebral hæmorrhage is disease of the cerebral blood-vessels — *atheroma* and *miliary aneurisms; fatty degeneration of vessel-walls*, as in pernicious anæmia, scurvy, purpura and post-infective diseases—and any sudden increase of the blood pressure may produce the rupture. The latter may arise in hypertrophy of the heart and gout; cirrhosis of the kidney; muscular effort; the use of stimulants; hot and cold baths; and violent emotions. The tendency to *atheroma* is often inherited; and it rarely occurs before the age of forty years.

In *thrombosis* and *embolism*, there is occlusion of a blood-vessel by the formation of a clot, in the former disease—while in the latter, a small clot, concretion, or particle of fibrin (embolus) is carried up in the circulation and becomes impacted. Emboli may be derived