eyes are moved to the right; the affected eye cannot be moved inwards, and a *divergent* squint is produced. The *secondary deviation* of the sound eye is towards the right. The double vision, and other symptoms, are the same as in paralysis of the external rectus, only *converse*, as regards the direction.

The (left) *inferior rectus:* the squint is a little upwards when the patient looks downwards, and the rotation of the eyeball is to the left. Diplopia is present, and the false image is below and to the right of the true one, and it has its upper end "tilted" to the left.

The (left) *inferior oblique:* the affected eye squints a little downwards when the patient looks upwards, and the eyeball is rotated to the right. The false image lies above and to the left of the true one, and it has its upper end tilted to the left.

The (left) *superior rectus:* the affected eye squints a little downwards when the patient looks upwards, and the eyeball is rotated to the left. The false image lies above and to the right of the true one, and it has its upper end tilted to the right.

When the *whole* of the third nerve is paralysed, the eyeball on the affected side *cannot be moved inwards or upwards.* It can be lowered slightly if, at the same time, it be carried a little outwards. Giddiness is generally marked. The primary and secondary deviations vary with the position of the object upon which he is directed to look. In addition to these symptoms, the *levator palpebræ* is paralysed, and the upper eyelid droops (ptosis); but this symptom may occur without paralysis of the ocular muscles. There is also paralysis of the sphincter of the pupil, and consequently *dilatation* (*mydriasis*); and paralysis of the ciliary muscles causing *loss of accommodation.*

The **causes** of paralysis of the third, fourth, and sixth nerves are often obscure. When a single nerve, or a branch of the third is affected only, recovery appears to be the rule. Aneurism of the internal carotid is a possible cause; and syphilitic and malignant growths are possibly the commonest causes when more than one nerve, or branch, is involved. Syphilis may sometimes be the cause of ptosis, without there being any evidence of a gummatous formation. Ptosis is common in the aged, and it is probably due to dilated and tortuous arteries compressing the fibres (in part) of the third nerve. Paralysis of the nerves to the eyeball muscles may be due to disease at the base of the brain, and it is frequently an early symptom of locomotor ataxia.

The **treatment** should consist of large doses of iodide of potassium whether the suspected cause be syphilis or not. Galvanism is also good treatment—a current of from six to fourteen cells being used for two or three minutes, the anode being placed on the back of the neck and the cathode upon the closed eyelids.

Facial paralysis, Bell's paralysis. A *peripheral* facial paralysis may be caused by an inflammation of the nerve itself, and the swelling extending to the part which issues from the narrow stylo-mastoid foramen, a

compression results. Prolonged exposure of one side of the face to cold, or even general exposure, may give rise to this condition. At other times inflammation of the parotid or cervical glands, tumours, or wounds of the face, may produce facial paralysis.

Within the aqueductus Fallopii, the nerve may be affected by disease of the petrous portion of the temporal bone. Chronic disease of the tympanum very often leads to caries and necrosis. Facial paralysis has been known to follow a severe blow upon the side of the head. Within the cranium, disease of the base of the skull, tumours, and syphilitic growths, may be the cause; but in this latter group *other* nerves are likely to be involved, and the symptoms of brain disease are superadded. A minute point of softening in the pons has been found to cause facial paralysis; and facial paralysis has been known to precede tubercular meningitis as an early symptom. Some cases may appear to be "post-infective," as after influenza, or some infectious fever. The "central" causes of facial paralysis are considered with cerebral hæmorrhage and hemiplegia.

The **symptoms** are generally very obvious. The mouth is drawn to the sound side when the paralysis is complete, and the cheek of the paralysed side is smooth and devoid of wrinkles. The eye remains wide open and cannot be closed, although during sleep the eyelids approximate far more closely than it is possible for the patient to produce voluntarily. When the attempt is made to forcibly close the eyelids, the cornea is turned up under the lid—this being due to the association of the nerves of the ocular muscles with the act of forcibly closing the eyelids. The conjunctivæ may become inflamed by dust, &c., entering the unprotected eye. The reflex movement of the eyelids is abolished. The cheek of the affected side is not held close to the teeth, and it bulges out with the breathing. Mastication is imperfect, and food accumulates between the cheek and the teeth. Attempts at laughing, smiling, and whistling fail to produce any alteration in the expression of the affected side. The pronunciation of the labial consonants is imperfect. In some cases, the mouth in repose is not drawn to the opposite side, and this is frequently the case in facial paralysis the result of exposure to cold, and in the so-called "rheumatic" type. In the severe forms resulting from caries of the bones, the muscles seem to lose their tone, and the antagonistic muscles of the opposite side produce extreme distortion, and the eye stares very fixedly.

Smell and *taste* are impaired, the former being due to a dry state of the nostril resulting from the tears escaping over the cheek in extreme cases, and the latter due to the implication of the chorda tympani. The sense of *hearing* is often rendered very acute, and this occurs when the stapedius muscle (supplied by a branch of the seventh) is paralysed, and allows its antagonist, the tensor tympani, to keep the tympanum overstretched. The soft palate is often affected, and the uvula is frequently turned to the paralysed side.

A double facial paralysis may be produced by a syphilitic gumma involving both nerves at the base of the brain; or by disease of the petrous portion of the temporal bones on both sides. The face then

is absolutely expressionless, when any of the movements, as smiling, whistling, weeping, &c., are attempted.

In the **diagnosis** of facial paralysis, it is only the very mild forms that may escape notice. A careful examination will generally reveal the symptoms, as for instance, the *incomplete* closure of the affected eye. *Peripheral* facial paralysis is differentiated from *central* paralysis by the fact that in the latter the patient is quite able to close the eye.

During the **course** of a facial paralysis, when the muscles begin to show signs of recovery, the face is frequently distorted by spasmodic contractions, due to the efforts of the patient to use the muscles of the affected side. These generally disappear as the patient gets well; but sometimes a permanent *tonic contraction* is induced, and this is invariably the case when the paralysis is incurable. The cases of facial paralysis which result from cold generally get well within four or six weeks; but sometimes they are prolonged to six months. In the *mild* cases, the muscles and nerves re-act to both galvanic and faradic currents; in the severe cases the "re-action of degeneration" (see p. 206) is present.

The **prognosis** is estimated by the electrical re-action; for should the re-action of degeneration be present, no improvement can be expected for two or three months, and perhaps, for six or eight months—and even then there may be permanent stiffness or weakness of the muscles for years, or for life.

The **treatment** of facial paralysis consists of blistering behind the ear; and the administration of small doses of mercury, and large doses of iodide of potassium. Both kinds of electricity are used, but neither will shorten an attack. Electricity seems to be most useful when the case is beginning to recover. In using *galvanism* the anode should be placed behind the ear, and the cathode moved over the paralysed half of the face.

Paralysis of the ninth or hypoglossal nerve is a rare affection, characterised by an extreme wasting of one-half of the tongue. A cancerous nodule pressing upon the nerve, and disease of the atlas and neighbouring bones, have been known to produce it.

The Brain and Spinal Cord.—In attempting to prepare a semi-diagrammatic scheme which may be useful in understanding the diseases of the brain and spinal cord, I have avoided those details which seem, at present, to be of less practical importance, as they only serve to complicate the diagram. The beautiful drawings of the spinal cord in Dr. Bramwell's work, have served as a model for the spinal portion of the scheme.

The brain is represented as a coronal section, with the posterior third removed, so that the reader looks into the brain from behind. The first, second, and third frontal convolutions (1, 2, 3), with part of the superior temporal lobes (Te), are seen in the section. The corpus callosum (Co-Ca), corpora striata (Cs), optic thalami (O.T), lenticular nuclei (L.T), and internal capsules (I.C) terminating in the crura cerebri (Cr), which are cut, and show the divisions into crusta (C) and tegmentum (T)—are the anatomical points, figured

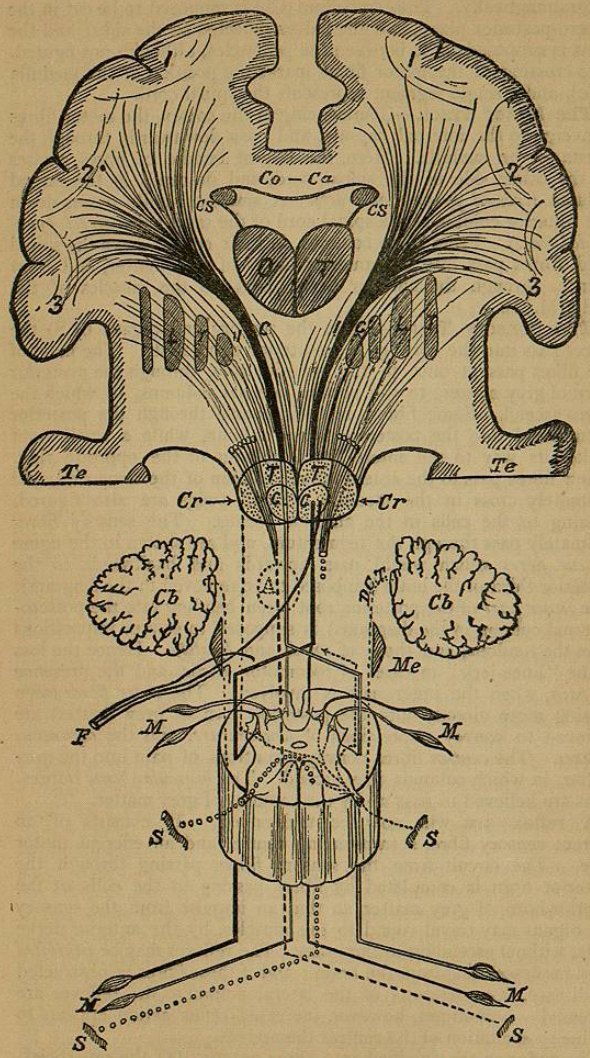

Fig. 33.—The brain and spinal cord with sensory and motor tracts.

diagrammatically. The cerebellum (Cb) is supposed to be cut in the antero-posterior plane, and the halves reflected to the side; and the pons is supposed to lie between the peduncles, although not figured. The crossing of the motor fibres marks the position of the medulla (Me), and a single segment represents the spinal cord.

The Motor Tract.—Commencing at the cortex, the motor fibres converge to the internal capsule, and pass downwards through the crusta to the medulla. Here, the largest bundle of fibres crosses to the other side of the spinal cord, and descends in the lateral column (*crossed pyramidal tract*). A smaller bundle passes down the anterior column of the spinal cord of the same side (*direct pyramidal tract*). From these columns, in their course down the spinal cord, fibres pass into the anterior horns of grey matter, and from thence, through the anterior nerve roots, to the muscles of the body (M.M).

The Sensory Tract.—From the sensory end organs (S.S) the fibres pass into the cord by the posterior nerve roots. The most of the fibres pass at once to the opposite side, through the posterior horn of grey matter, to the postero-internal columns, in which the fibres ascend. Some fibres, in their course through the posterior horn, bend into the postero-external column, while a small tract makes its way to the antero-lateral column of the opposite side. A few fibres pass to the antero-lateral column of the same side, and ultimately cross in the medulla. Small twigs are also figured, passing to the cells in the anterior cornua. The sensory fibres ultimately pass through the tegmentum, and are shown in the figure as *cut fibres* which would pass upwards and backwards to the posterior third of the brain (supposed to be removed in the diagram). The *inner part* of the posterior root (seen bending into the postero-external column in the diagrams) is supposed to convey impressions from the tendons, and those of *touch* and *locality*. Hence the loss of the "knee-jerk" in cases of locomotor ataxia, and the presence of *pain*, when the inner part is involved. The *outer fibres* (seen passing more directly into the posterior horn of grey matter) are believed to convey impressions of *temperature*, and the *cutaneous reflexes*. The *central* fibres convey sensations of pain into the grey matter, in which columns they ascend. The *muscular sense* impressions are believed to pass up by the columns of grey matter.

A **reflex arc** will now be understood. It consists of an afferent sensory fibre, a transferring centre, and an efferent motor fibre. The circuit from the sensory fibres passing through the posterior horn is completed by fibres passing to the cells of the anterior horn of grey matter, so that an impulse from the sensory end organs may travel round to the muscles, by the anterior nerve roots, without ascending to the brain. The reflexes may be *inhibited* by fibres descending from the brain; and if these fibres be obstructed by disease, *e.g.*, sclerosis of the lateral columns, the reflexes are increased. Sometimes, however, increased reflex action appears to be due to excitation of the centres themselves.

The **reflexes** are classified into three groups—(1) the superficial, (2) the deep or tendon reflexes, and (3) the organic reflexes. The *superficial* reflexes may be arranged in an ascending series, beginning with the lowest, viz., the plantar (sacral), gluteal, cremasteric (lumbar), abdominal, epigastric (dorsal), and interscapular reflexes (lowest cervical). They are excited by scratching or tickling the cutaneous surfaces in these regions. The conjunctival and pupillary reflexes are also important. The *cilio-spinal centre* lies in the lower cervical part of the cord, and it is connected with the dilatation of the pupil. The motor fibres pass from this region by the anterior roots into the cervical sympathetic, and they are antagonised by the fibres, to the pupil, of the third cranial nerve. The *deep* reflexes are the "knee jerk" and ankle clonus. According to Gowers, the centre for the former is in the second, third, and fourth lumbar—and for the latter, in the first, second, and third sacral segments. The *organic centres* are numerous, but in the sacral region of the cord those concerned with micturition, defæcation, and the sexual functions are highly important in relation to paraplegia and other diseases of the spinal cord.

Trophic cells exist in the anterior horns of the grey matter of the cord, and any interference with them by disease causes wasting of the muscles, and there is an increased liability to the formation of bed sores.

The facial nerve (F) has been introduced to explain *crossed* paralysis. Three fibres only have been figured—one descending from the cortex with the motor fibres, and crossing in the pons; a second, supposed to spring from the floor of the fourth ventricle; and a third, arising from the lower part of the medulla. These join, and emerge from the side of the medulla, as the seventh or facial nerve. A lesion affecting the fibres in the right internal capsule will cause hemiplegia and a facial paralysis of the left side; a lesion in the left half of the pons (at A) will produce a hemiplegia of the *right* side and a facial paralysis of the *left* side (*crossed paralysis*). The different origins of the seventh nerve fibres may also account for some of them (in central lesions) escaping destruction and producing only partial paralysis. In *Bell's paralysis* the whole nerve is involved, and the paralysis is complete.

The direct (ascending) cerebellar tract (D.C.T.) is of less importance clinically; but it may ultimately be proved to be concerned with co-ordination of the muscular movements. The **commissural** fibres and "association" fibres connect corresponding parts of the brain. They exist also in the spinal cord, connecting the different segments; but they have not been figured.

The various **conducting paths** of the spinal cord are known by the effects of injuries and disease. The trophic or nutritive centres for the *descending* fibres are situated in the cerebrum; and for the *ascending* fibres they are believed to be situated in the spinal ganglia of the posterior nerve roots. When the conducting paths are separated from their trophic centres (by section, experimentally, or by disease), degeneration results, and this serves to map out the columns into tracts.

The accompanying diagram of a transverse section of the spinal cord (from Landois and Stirling's *Physiology*) shows the parts affected:—

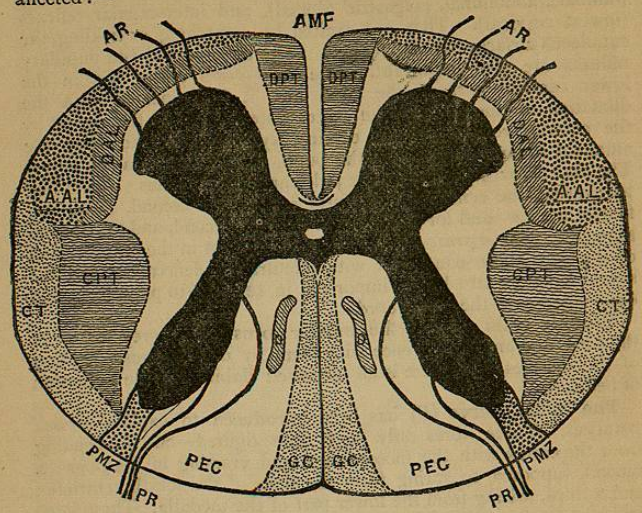

Fig. 34.—Scheme showing the degeneration tracts and the paths that do not undergo degeneration in the cord. A.M.F., anterior median fissure; D.P.T. and C.P.T., direct and crossed pyramidal tracts; A.R. and P.R, anterior and posterior roots; A.A.L. and D.A.L., ascending and descending antero-lateral tracts; C.T., cerebellar tract; D., comma-shaped tract; P.M.Z., posterior marginal zone; P.E.C., postero-external column; G.C, Goll's column (postero-internal column).

Note.—Other two shorter tracts are described, viz.:—The "sulco-marginal tract" (ascending and descending) in the anterior column (D.P.T.); and in the lumbo-sacral region lies Bruce's posterior septo-marginal tract (septal edge of G.C.).

Of these tracts, the important ones, clinically, are the direct and crossed pyramidal tracts; the anterior and posterior horns of grey matter; and the postero-external columns, and postero-internal or columns of Goll.

Paraplegia is a form of paralysis, variable in amount, which affects both sides of the body up to a certain level. The lesion may only attack a single segment of the spinal cord (and secondary descending degeneration of the motor tracts then follows); or the disease may implicate the cord for a considerable part of its whole length. If the disease be situated in the lumbar enlargement, the lower limbs are paralysed. If situated higher, the abdomen and chest are also involved; and if it extend to the cervical regions the upper limbs will be paralysed as well. A transverse lesion of the

cord, beginning at the periphery, may gradually extend across, and as the motor fibres for the more distant parts appear to occupy the *outermost* layers of the lateral tracts, this may account for the feet and legs often being paralysed before the thighs and hips. Complete destruction of a segment is always associated with *anæsthesia*, in all the parts below the lesion; but this does not often occur, as the lesion is seldom so complete and the sensory impulses seem to be more easily transmitted than the motor. *Paræsthesia*, however ("pins and needles," tingling, formication, &c.), is often complained of.

The *gait* of the paraplegic patient varies with the degree of loss of power. When sufficiently developed, the limbs are lifted with difficulty and generally too high, and the foot hangs down and is placed on the ground very clumsily. The legs seem stiff. The paralysis is often complete, and walking is impossible. The inability to stand with the feet close together and the eyes closed, and also the sensation of walking upon soft carpets, are symptoms which point to sclerosis of the posterior columns. In such cases, the disease is not necessarily locomotor ataxia. The lower limit of a lesion is sometimes ascertained by the condition and height of the reflex actions already mentioned. The superficial reflexes may be present when the deep reflexes are absent, or *vice versâ*—in the same patient. When the paraplegia is due to a limited lesion, so that the reflexes are present, the muscles as a rule do not waste. The bladder and rectum are often affected in cases of paraplegia. In exceptional cases the mechanism of micturition is normal, but in most there is either retention or incontinence.

Fig. 35 shows the sensory fibres passing up to the brain, and to the two micturition centres in the cord. Normally, when the bladder is full, it excites the mucous membrane and the impulse travels up these fibres. From the brain, an impulse may travel down (probably in the lateral columns) *inhibiting* the centre for the sphincter muscle which is constantly kept contracted by tonic nerve force, and at the same time *exciting* the detrusor centre to empty the bladder by an impulse to the motor nerve. If it should be inconvenient that micturition should take place, then the impulse travels down the other nerve *exciting* the sphincter to keep up its contraction, while *inhibiting* the detrusor.

In paraplegia, with the sacral regions unimpaired—the lesion being higher in the cord—micturition may be carried on *reflexly*, no stimulus reaching the sensorium, and of course no possibility of controlling the act. When the reflex centres are destroyed (myelitis, &c.) there is complete paralysis of the bladder (and rectum) with the paraplegia. The bladder is capable of retaining a certain amount of urine, but when the pressure increases it escapes, and *incontinence* is the result. In paralysis of the detrusor muscle, there is *retention*, until the pressure of the urine in the bladder is sufficient to overcome the sphincter muscle; or the patient requires to strain with the abdominal muscles in order to empty the bladder. With paralysis of the sphincter, there is *incontinence*. Spasmodic contractions of these muscles, leading to incontinence (detrusor) and retention (sphincter), are common. The former condition is a frequent cause

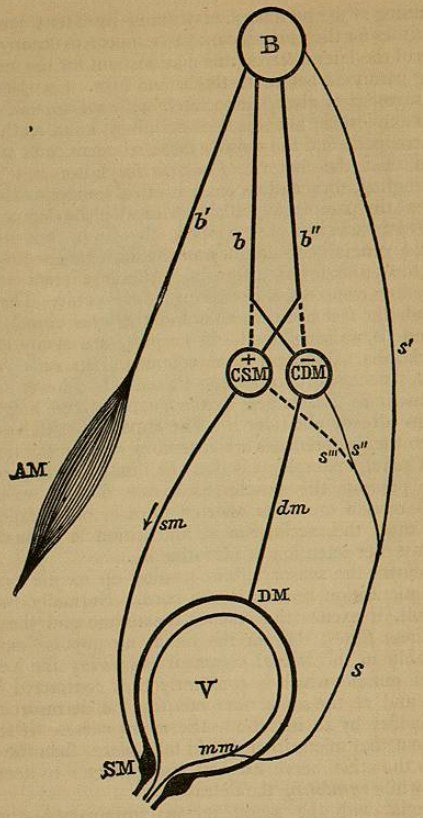

Fig. 35.—Diagrammatic representation of the parts concerned in the mechanism of micturition while at rest. (After *Gowers*, but considerably modified by Bramwell.)—The sphincter muscle (S.M) is in a state of contraction, the result of nerve force continually sent to it from its tonic centre (C.S.M.) in the spinal cord, through *sm*, as indicated by the arrow. V = the bladder, which is represented as empty. S.M, Sphincter muscle. D.M, Detrusor muscle. A.M, Abdominal muscles. *mm*, Mucous membrane of bladder. B, The brain. C.D.M, Spinal centre for the detrusor muscle. C.S.M, Spinal centre for the sphincter muscle. S, S′, S″, S‴, Sensory fibres proceeding from the mucous membrane of bladder up to the spinal cord and brain. *dm*, Motor nerve, from the spinal centre for the detrusor muscle. *sm*, Motor nerve from the spinal centre for the sphincter muscle. *b*, Nerve filament proceeding from the brain to the spinal centres of the detrusor and sphincter muscles. An impulse from the brain through *b* inhibits the sphincter centre (dotted line) and excites the detrusor centre. *b″*, Nerve filament proceeding from the brain to the spinal centres of the sphincter and detrusor muscles. An impulse along *b″* strengthens the sphincter and inhibits the detrusor centre. *b′*, Nerve filament from the brain to the abdominal muscles.

Note.—When the bladder is empty its walls are collapsed. The condition represented in the figure is purely diagrammatic.

of incontinence in children, and it may be due to reflex causes (worms, &c.). Spasm of the sphincter is common in hysteria. When the micturition centres are deprived of their functions by disease or injury, the bladder becomes distended and the urine dribbles away. (As this, sometimes, gives rise to the belief that the patient is passing his urine freely, the abdomen should always be carefully palpated).

In paraplegia the urine is often turbid, alkaline, and ammoniacal. Cystitis and nephritis are common, and the latter may be the ultimate cause of death. Bed-sores are also common. The *sphincter ani* is often paralysed along with the vesical changes; and priapism is often present, in cases of paraplegia.

Hemiparaplegia, and **hemianæsthesia**, are conditions which may be produced by injuries, tumours, or sclerosis, of one half of the cord. The symptoms are characterised by paralysis of the same side, below the lesion; with anæsthesia of the *opposite* side. A sclerosis may sometimes, at the beginning, affect only one half of the spinal segment, and then ultimately extend across.

Myelitis—*Acute and Chronic*—**Softening of the Cord.**—In acute inflammation of the spinal cord the central grey matter is often the chief seat of the disease, but it extends to all parts of the cord; hence varieties are described as *acute*, *general*, *central*, *transverse*, *unilateral*, and *disseminated myelitis* (Bramwell). There is first hyperæmia, with extravasations and serous transudations which give a moist and softened appearance to the parts affected. The colour ultimately changes to yellow-white, the nerve elements becoming fatty. The adjacent meningeal membrane becomes thickened and adherent. Microscopically, the cells are seen to be swollen and the reticulum thickened. There is exudation of colloid material around the minute vessels, the capillaries and veins being much dilated. There is granular disintegration of the nerve fibres and ganglion cells. *Softening* of the cord may follow the acute inflammation; but it is a pathological condition which sometimes arises without any previous myelitis, and is followed by secondary inflammatory reaction. In rare cases an *abscess* may be formed. The *chronic* form of myelitis is characterised by the greater development of the neuroglia with thickening of the vessels, and a deposit of numbers of amylaceous bodies with atrophy of the nerve elements. The cord is *firmer* than the normal in its affected parts.

The **causes of myelitis.**—Myelitis may be produced by injuries, and fractures of the vertebræ; the neighbouring inflammations may extend to the cord—as in spinal meningitis, with which myelitis is almost always, more or less, associated. Cancerous, tubercular,

and syphilitic inflammations may also extend and affect the cord. Syphilis is believed by Erb to be an important and undoubted cause of myelitis. It is, then, generally the *disseminated* form. Myelitis is sometimes a complication of fevers, and of acute rheumatism. It may also be excited by prolonged functional activity of the cord, as in standing for long periods, muscular fatigue, sexual excesses and abuse; or by exposure to cold and damp—especially the lying for a time upon damp ground. The age at which the greatest number of cases occur, is between ten and thirty-five years; but cases are frequently met with later in life. The *chronic* forms, and *softening* of the cord, are due to the same causes. Defective nutrition may be an additional cause of the latter, when it does not follow an acute inflammation. *Softening* is more apt to follow the acute myelitis; while *sclerosis* results from the chronic forms.

The symptoms of a typical case of acute myelitis—when it occurs as a *primary* disease—are ushered in by fever and general *malaise*. Sometimes, however, the spinal symptoms attack the patient at once, and, again, a premonitory stage is sometimes described in which there are derangements of sensation and slight pains in the limbs or back, without much fever. When fully developed there is "girdle sensation," and pain in the back; but the latter is not intense, nor aggravated by movements, unless the associated meningitis be severe, and, then, there is much more soreness developed by percussion of the spine. Pain, or sensations of intense fatigue in the lower limbs, with tingling or formication, sensations of heat or cold, partial anæsthesia, and sometimes hyperæsthesia, are complained of; and also sometimes "bearing down pain" in the bladder and rectum. Priapism is a frequent symptom. Tremors or spasms may precede the paralysis of the lower limbs which soon supervenes and becomes more or less complete, with loss of electro-contractility. The bladder and bowels are often affected by the paralysis, and the urine becomes alkaline and ammoniacal. Cystitis and nephritis may follow. Sensation becomes more or less completely lost up to the middle of the body, where a zone of hyperæsthesia may sometimes be made out.

The reflex activity is, generally, at first increased, but later it is lost. The differences depend upon the site and variety of the lesion within the segment of the cord. The lesion may spread up, or across the cord. When the anterior cornua are affected, there is wasting of the muscles, which present the "reaction of degeneration," and there is a marked tendency to the formation of bed-sores. The arms are paralysed if the lesion be in the cervical cord. If the myelitis ascend unusually high the pupil is affected. It is *dilated*, if the sympathetic centres be irritated; and *contracted*, if they be destroyed. The muscles of respiration may become paralysed if the cervical regions be affected, and severe dyspnœa then precedes a fatal termination. Fever may be present, absent, or irregular, throughout. The pulse is quick and often irregular, especially when the cervical region is the seat of the disease. Acute myelitis very rarely extends to the brain. The *course*, obviously, must vary. Complete recovery is only possible when the disease runs a short course and has not been severe. This is somewhat rare, and more frequently the acute passes into the chronic form, with secondary degenerative changes. *Central* myelitis affects the grey matter and includes the anterior horn. This form runs a rapid course, and death may result from asphyxia—the muscles of respiration being more apt to be affected by the rapidly ascending lesion. Rapid wasting of the muscles, bed-sores, and early loss of reflex action, characterise the central form of myelitis. The *traumatic* form of myelitis is a transverse lesion, usually situated above the dorso-lumbar enlargement. The muscles do not rapidly waste, and the reflexes are exaggerated. In many cases permanent deformities result from contraction and wasting of the paralysed muscles. Death may be due to cystitis and nephritis, or to secondary developments, as pneumonia, bronchitis, &c.

The differential diagnosis of acute myelitis and spinal meningitis is tabulated on p. 232 (spinal meningitis); while that of myelitis from other conditions will be considered further on.

The prognosis in acute myelitis is generally unfavourable. In mild cases, recovery is possible. Syphilitic cases are more hopeful; but the majority, if they live through the acute stage, end in becoming chronic.

The symptoms of chronic myelitis vary with the form. These are the *chronic transverse*, *disseminated*, *peripheral* or *annular*, *focal* (a single patch), and *general* varieties (Bramwell). In most cases the onset is gradual. Disorders of sensation precede the motor disturbances. Numbness, tingling, a feeling of softness under the feet as if walking upon cushions, pain in the back, and "girdle sensation," are some of the first symptoms. A feeling of fatigue or weight in the lower limbs, with slight paresis, constipation, and sometimes a difficulty in emptying the bladder—are also among the early symptoms. As the case progresses there is more or less complete loss of sensibility in the sensory nerves which pass into the affected area of the cord. The impressions of tickling disappear first, and then touch, pressure, temperature, and finally pain (Rosenthal). The sensations in the anæsthetic regions are often strangely altered—as a hot test-tube being supposed to be a cold one, and *vice versâ*. There is often great delay in the transmission of sensations. The sexual functions are lost.

The paresis, or paralysis, almost always extends from below upwards; and its amount, along with the variations in the symptoms, depends upon the seat and extent of the myelitis. Paraplegia is the usual condition, and the paralysed muscles lose their electrical re-actions. The general health is at first good; but ultimately there is complete paralysis, and the development of bed-sores, cystitis, and nephritis—and death may result from these conditions or from intercurrent maladies. There is never true recovery from chronic myelitis, although the morbid processes are often arrested for long periods of time. The progress is very slow.

The treatment consists of absolute rest in the *acute* cases of myelitis. The patient should lie upon the side or face. Sinapisms,

ice, or hot douches to the spine, are useful. Leeches may be highly beneficial in the early stages. Quinine may be prescribed, and iodide of potassium is often indicated. In *chronic* cases, Erb recommends the hydropathic treatment, and galvanism. The treatment should also include a consideration of the cause—when possible to remove or relieve it.

From the short sketch given of the pathology of myelitis, it will be seen to be a disease which may affect any part, or parts, of the cord, and the signs will vary according to the seat of the inflammation. According to Hilton Fagge, it is also the commonest disease of the cord, and several authors believe with him, that many of the so-called functional disorders—as reflex paraplegia—are really due to myelitis. For these, and other reasons, it is convenient to regard myelitis as the *type* of spinal cord disease, and a reference to the diagram of the motor and sensory tracts, &c., will assist one to understand the physical signs produced by myelitis, and all other obstructive lesions of the cord. *Paraplegia* (or paresis) is the prominent symptom of this group, and although it occurs as a symptom in other diseases not included here, the description already given applies to all of them. The further advantage of a "paraplegic" (or myelitic) group is the fact that in obscure cases—and there are many—it is difficult or impossible to diagnose the etiological factor. In most cases of spinal cord disease, the diagnosis is made by inference, or by the process of exclusion, and it is often so doubtful that the mind is left with a certain number of possibilities. The diseases excluded from this group are best considered as "special;" or another prominent symptom serves—as *rapid wasting of the muscles*—to classify them more usefully. The adoption of myelitis as the type of this group, is also convenient, as the other members of the group may have their symptoms contrasted, and the differential diagnosis made at the same time. The diseases included in the "paraplegic" group are *compression paraplegia, secondary degenerations, spastic spinal paralysis, and alcoholic, syphilitic, hysterical, and reflex paraplegia.* The neurasthenic group—*spinal weakness. irritation, anæmia, and congestion*—is closely allied to the paraplegic group, clinically; and it may conveniently be considered as part of it.

Compression-Paraplegia.—The most frequent cause is *caries of the spine* (Pott's disease), often producing "angular curvature." It may be simple or scrofulous. *Malignant disease* of the spine is the next in frequency. It often is secondary to malignant disease of other organs. Erosion of the vertebræ by *aneurisms*, and very rarely by *hydatids; hæmorrhages* in and around the cord; *meningeal tumours*, or *gummata*—are also causes of compression-paraplegia. The pathological condition of the cord is that of a transverse myelitis. In old cases there is secondary degeneration of the descending lateral columns.

The *symptoms* frequently begin with indefinite pains, due to irritation of the nerves coming off from the cord about the level of the

lesion. These pains are referred to the parts which the nerves supply, and hence pleurodynia, intercostal or brachial neuralgia, colic, or sciatica. There is usually anæsthesia of these parts, or impaired sensibility, and often hyperæsthesia. Sometimes the muscles are thrown into clonic, or even tonic spasms. These symptoms are more common in malignant disease, and the pain is described as shooting and burning in character. The early symptoms sometimes precede the paraplegia for months, and even longer. The paralysis may occur rapidly, but oftener it develops slowly. It has no tendency to extend up the cord. The reflex contractions are exaggerated. The bladder is often unaffected—except in the late stages when the paraplegia is complete. The sensory impressions are often much slowed in their transmission to the brain. A painful sensation (dysæsthesia) is often complained of, when the leg is only slightly pinched; and it is sometimes referred to the opposite limb. In the *diagnosis* of the different causes of compression-paraplegia, Pott's disease may be observed; but caries of the cervical or lumbar regions may not present any external deformity. The patient should be made to stoop, that the *flexibility* of the spine may be noticed. Hæmorrhages are characterised by *sudden* pain and onset of the symptoms. Malignant disease is more common in the lower, and caries in the upper half of the spinal column. Pain in the back points to a growth; but *rachialgia* and *hysteria* have to be remembered. Meningeal tumours and gummata are often difficult, and to diagnose them from caries, without deformity, and from a simple primary transverse myelitis, must sometimes be impossible. Meningeal tumours are said to be slower in growth, and a case may extend to five years or longer. The average duration of cases of compression-paraplegia is about a year.

The *treatment* consists of mercury and iodide of potassium in suitable cases. The actual cautery, extension, and plaster of Paris jackets in the cases of caries of the spine—are surgical considerations.

Fig. 36. — Transverse spinal cord, secondary *descending* degeneration.

Secondary degenerations of the cord are grey degenerations found in certain strands, and which arise from loss of function by disease or injury parting the fibres from their trophic centres. The ascending fibres in the postero-internal and antero-lateral columns may be affected, but these degenerations give rise to no symptoms; but degeneration of the descending fibres in the anterior and lateral columns causes motor weakness and rigidity in the lower limbs, and ultimately contractions and deformity. The rigidity occurs late, and after the paraplegia has existed for some time. *Early* rigidity can only be explained by supposing the spinal (or in the case of cerebral disease, the brain) centres to be in a state of irritation. The knee jerk and reflexes are exaggerated, the reflex arc not being obstructed.