its are determined. Thus in fœtal cords of various ages different tracts can be distinguished by the fact that they develop at different times. And in diseased cords pathological processes are often strictly confined to certain tracts. This is especially true of the processes known as secondary degenerations, by means of which the exact boundaries, the length, and the function of the various tracts have been ascertained. The older division of the columns of the cord into anterior, lateral, and posterior must be set aside in favor of the late divisions founded on these facts.

In a cross-section of the cord at the cervical region the following tracts are seen in each half of the segment:

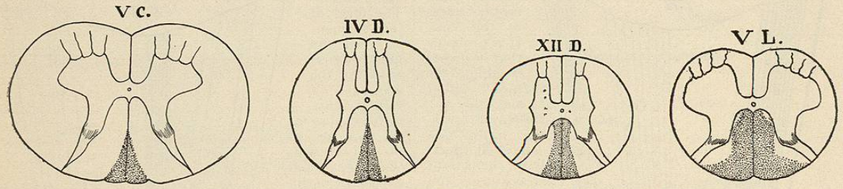
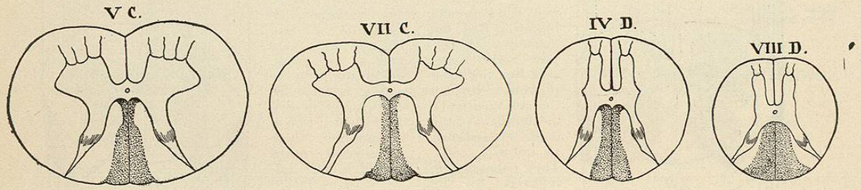


FIG. 4416.—Area of Ascending Degeneration in the Posterior Columns after Compression of Cauda Equina, Involving the Sciatic Nerve Roots only.

1. *The Motor Tracts* (Fig. 4412, *Py, AM*), two in number, which come through the anterior pyramids of the medulla, from the motor region of the cerebral cortex on either side of the fissure of Rolando. It will be remembered that the pyramids of the medulla decussate partially just at the upper limit of the spinal cord.[3] The majority of the fibres of each pyramid cross the median line to the lateral column of the spinal cord. The remainder pass directly onward into the anterior column. Those that cross over are called the crossed pyramidal tract. Those that do not cross are called the direct or anterior pyramidal tract, or column of Türck. The latter lies along the side of the anterior fissure of the cord, and is called the anterior median column (Fig. 4412, *AM*). The former lies in a triangular space in the posterior part of the lateral column, bounded by other tracts on all sides (Fig. 4412, *Py*). These motor tracts differ somewhat in size in different cords. When only a few fibres from the anterior motor cells of the cord (Fig. 4412, *m*), a single nerve fibre in the motor tract carrying an impulse which reaches several groups of cells. They both degenerate downward after any lesion which cuts them off from their nutrient cells in the cerebral cortex. If that lesion is in the brain on one side, the anterior median column on that side, and the crossed pyramidal tract on the opposite side, will be degenerated in the cord. If the lesion is in the cord on one side, both motor tracts on that side will degenerate downward. If the lesion divides the entire cord, the degeneration will be bilateral in both columns. These motor tracts transmit not only voluntary impulses, but also inhibitory impulses, which hold in check the reflex activity of the spinal centres. Hence a lesion in their course produces not only paralysis, but also a loss of control over the bladder and rectum, and an increase in the spinal reflex activity.

2. *The Association Tracts* (Fig. 4415).—Each spinal segment has been shown to have functions of its own. But the different segments always act in harmony, and in hardly any act, either motor or sensory, is any segment independent of the rest. Hence a large part of the white matter of the cord contains fibres, shorter or longer, joining the various segments with one another, and associating their actions. The cell bodies which give origin to these fibres lie in the various parts of the gray matter, and send fibres both up and down in all the columns. These fibres give off collaterals in their course. The fibres lie about the anterior horns of the cord, on their different sides, making up together a large antero-lateral column which has been divided into an anterior column (Fig. 4415, *AL*), and a general lateral column, the latter having a portion between the crossed pyramidal tract and the outer surface of the gray matter which has been called the lateral limiting layer (Fig. 4415, *L*). Some association fibres also pass in the posterior columns adjacent to the posterior commissure. All these tracts are about the same size at all levels of the cord, thus differing from the motor tracts, which decrease in size from above downward, and from the sensory tracts, which increase in size from below upward. They degenerate but a short distance in any transverse lesion of the cord. They degenerate both upward and downward. It should not be forgotten that the anterior nerve roots pass out of the cord through the anterior column, and that many of these roots pass upward or downward for some distance before making their exit. Hence the antero-lateral column is not wholly made up of association tracts. There is no form of disease limited to the association tracts exclusively, hence it is impossible to bring any known symptoms into connection with the lesion when they are affected in a general myelitis.



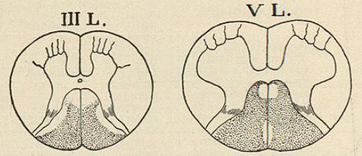


FIG. 4417.—Area of Ascending Degeneration in the Posterior Columns after Compression of Cauda Equina, Involving all the Sacral and Lumbar Nerves.

3. *The Sensory Tracts.*—These occupy the posterior columns of the cord, of which there are two on each side of the posterior median septum, viz., the postero-external column, or column of Burdach, and the postero-median column, or column of Goll. They also pass in the lateral columns of the cord, in the antero-lateral ascending tract or column of Gowers, and in the direct cerebellar column.

The column of Burdach is made up very largely of the posterior nerve roots which enter it and pass upward or downward for some distance before leaving it, to end in the posterior gray horn, or to enter the column of Goll (Fig. 4415, *B*). The column of Goll is made up wholly of long fibres extending from the posterior nerve roots to the medulla (Fig. 4415, *G*). The exact areas taken up respectively by the short posterior roots and the long fibres differ greatly at different levels, and they have only recently been determined by a study of the tracts degenerated after transverse lesions at different levels. These recent investigations deserve a moment's notice.

If the posterior nerve roots are divided between the posterior spinal ganglia and their entrance into the cord, an ascending degeneration occurs in the cord. It is by observing the course of this ascending degeneration that the upward continuation of the sensory nerves has been determined.[4] The area of the posterior columns of the cord which degenerates upward differs in different cases. When the sciatic nerve roots alone are divided, or the sacral portion of the spinal cord is destroyed, the ascending degeneration occupies a large area of the posterior columns in the lumbar region, a smaller area in the dorsal region lying wholly in the column of Goll, and the posterior median portion only of the column of Goll in the upper dorsal and cervical regions (see Fig. 4416). When all the nerve roots of the cauda equina, including both sacral and lumbar nerves, are compressed and destroyed, the ascending degeneration occupies a somewhat larger area than in the first case, involving both posterior columns as high as the middle of the dorsal region and a large part of the column of Goll in the cervical region (see Fig. 4417). When a transverse lesion of the cord in the dorsal region cuts off all sensory conduction from below the level of the mid-dorsal region, the area of ascending degeneration is still larger than in the first two cases, and in the cervical region involves the entire column of Goll (see Fig. 4418). When the cord is divided in the lower part of the cervical enlargement, the ascending degeneration involves a very large area, including both the entire column of Goll and a part of the column of Burdach in the upper cervical region (see Fig. 4419).

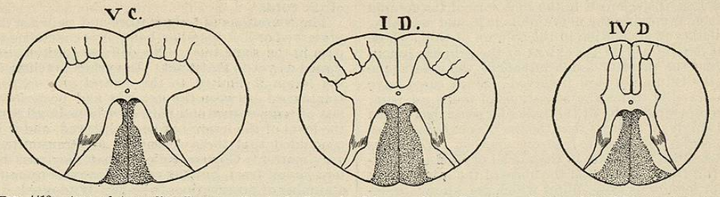


FIG. 4418.—Area of Ascending Degeneration in the Posterior Columns after a Transverse Lesion of the Mid-dorsal Region.

From these facts it becomes evident that the posterior nerve roots contain a number of fibres which, after entering the cord, turn upward and pass on to the medulla oblongata, each successive set from below upward lying a little in front of, and outside of, the preceding set, and gradually filling out the entire column of Goll and a portion of the column of Burdach. This is shown in Fig. 4415, *G*, *B*, at level *C VI*. In a cross-section in the upper cervical region it can, therefore, be affirmed that the fibres in the posterior median part of the column of Goll transmit sensory impulses from the legs; that the fibres in the median and lateral portion of the column of Goll transmit sensations from the thighs and pelvis; that the fibres in the anterior portion of the column of Goll transmit sensations from the body exclusive of the arms; and that the median part of the column of Burdach transmits sensations from the arms. Experimentation on animals has proven that the nerve fibres entering the cord in the posterior nerve roots, and passing upward in this manner to the medulla, degenerate upward upon the side on which they enter. There is no reason, therefore, to believe that in man there is any decussation, in the spinal cord, of the fibres thus far described. But since all sensations, except those of muscular sense, are known to cross over to, and ascend in, the opposite side of the cord in man, immediately after their entrance, it follows that the fibres thus far considered have for their function to transmit the sensations of muscular sense. And this

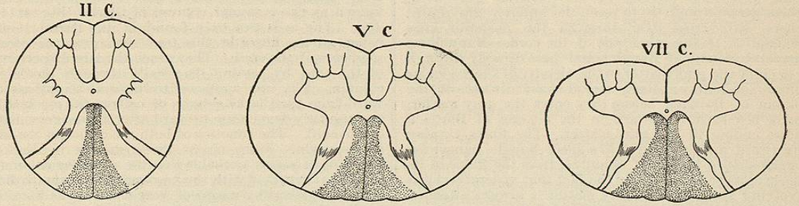


FIG. 4419.—Area of Ascending Degeneration in the Posterior Columns when the Lower Cervical Region of the Cord is Involved by a Transverse Lesion.

conclusion is further established from the facts gathered from the pathology of locomotor ataxia. For in this disease, in which the muscular sense is the one most seriously impaired, the same areas of degeneration are found. There is, first, the sclerosis in the root zone of the column of Burdach, involving the nerve roots in it, and secondly, the secondary degeneration in the column of Goll, whose extent is determined by the extent of the primary lesion. The higher the primary lesion advances, the greater the area of the column of Goll involved. Since cases have been observed in which the sensations of touch, of pain, and of temperature, and of the muscular sense, have been affected singly, it follows that the tracts conveying these sensations must be separate from one another. The fibres so far described terminate in the nuclei of Goll and Burdach. But from these nuclei the fibres of the interolivary tract and lemniscus arise, fibres which are known to decussate in the sensory decussation of the medulla, and are known to transmit sensations of muscular sense exclusively. Hence the conclusion seems warranted that the sensory tract for muscular sense lies in the column of Goll for all parts below the arms, and in the median part of the column of Burdach for the arms.

With regard to the sensory tracts for touch, pain, and temperature, our knowledge is much more indefinite. These sensations enter the cord by the posterior nerve roots. But these roots do not send all of their fibres upward in the path already described. And if the area of the cord occupied by the column of Goll in the cervical region be compared with the entire area of the posterior nerve roots, it will be seen that a mere fraction of the fibres entering the cord by these roots ascends to the medulla. The large remainder terminate in the cord. Anatomists describe various manners of termination. Some fibres end directly in the posterior gray horn; others pass through it to reach the anterior gray horn; others, still, cross over through the posterior gray decussation to the other side of the cord. Many fibres enter the column of Burdach and pass directly through it to enter the gray matter in the vicinity of the vesicular column of Clarke; others ascend some distance in the column of Burdach before they enter the gray matter, and a few turn downward in the column of Burdach before ending in the gray matter. The fibres turning downward are collected into a small bundle, named the comma-shaped bundle of Schultze, from the shape of its area in cross-section. From the fact that general myelitis involving the posterior gray matter is always attended by sensory symptoms, it is concluded that many sensations are sent to the cells of the posterior horns. From the cells in the posterior gray matter some nerve fibres pass backward into the columns of Goll and Burdach, and mingle with the fibres of those columns, presumably ascending with them to the medulla. It is not improbable that these fibres transmit sensations of touch. Even in the most extreme cases of secondary ascending degeneration in the posterior columns, after division of the nerve roots, many fibres in those columns escape. It is therefore certain that some of the fibres making them up have their origin and nutrient cells in the gray matter of the posterior horns, rather than in the posterior spinal ganglia. Hence the facts do not exclude the possibility of the transmission upward of sensations of touch in the posterior columns of the cord after such sensations have crossed the median line in the gray matter. And that they are transmitted in this region the older physiological experiments established. Other physiological experiments point to a transmission of sensations of touch in the lateral columns of the cord. And Gowers has established the existence of a tract in the periphery of the antero-lateral column, lying anterior to the direct cerebellar tract, which degenerates upward after transverse lesions. This is called the antero-lateral ascending tract. Its fibres arise from cells in the gray matter (Fig. 4414, *e*), cross to the other side, passing through the antero-lateral column, and turn upward in this column. This column ascends through the antero-lateral part of the medulla and pons. Some of its fibres have been traced into the lemniscus and some into the cerebellum. There are other fibres which arise from cells in the posterior gray matter of the cord, and crossing to the opposite side, ascend in the antero-lateral column (Fig. 4414, *X, AL*) of the cord.

The sensations of temperature and pain are uniformly preserved or lost together, hence it is concluded that they pass in the same tract. No definite position can be assigned as yet to that tract. In syringomyelitis, in which the lesion is limited to the central gray matter of the spinal cord between the anterior and posterior horns, a loss of temperature and pain sensations in all parts below the level of the lesion has been observed, and it has been concluded that these sensations are transmitted by the gray matter. Gowers believes that they pass in his antero-lateral tract, but the conclusion rests upon too small a number of observations to be hastily adopted. It seems to be likely that the transmission of sensations through the cord is not merely attained by these long tracts, but that they pass chiefly through a series of the association tracts already described, which are scattered through all the columns. A given sensation sets up many reflex and vaso-motor impulses in the cord in addition to being sent to the brain to awaken a perception. Hence it is likely to pass by a broken rather than a continuous tract. But the exact course of sensory impulses is not yet determined with accuracy.

4. *The Direct Cerebellar Column.*—The last column of the cord to be described is one lying upon the outer surface of the lateral column, and extending from the lower dorsal region to the corpus restiforme of the medulla, and thence to the cerebellum. Its termination in that organ has led to its name—the direct cerebellar tract. It is made up of fibres whose origin is in that column of cells which lies in the median part of the posterior horn known as the vesicular column of Clarke (Fig. 4414, *c, DC*). The cells are only found in the dorsal region, hence all the fibres in this tract come from the dorsal segments of the cord. They reach the lateral periphery of the cord by passing diagonally through the lateral column. They are supposed to transmit sensations upward from the Clarke column of cells to the cerebellum, because they degenerate upward after a transverse lesion of the cord. The function of both the cells and the tract is uncertain. From recent investigations by Gaskell, however, it seems probable that the vesicular column of Clarke is connected with the vaso-motor and sympathetic nervous systems by means of very small nerve fibres extending from the sympathetic ganglia into the cord. If this is so, the function of the direct cerebellar tract is to transmit those rather indefinite sensations from the viscera, or to act as a tract for unconscious sensations or motor impulses necessary in a central regulation of visceral and vascular action. The hypothesis that they convey muscular-sense sensations from the trunk is hardly warranted, since these must be of little importance in lower animals, who do not walk erect—in which animals, however, this column is well developed.

II. Symptoms Leading to the Diagnosis of Local Lesions in the Spinal Cord.—Such being the functions of the various parts of the spinal cord, it remains to discuss the symptoms arising when various parts are diseased. And it will be as well to approach this subject from the side of the symptoms rather than from that of the lesion, since it is the object to determine the lesion in any case.

1. *Spinal Paralysis.*—The motor tract conveying voluntary impulses from the brain to the muscles consists of two elements: first, the cerebro-spinal element, and, secondly, the spino-muscular element. Each element consists of a set of nerve cells and their outgoing fibres, which not only transmit impulses from the cells, but are nourished by them. The cells of the cerebro-spinal element lie in the cerebral cortex. Their fibres make up the motor tract through the brain and through the direct and crossed pyramidal tracts of the spinal cord.[5] These fibres terminate about the motor cells of the anterior horns of the cord at various levels, some of them reaching its very lowest part. Any lesion in the cells of the cortex, or in the course of the fibres, which cuts them off from those cells, results in the degeneration downward of the cerebro-spinal element to its termination in the motor cells of the spinal cord. The *first form of spinal paralysis* is due to a lesion at the spinal part of this cerebro-spinal element of the motor tract. If the cord is divided by a transverse lesion at any point, the function of this element of the motor tract is thereby suspended. As a result, voluntary motion is arrested in the parts below the lesion. If the lesion involves but one-half of the cord, it is the limbs on the side of the lesion which are paralyzed. If it involves the entire cord, both sides are paralyzed. The extent of the paralysis depends upon the level of the lesion; the higher the lesion the more extensive the paralysis. The degree of the paralysis will depend on the character of the lesion, slight compression of the cord at one point by a tumor, or a pachymeningitis, or a projecting vertebra, being followed by some stiffness of movement and rigidity of the muscles, with weakness, rather than by absolute loss of power in the parts below the level of the pressure. The cerebro-spinal element of the motor tract also transmits the inhibitory impulses which continually keep the spinal reflex and automatic mechanisms in check. A lesion of this tract, therefore, produces not only weakness and paralysis, but also increase of the deep reflexes, and impairment of control over the bladder and rectum. The muscular action of the limbs, being no longer controlled by the brain, is governed wholly by the centres in the spinal cord. These act in response to sensory impulses, or spontaneously, without check, and hence the preponderating strength of flexor over extensor muscles tends to produce a position of adduction and flexion of the limbs which are paralyzed, and a heightened muscular tone, with tendency to rigidity. The nutrition of the paralyzed muscles may suffer somewhat from disuse, and from the attendant vaso-motor paresis, but no rapid atrophy is noted when the cerebro-spinal element of the motor tract is alone involved. And it is also to be noted that the paralysis affects the entire limb or limbs, and not any special group of muscles. In these cases the electric contractility remains normal in the paralyzed limbs.

A typical example of this form of spinal paralysis is seen in compression of the spinal cord, below the lesion, and in lateral sclerosis or spastic paraplegia (*q.v.*).

The *second form of spinal paralysis* is due to a lesion in the spinal part of the second element of the motor tract, viz., the spino-muscular element. This consists of the cells of the anterior gray horns of the cord, and the anterior nerve roots which pass out through the anterior columns of the cord. Destruction of the cells suspends both voluntary and reflex motor impulses to the muscles. The cells not only control the motion, but also the nutrition, of the nerves to which they give origin, and of the muscles to which these nerves go. Therefore destruction of the cells produces atrophy of the muscles with which they are connected. If the destruction is gradual, the atrophy is gradual, as in progressive muscular atrophy. If the destruction is rapid, the atrophy is rapid, as in infantile paralysis. The degree of the atrophy depends upon the degree of destruction of the group of cells which govern the particular muscle affected. If the group is wholly destroyed, the muscle becomes totally atrophied. In addition to paralysis with atrophy there is in the second form of spinal paralysis a change in the electric reaction of the paralyzed muscles. They lose their contractility to the faradic current, and alter their contractility to the galvanic current, responding in a sluggish manner, and to the positive more readily than to the negative pole. This is called the reaction of degeneration (*q.v.*).

The extent of the paralysis depends upon the extent of gray matter affected, and a reference to the table of the localization of functions already given (page 340) will enable one to determine the effect of a lesion at any particular segment, or through a group of segments, of the spinal cord. A typical example of the second form of spinal paralysis is found in infantile paralysis or poliomyelitis anterior. The muscles in this disease are paralyzed, atrophied, exhibit the reaction of degeneration, and lose their reflex excitability. An entire limb is rarely affected, certain groups of muscles being usually paralyzed together, *e.g.*, the deltoid, biceps, brachialis anticus, and supinator longus (upper arm group); or the extensors of the wrist and hand muscles (lower arm group); or the glutei and thigh muscles (thigh group); or the anterior tibial and peroneal groups of the leg (leg group). The muscles affected are not those which are supplied by a single peripheral nerve—a fact which enables a diagnosis between a lesion in the spinal cord and a lesion in a peripheral nerve to be easily made—but those which act together to produce a definite physiological act.

The contrast between these two forms of spinal paralysis can be seen at a glance in the following table:

First Type of Spinal Paralysis.	Second Type of Spinal Paralysis.
Lesion in pyramidal tracts.	Lesion in anterior gray horns.
Paralysis usually on both sides equally, in legs or in legs and arms, never in arms alone.	*Paralysis* may be limited to any single limb, and rarely affects both limbs equally.
All muscles are about equally affected. No muscles are entirely normal.	Certain groups of muscles only are affected. Others escape wholly.
Muscular tone is heightened.	Muscular tone is diminished.
Tendency to rigidity appears.	Muscles are relaxed.
Reflex excitability is increased.	Reflex excitability is lost.
Atrophy is absent or is slight, and merely due to disuse, hence is gradual in progress. It affects the entire limb.	*Atrophy* is always present in the paralyzed muscles. It advances rapidly, and may become extreme.
Electric contractility is unchanged.	*Electric contractility* is changed. Reaction of degeneration is present within two weeks of the onset.
Vascular tone is diminished; cyanosis and œdema may occur.	*Vascular tone* is diminished, but œdema does not occur.
Paralyzed limb is cold, and sweat may be increased.	Paralyzed limb is cool, but sweat is not increased.
Trophic disturbances in the skin are not infrequent.	*Trophic disturbances* in the skin do not occur.
The control over the bladder and rectum may be diminished or lost.	The control over the bladder and rectum is not impaired.
Example: Spastic paraplegia.	Example: Infantile paralysis.

The *third type of spinal paralysis* is a combination of the first and second types. When a transverse lesion of the spinal cord entirely destroys a single segment, it produces paralysis of the first type in the parts below the level of the lesion by cutting off the tracts to those parts, and paralysis of the second type at the level of the lesion by destroying the gray motor cells at that level The general effect of such a lesion depends entirely upon the level at which it occurs; the higher the lesion, the greater the extent of the first type of paralysis. The distribution of the second type will depend on the level of the segment involved. The greater the extent of the lesion at the level affected, the greater the extent of the second type of paralysis. An example of this is also found in amyotrophic lateral sclerosis. When a longitudinal lesion of great extent occurs—such as the general destruction of the cord in general myelitis—the second type of paralysis is the form which is found, but all the muscles are affected, not merely a few groups. The bladder and rectum are also affected, and bedsores are frequent.

In any case of spinal paralysis, if the electric condition of the muscles paralyzed be ascertained by the aid of a faradic battery, and the diagnostic points here brought together be applied, reference to the table of the localization of functions will enable the exact level of the lesion to be determined.

Spinal Anæsthesia.—The course of the sensory tract in the spinal cord is still somewhat imperfectly understood. It is known that all sensory impulses reach the spinal cord through the posterior nerve roots, which partly enter the apex of the posterior horn, and partly enter the column of Burdach, and pass upward as already described. The sensations of muscular sense ascend on the same side as that on which they enter. Those of touch, temperature, and pain cross over, as soon as they

enter, to the opposite side and ascend in it. The various views regarding the tracts transmitting these sensations have been already stated.

In transverse lesions of the spinal cord the area of anæsthesia present in the skin depends upon the level of the lesion. The various areas of the body which are related to the various segments of the spinal cord are shown in the plate. (Plate LII.) A lesion at any given segment will cause anæsthesia in the surface of the body related to that segment. If it is a transverse lesion of the cord it will cause anæsthesia in all parts of the body below the level of the area of anæsthesia caused by destruction of the affected segment. This plate has been made up from a study of a large number of cases with autopsy, and may be taken as a reliable guide to the level of the lesion. Transverse lesions higher than the fifth cervical segment cause sudden death from paralysis of the phrenic nerves. Limited areas of anæsthesia in the skin, at any part of the body, not corresponding to these areas, are to be ascribed rather to lesions in the peripheral nerves than to any local lesions in the cord itself; for posterior poliomyelitis as a distinct lesion is unknown. When a transverse lesion involves but one-half of the spinal cord, the anæsthesia is found upon the side opposite to the lesion, below the level of the lesion, and extends around the trunk in a band at the level of the lesion, the width of the anæsthetic band depending upon the longitudinal extent of the lesion. On the side of the lesion below the level of the anæsthetic band the skin is hypersensitive to touch. Such unilateral lesions produce a loss of muscular sense on the hyperæsthetic and paralyzed side, not upon the side of the anæsthesia—a fact which proves that the sensations of muscular sense do not decussate within the cord. This is the syndrome named after Brown-Séquard.

Hyperæsthesia sometimes occurs from spinal lesions, but is quite rare. It indicates an irritation of the sensory tracts in the cord by hyperæmia, or by pressure, rather than destruction of those tracts. Gowers suggests that this hyperæsthesia may be due to an increased irritability of the part of the cerebral cortex to which the injured tracts pass, as well as to an intensification of the impression passing in them. *Pain* is a rare symptom in spinal-cord disease, excepting in locomotor ataxia. And here it is to be ascribed to irritation of the posterior nerve roots within the cord, similar in character to their irritation without the cord, as occurs in meningitis and in diseases of the vertebral column. Its location on the surface is an indication of the level of the nerve root irritated, and by comparison with the plate this level can be diagnosticated. *Numbness* is a frequently mentioned symptom of spinal-cord disease, and has some value in local diagnosis, as the area of the skin in which the numbness is felt depends upon the level of the cord affected. Hence, when the numbness is limited to certain parts, especially to the extremities, a reference to the plate will indicate the segment of the cord which is diseased. Thus, in locomotor ataxia the beginning of numbness or pain in the little fingers indicates that the disease has advanced up the spinal cord and has reached the first dorsal and lowest cervical segments.

Spinal Ataxia.—This symptom always indicates an affection of the posterior nerve roots in their passage through the column of Burdach. Inco-ordination is due to an interference with the reception of sensations of muscular sense which are sent in from the skin, joints, and muscles. These sensations may be intercepted as they pass through the nerves, for ataxia is a symptom of toxic multiple neuritis; they may be intercepted as they pass through the column of Burdach, as is the case in locomotor ataxia; they may be intercepted as they pass up the cord in the column of Goll; they may be intercepted in the brain by lesions in the lemniscus (see *Brain*) or in the cerebellum. It is probable that a portion of the muscular sensations are sent to the gray matter of the cord, producing the reflex action of balancing, and unconscious co-ordination, and that the remainder are sent upward to the brain. For the inco-ordination in cerebral disease, when the latter only are disturbed, is less severe and intense than in spinal-cord disease, where all are implicated. Ataxia from neuritis is usually accompanied by tenderness in the nerves and muscles. Ataxia from cerebellar disease is only present in the act of walking, and is attended by vertigo. Ataxia from spinal disease is not attended by these two symptoms, but is usually accompanied by severe lightning pains and by loss of deep reflexes, together with other characteristic symptoms of locomotor ataxia. (Vide *Spinal-Cord Diseases: Tabes Spinalis.*)

It must be stated here that in lesions of the spinal cord, as in those of the nerves, the motor symptoms are usually more pronounced than the sensory symptoms; and even when the spinal cord is greatly compressed or disintegrated, sensory impulses may continue to pass after motor impulses are entirely arrested.

Disturbances of the Spinal Reflex, and Automatic Action.—Whatever view may be held regarding the nature of spinal reflex action, it is well established that certain structures are necessary to its production. It is necessary that a sensory nerve from the surface of the body be capable of transmitting impulses to the spinal cord. It is known that the fibres transmitting the centripetal impulses from the skin enter the apex of the posterior horn, while those transmitting impulses from the tendons enter the median surface of the posterior gray matter after traversing the lateral part of the column of Burdach called the root zone. It is also necessary that the network of nerve fibres through which impulses pass from the posterior gray matter to the cells of the anterior horn be intact. Finally, it is necessary that the groups of cells in the anterior horn, and the motor nerves from them to the muscles, be in a normal state, or capable of exercising their functions. These structures, together, make up a reflex arc, and a lesion in any part of this arc will arrest the reflex activity. Thus neuritis, outside the cord or due to meningitis, may interfere with the conduction of impulses to and from the cord; posterior sclerosis may arrest centripetal impulses as they reach the root zone; general myelitis may destroy the network of fibres within the gray matter; and anterior poliomyelitis may destroy the motor cells in the anterior horn. All these diseases, therefore, may cause a loss of tendon reflex. There are reflex activities governed by almost every segment of the cord, as may be seen in the table; and the particular reflex which is suspended in disease will depend wholly on the location of the lesion. Hence the loss of any one or more reflexes gives important information as to the seat of the lesion. And this can be ascertained after examination of the patient by a reference to the table. It has been already stated that an inhibitory influence is exerted by the brain upon spinal activity, and that this influence is conducted to the spinal motor cells through the motor tracts in the lateral column. Anything which impairs the conduction of impulses through this tract will result in removing restraint from the spinal reflexes and allowing them full sway. Hence an increase in deep spinal reflexes indicates a suspension of function in the lateral pyramidal tracts. A transverse myelitis, therefore, will cause an increase of the reflexes below the level of the lesion, and a loss of the spinal reflex governed at the level of the lesion. This has been already mentioned in connection with spinal paralysis. The skin reflexes are, however, not increased by lesions in the pyramidal tract.

The automatic activity of the cord includes the mechanisms of micturition and defecation. These mechanisms are complex reflexes, several sensory impulses combining to produce a compound motor effect, a part of which is inhibitory and a part of which is active. Thus, in micturition, the sensations of pressure on the sensitive neck of the bladder, and of distention of the entire organ, produce an inhibition of the motor impulses which normally hold the sphincter tight, and set in activity the motor impulses which contract the detrusor urinæ, thus emptying the bladder. The same is true, *mutatis mutandis*, of the other automatic acts. The structures necessary for

EXPLANATION OF PLATE LII.