dependent upon general cachexia or toxæmia—will be attempted in what follows. Anæmia has undoubtedly been given a place of undue importance in relation to these lesions. The insistence upon other and more general causes is due primarily to Putnam's work, published in 1891.

Pathological Anatomy.—The alterations in the cord in this type of disease consist macroscopically in degenerations of the white matter, chiefly in the dorsal and lateral tracts, irregular in distribution, and either not involving whole groups of fibres or extending beyond the confines of such tracts. The dorsal tracts, for example, are affected irregularly, groups of fibres in the immediate neighborhood of much degenerated areas are spared, and the root zones are rarely involved, in striking contrast to the systemic degeneration of tabes. The region of the pyramidal tracts is often much involved, but the alterations always extend beyond the confines of these tracts, chiefly along the peripheral portions of the cord, and on either side of the ventral fissure. The alterations are least marked in the lumbar region, and increase markedly in extent in the upper thoracic and cervical regions. Even to the naked eye the stained section often presents a peculiar, vacuolated appearance. Microscopic examination of the lesions shows the alterations to be essentially limited to the cord, with exceedingly slight alterations of the oblongata or hemispheres. The lesions are characterized by disintegration of myelinated fibres, overgrowth of neuroglia, in part sufficient to form a dense sclerosis and in part slight, vacuolation being due apparently to dilating and degenerating myeline sheaths, many fat granule cells, irregularity of distribution, often with very small foci of degeneration, scattered irregularly through the white matter, and at times focal softening. The gray matter is very slightly involved, and frequently not at all; the nerve roots are not involved; there is no evidence of meningeal thickening, and the blood-vessel walls rarely show changes. The pathological features common to the general group may, therefore, be summarized as follows:

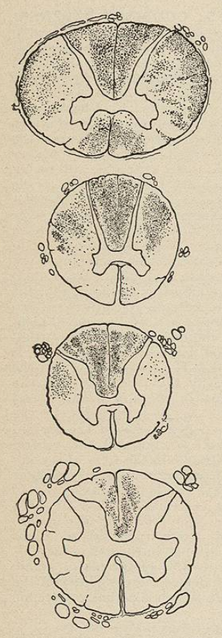

Fig. 4411.—Diffuse (Combined) Degeneration of the Spinal Cord.

1. A diffuse degeneration for the most part limited to the cord, often in more or less discrete patches.
2. A constant involvement of the dorsal and lateral columns, without strict regard to neurone systems.
3. A predominance of the lesion in the cervical and thoracic regions.
4. The common freedom from degeneration of nerve roots, both motor and sensory, and peripheral nerves.
5. The essential non-involvement of gray matter.
6. Insignificant vessel changes.

The reason for the peculiarly constant location of the lesions is probably to be sought in the distribution of the blood supply to the cord, a theory first advanced by Marie. Those who cling to the systemic character of the affection are inclined to search for a more subtle explanation, but the involvement of the cord alone, the tendency toward a peripheral distribution of the lesions, the non-involvement of gray matter, and the frequent occurrence of the disease in impoverished blood states, all indicate the rôle of the blood-vessels and their contained blood. Certain writers have maintained a double lesion, one of diffuse character and the other systemic, with possible secondary degenerations.

Etiology.—The persons in whom the disease occurs in typical form are usually of middle age, weak constitution, often with neurotic personal or family antecedents, of poor nutrition, cachectic in appearance, and of reduced bodily vigor. It is considerably more frequent in women than in men, according to Putnam's statistics. Syphilis may be disregarded as an etiological factor. A frequent though by no means universal accompaniment or possible cause of the disease is anæmia of the pernicious type. This association has led to the popular but wholly erroneous assumption that the alterations of the spinal cord described above are peculiar to anæmia. Lead poisoning, malaria, grave secondary anæmia, and other debilitating influences have also been given as causes, and the disease has at times occurred in otherwise healthy individuals. Overwork, excessive anxiety, gastro-intestinal disorders, may have a predisposing influence. Further than this our accurate knowledge does not go. Whether or not a subtle toxæmia, of a character as yet not understood, may ultimately be found responsible is possible, but far from being proved. It may be said with assurance at the present time that a well-characterized affection of the cord occurs in cachectic individuals toward middle life, leading to diffuse, quasi-systemic degenerations, dependent, in part at least, upon the distribution of the vascular supply.

Symptomatology.—In general, during the early stages, the symptoms are somewhat vague in character and may simulate functional disorders. The most conspicuous early symptom is paræsthesia of the extremities, noticed earlier in the feet. Disordered sensations may occur in other portions of the body—genitals, distributions of cranial nerves—but are much less usual. This paræsthesia is frequently very distressing. Slight disorders of micturition and rarely of defecation may occur. Objective disorders of sensation are not conspicuous, though they certainly occur, and in a well-reported group of cases (Russell, Batten, Collier) take an important place in the later stages of the clinical picture. Disorders of motion are of the character of a slight spastic paraplegia, with exaggerated deep reflexes, and the Babinski phenomenon. As the disease progresses, the symptom-complex assumes the general character of an ataxic paraplegia, with a predominance of ataxia or paraplegia depending upon the greater involvement of the dorsal or lateral columns. In later stages, as the lumbar portion of the cord is invaded, the knee-jerk may be abolished, and the patient be reduced to a helpless paraplegic condition, with loss of sphincter control. The mind is rarely affected, and the cranial nerves show no noteworthy involvement. Pigmentation of the skin, epileptic attacks, excessive diarrhœa or constipation, atrophy of the optic nerve, mental instability, dissociation of sensation, pain in the back and limbs, transient œdema, muscular atrophy, altered electrical reactions, have all been described as occasional symptoms.

Certain writers, notably Bastianelli and Russell, Batten and Collier, have attempted to classify cases either by etiology (Bastianelli) or by symptomatic course (Russell, etc.). The latter divide the disease sharply into three stages: first, slight ataxic paraplegia, constituting one-half to three-quarters of the duration of the illness; second, sudden loss of sensation, with increasing motor paresis, leading to inability to walk or stand, marked spastic paraplegia; third, stage of flaccidity, flaccid paraplegia, complete flaccid motor paralysis, complete anæsthesia, with loss of deep reflexes, incontinence, wasting

of muscles, and loss of faradic reaction. In the experience of others these sharp divisions cannot be maintained; such a course evidently marks a variety of the general affection, which is highly important, but cannot be regarded as characteristic of the entire group. In general, it must be said that on the symptomatic side attempts to deduce a perfectly characteristic clinical picture have as yet failed, although the grouping of the symptoms of primary paræsthesia, ataxic paraplegia increasing rather rapidly in intensity, followed by a stage of more or less complete motor and sensory paralysis, in the absence of lancinating pains and Argyll-Robertson pupil, point strongly toward combined lesions of the character under consideration.

Diagnosis, Course, and Prognosis.—The diagnosis in the later stages should not be difficult, and in the early stages should be assumed as probable in cases in which an otherwise unexplained and persistent paræsthesia, especially of the legs, is associated with slight ataxia and a tendency toward spasticity with exaggerated deep reflexes, particularly if the patient be of middle age, with evidences of cachexia. Multiple sclerosis may simulate this affection at various stages, but in general it may be distinguished by the youth of its victims, the characteristic speech defect and tremor, with a marked preponderance of motor over sensory involvements. Myelitis should also receive consideration in the differential diagnosis. Uncomplicated tabes should give rise to no confusion.

The course of the disease is from six months to two years, with occasional instances of much longer duration. As a rule, the patients are already in a debilitated condition when the symptoms manifest themselves, a fact which evidently must have a more or less direct bearing upon the outcome.

The prognosis is always bad, both from the point of view of the general condition of the patient and also because of the usually rapidly advancing cord degenerations.

Treatment.—Attention should first be directed toward the discovery and amelioration of the underlying condition causative of or associated with the alterations in the cord. Anæmia, whether of the so-called pernicious variety or secondary, general debility, and disordered bodily functions in general, should be treated by the recognized means at our disposal. It is possible that much might be done in the earliest stages to avoid or modify the serious and ultimately irremediable cord degenerations. If they have developed, however, and the patient is ataxic, or ataxic paraplegic, recourse should certainly be had to the Frenkel exercises, modified to meet the indications in individual cases. The usual drugs—iron, strychnine, arsenic, the iodides, etc.—may be given, but with small hope of permanent benefit.

II. *Combined Dorsal and Lateral Systemic (Neurone) Disease.*—In spite of the doubt which has been thrown upon the existence of a true neurone degeneration involving dorsal and lateral tracts, cases have been reported clinically, and in a few instances anatomically verified, of the existence of such a combined lesion (Oppenheim). Degenerations of the columns of Goll and Burdach, the pyramidal tract, the direct cerebellar tract, and less frequently of Gowers, and the uncrossed pyramidal tracts, have been described, with the assumption that these tracts were degenerated as systems and not as a part of a more general process.

Such cases of primary degeneration of lateral and dorsal neurone systems give rise to the symptoms of tabes and lateral sclerosis, the one or other type of symptom predominating in direct relation to the extent of the dorsal or lateral pathological change. Symptoms of tabes, with lancinating pains and Argyll-Robertson pupil, but with exaggerated knee-jerk, should always give rise to a strong suspicion of a combined systemic degeneration, giving the general clinical picture of ataxic paraplegia. Should the definite signs of tabes, including loss of knee-jerk, be associated with muscular weakness, a similar diagnosis of combined lesion is justified. The signs of tabes, on the other hand, may be in great measure subordinated to those of lateral degeneration—spastic paraplegia. In this case slight disorders of sensation, sharp pains, condition of the pupils, bladder weakness, should be carefully investigated to determine the possible involvement of sensory neurones. Ataxic paraplegia, as commonly observed, is no doubt usually due to the more diffuse process described above. The important differential point is the existence of a true tabes, which does not occur in the diffuse combined lesions.

The treatment is as indicated for the foregoing disease, and for Friedreich's ataxia.

III. *Combined Systemic Disease.*—(*a*) *Result of congenital defect (Friedreich's ataxia); cerebellar ataxia; hereditary cerebellar ataxia;* (*b*) *in dementia paralytica.*

(*a*) *Friedreich's Ataxia.*—See article on this subject.

Cerebellar Ataxia.—Hereditary cerebellar ataxia, type Nonne-Marie, bears a close resemblance to Friedreich's ataxia, except for the facts that the knee-jerks are increased, the pupils are immobile, there is atrophy of the optic nerve, there are marked disorders of sensibility, and club-foot and kyphosis do not develop. Lesions of the cerebellum and other portions of the brain have been found, and in one case (Menzel) a combined system disease of the cord.

(*b*) *Combined Systemic Disease in Dementia Paralytica.*—Alterations of the spinal cord (Westphal), in the form of degeneration of the pyramidal tracts or of the dorsal, sensory tracts, or of both, are frequent in dementia paralytica. Whether the alterations in the pyramidal tracts are to be regarded as primary neurone degenerations or as secondary to changes in the cortex remains in doubt.

Gowers' Ataxic Paraplegia.—In 1886 Gowers drew attention to a condition presenting the symptoms of degeneration in the dorsal and lateral tracts, to which the clinical name ataxic paraplegia was given. The classification of this condition is difficult, but from the description of the pathological alterations it should rather be included among the diffuse combined degenerations than among the true systemic diseases. After what has been said it does not require separate description.

E. W. Taylor.

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SPINAL-CORD DISEASES: CONGESTION OF THE CORD.—Our actual knowledge regarding spinal congestion is very limited, and hypothetical statements that it is the basis of many nervous symptoms are unwarranted. The diseases in which it is uniformly found after death are those in which the patient has died in convulsions complicated by asphyxia, or in the early stages of myelitis. The only positive evidence that a spinal congestion has existed during life is the discovery of distended capillaries, accompanied by small capillary hemorrhages. Without the latter the congestion found may have been a post-mortem occurrence, due to the position of the body on the back.

The *causes of active congestion* are excessive muscular exertion, violent sexual excesses, poisoning by strychnine, alcohol, and carbonic oxide, the sudden arrest of menstruation or the stoppage of hemorrhage from piles, and possibly exposure to cold. Traumatism of the vertebræ, especially general concussion of the spine, such as

occurs in railway injuries, probably causes active spinal hyperæmia, in a few cases examined capillary hemorrhages having been found after death in the cord. It is probable that spinal congestion is usually localized in the lower half of the cord, though the entire organ may be affected. An active hyperæmia of the anterior cornua is the first occurrence in poliomyelitis anterior, and this is usually quite extensive, while the actual process of inflammatory degeneration is subsequently limited to a small area. The latter produces the permanent atrophic paralysis of infants; to the former must be ascribed the temporary paresis of the first stage of the disease, which is always more extensive than the permanent paralysis.

The causes of passive congestion are the same as those producing this effect in the brain or other organs (*q.v.*).

The *symptoms of spinal congestion* are a sensation of weight and fatigue in the legs and back, increased by any effort, so that continued exertion is impossible; pains, numbness, formication, and sensations of heat and cold, with increased susceptibility to changes of temperature and to pain and touch in the extremities; weakness, but not paralysis, in the entire muscular system, attended by an increase of reflex excitability; a diminution of sexual power, and a diminution of control over the bladder and rectum not sufficient to be termed incontinence. Any symptoms more serious than these, such as severe tearing pains in the back and general hyperæsthesia, or such as girdle sensations, incontinence, and actual paralysis, must be ascribed to congestion of the spinal meninges as well as of the cord, or to disturbances of a grave nutritive kind initial to an actual myelitis. These symptoms are always bilateral and usually more marked in the lower half of the body, although the arms may become involved. They usually come on suddenly after some known cause; but occasionally a chronic congestion is suspected, which lasts for months, and is attended by symptoms of neurasthenia.

The *diagnosis of spinal congestion* is an uncertain one. When symptoms such as those described appear, and do not go on to more serious conditions, but gradually pass off, and no adequate cause can be found, the diagnosis can be made. If, however, serious symptoms of myelitis ensue, it must be admitted that the disease was myelitis from the outset, and not a simple hyperæmia. A long duration of symptoms of spinal congestion points rather to the existence of capillary hemorrhages, attended by small foci of inflammatory degeneration, or to nutritive changes in the spinal cord.

The *prognosis* should always be reserved, in view of the uncertainty of diagnosis and the possibility of myelitis.

The *treatment* should consist of absolute rest in bed, in a prone position; the application of cool cloths, wet with an evaporating lotion, to the spine, or an ice bag or the ether spray; saline purgatives; and full doses of ergot with small doses of belladonna. *M. Allen Starr.*

SPINAL-CORD DISEASES: DIAGNOSIS OF LOCAL LESIONS IN THE CORD.—The spinal cord is a long, cylindrical organ, made up of numerous segments, each of which not only has a function of its own, but also bears an important part in relation to the functions of other segments. Each segment of the cord consists of a mass of gray matter, surrounded by a series of white tracts, from which passes out a pair of spinal nerves. In some of the lowest order of vertebrata the comparative independence of each segment is indicated by the fact that the spinal cord consists of a series of bulbous enlargements joined together by only a few connecting fibres. And even in man there are some evidences that the functions of each segment of the cord are independent of all others. But in the higher vertebrata the various segments are closely united to one another, and are also connected with the brain, which controls them all by means of the white tracts surrounding the gray matter. Hence, in addition to its own special function as a nervous centre, each segment has functions of transmission of impulses to adjacent segments and to distant parts of the nervous system. Therefore, in dealing with local lesions in the spinal cord, the first point to determine is whether the lesion involves the nerve centres of a single segment, or the tracts which pass through that segment to other centres. In the first case, when the gray matter of a single segment is affected, the symptoms are limited in extent and in number, consisting of localized paralysis, limited anæsthesia, loss of certain reflexes, disturbance of certain automatic actions, and local vaso-motor and trophic disturbances. In the second case, when the white matter of a single segment is affected, the symptoms are widespread and numerous, consisting of partial or complete paraplegia, anæsthesia of the lower half or even of the entire body, loss of control over reflex and automatic activity, and extensive vascular and trophic changes. And when both gray and white matter of a single segment are totally involved, there will be a combination of local and general symptoms, the distribution and extent of which will depend wholly upon the particular level of the segment of the cord which is affected. It is therefore evident that the first step in the diagnosis of local lesions of the spinal cord is the determination of the functions of the various segments, and of the various tracts which pass through them.

I. The Functions of the Segments of the Spinal Cord.—Each segment of the spinal cord consists of that portion of the entire organ which gives origin to one pair of spinal nerves. There are, therefore, thirty-one segments in the human cord. There is no natural division between adjacent segments, but if a cord with its nerves be carefully removed, there will be no difficulty in cutting it up into segments, each of which will receive two afferent and give off two efferent nerves. Each segment is made up of two symmetrical halves, naturally separated by the anterior fissure and posterior septum, but joined by a commissure.

The afferent or sensory nerves enter the posterior surface of the segment, and, passing through the white matter, end in the gray. The efferent or motor nerves pass out from the anterior surface of the segment, having their origin in the anterior gray horn, and traversing the white matter bordering these horns. The figure (Fig. 4412) shows four such segments at different levels of the cord with their afferent (*s*) and efferent (*m*) nerves as well as the motor tracts and association tracts to the various segments.

A. The Gray Matter.—The size and shape of the area of gray matter, seen in horizontal section of the cord, differ in almost every segment, the difference between adjacent segments being more marked in the cervical and lumbar enlargements than in the dorsal region. The shape of the area of gray matter in the dorsal region resembles that of the letter **H**, and, accordingly, anatomists describe two lateral halves with a central gray commissure between them, and in each half an anterior and a posterior horn. In the enlargements of the cord the mass of the horns is much larger than in the dorsal region, and the shape varies in each segment. The amount of gray matter in any segment depends upon the number of cells in the anterior and posterior horns. These cells are not scattered irregularly through the gray matter, but are collected into groups. These groups are quite distinct in the anterior horns, in some cases being small, and found only in a single segment; in others being long, and extending through several segments. This varying arrangement of the anterior groups at different levels is seen in the figure (Fig. 4413). The function of the cells in the anterior horn is to govern the motion and nutrition of the muscles.

The more exact localization of motor functions in the groups of cells in the cervical and lumbar enlargements has been attempted by various writers. Fig. 4413 shows the groups found at three different segments of the cord. There are some of these groups—viz., the inner antero-lateral and postero-lateral groups—which develop early in fœtal life, and are common to man and the less highly developed vertebrates. These are thought to govern the fundamental movements common to man and animals,

and earliest acquired in children, viz., flexion and extension, abduction and adduction of the limbs. Other groups, viz., anterior and median groups, are found only in monkeys and man, and the central groups in man alone. These are also found to develop later than the others. They are therefore thought to govern the accessory movements, which are more specialized and depend on finer adjustment, such as the act of walking upright, pronation and supination, and the finer motions of the hands and fingers, actions which are learned some months later than those of a fundamental kind. In some cases of disease, limited to these groups of cells—*e.g.*, poliomyelitis anterior—the symptoms have been found to justify this distinction, loss of power and atrophy of certain muscles being produced by a lesion in certain groups of cells. The connection of the different groups, in various segments, with individual muscles, as far as at present known, is shown in the table accompanying this article.[1] (See p. 340.)

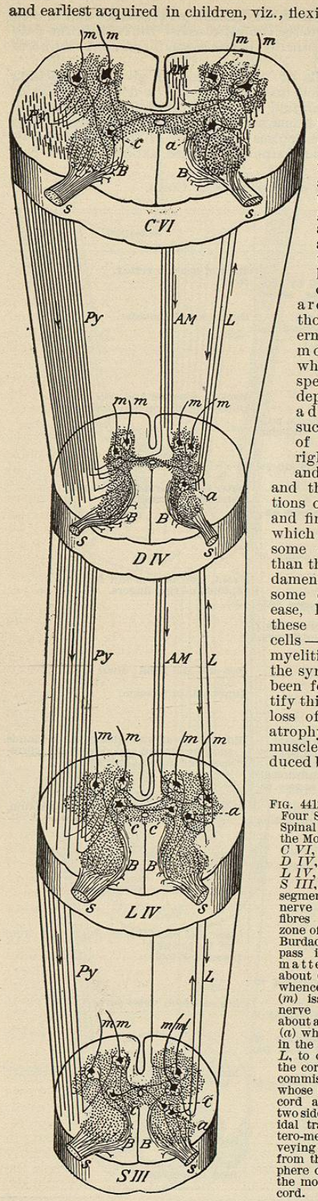

Fig. 4412.—Diagram of Four Segments of the Spinal Cord to Show the Motor Mechanism. *C VI*, Sixth cervical, *D IV*, fourth dorsal, *L IV*, fourth lumbar, *S III*, third sacral segments: *s*, sensory nerve root whose fibres enter the root zone of the column of Burdach, *B*, and then pass into the gray matter terminating about (1) motor cells whence motor fibres (*m*) issue as motor nerve roots; or, (2) about association cells (*a*) whose fibres pass in the lateral column *L*, to other levels of the cord, or (3) about commissural cells (*c*), whose fibres cross the cord associating the two sides. *Py*, Pyramidal tract; *AM*, antero-median tract conveying motor impulses from the right hemisphere of the brain to the motor cells of the cord.

The arrangement of cells in the posterior horns is different from that in the anterior horns. There is a column

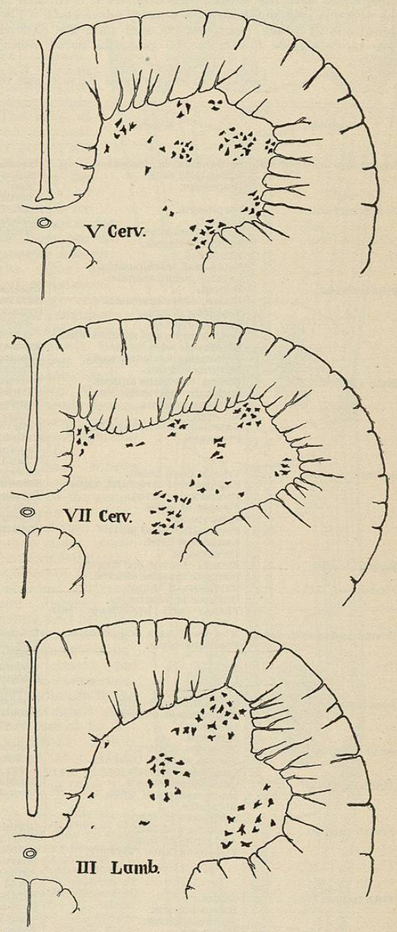

Fig. 4413.—Showing the Different Grouping of the Cells in the Gray Matter of the Spinal Cord at Three Different Levels. (Drawn from photographs.)

of cells extending through the lower dorsal region, known as the vesicular column of Clarke, and situated in the median and inner part of the horn (see Fig. 4412, *c*). The column begins in the third lumbar segment, and extends upward to the seventh dorsal segment. Its probable function is to regulate the vaso-motor and sympathetic nervous mechanisms.[2] There is a continuous

column of cells in the middle of the posterior horn, not collected together into groups, but scattered through the neuroglia and gelatinous substance of the posterior horn. These cells are very small in size, and thus contrast markedly with those already described. It is probable that the gelatinous mass in the posterior horn has something to do with the sensory function, for it is present in the nervous system wherever a sensory nerve ends. The sensations of touch, temperature, and pain are carried into the posterior horns by the posterior nerve roots, which terminate in the gelatinous substance and in cells which lie about the central canal of the cord in the gray matter at the junction of the posterior and anterior horns (Fig. 4414, *X*).

All the cells of the gray matter give off branching processes which interlace, forming a dense network of nerve fibrillæ around the group of cells. This interlacing mass of fibres is composed partly of the dendrites of cells which collect impulses, and partly of the axones of cells which send out impulses. These axones in their passage through the gray matter send off little fibrillary branches termed collaterals, which terminate in end-tassels about other cell bodies. Through this gray matter impulses pass in all directions, uniting the functions of the various cell groups, and of the sensory and motor areas of the cord. It is probable that impulses coming to the segment, either from the periphery or from the brain, are conveyed directly to the cells of the segment, and are distributed through the medium of the branches of the cells to several groups of cells.

Furthermore, each segment controls in some degree the processes of nutrition in the part of the body with which its sensory nerves are connected. It regulates the vasomotor tone in the organs and limbs, and it influences processes of growth and repair in the skin and mucous membranes. But the existence of trophic cells in the cord has not been proven. Nor can the automatic mechanisms of the cord be assigned to definite cells. They can only be referred to the gray matter of certain segments.

LOCALIZATION OF THE FUNCTIONS OF THE SEGMENTS OF THE SPINAL CORD.

Segment.	Muscles.	Reflex.	Sensation.
Second and third cervical.	Sterno-mastoid, trapezius. Scaleni and neck. Diaphragm.	Hypochondrium (?) Sudden inspiration, produced by sudden pressure beneath the lower border of the ribs.	Back of head to vertex. Neck.
Fourth cervical.....	Diaphragm. Deltoid. Biceps, coracobrachialis. Supinator longus. Rhomboid. Supra- and infraspinatus. Levator anguli scapulæ.	Pupil, fourth to seventh cervical. Dilatation of the pupil produced by irritation of neck. Scapular.	Outer part of shoulder.
Fifth cervical.....	Deltoid. Biceps, coracobrachialis. Supinator longus. Supinator brevis. Deep muscles and shoulder blade. Rhomboid, teres minor. Pectoralis (clavicular part). Serratus magnus.	Fifth cervical to first dorsal. Irritation of skin over the scapula produces contraction of the scapular muscles. Supinator longus. Tapping its tendon in wrist produces flexion of forearm.	Back of shoulder and arm. Outer side of arm and forearm.
Sixth cervical.....	Biceps, brachialis anticus. Pectoralis (clavicular part). Serratus magnus. Triceps. Extensors of wrist and fingers. Pronators.	Triceps. Fifth to sixth cervical. Tapping elbow tendon produces extension of forearm. Posterior wrist. Sixth to eighth cervical. Tapping tendons causes extension of hand.	Outer side of arm and forearm. Outer half of hand.
Seventh cervical.....	Triceps (long head). Extensors of wrist and fingers. Pronators of wrist. Flexors of wrist. Subscapular. Pectoralis (costal part). Latissimus dorsi. Teres major.	Anterior wrist. Seventh to eighth cervical. Tapping anterior tendon causes flexion of wrist. Palmar, seventh cervical to first dorsal. Stroking palm causes closure of fingers.	Front, back of arm and forearm. Middle and ring fingers.
Eighth cervical.....	Flexors of wrist and fingers. Intrinsic muscles of hand.	.....	Forearm and hand; ulnar area.
First dorsal.....	Extensors of thumb. Intrinsic hand muscles. Thenar and hypothenar eminences.	.....	Inner side of forearm.
Second and twelfth dorsal	Muscles of back and abdomen. Erectores spinæ.	Epigastric, fourth to seventh dorsal. Tickling mammary region causes retraction of the epigastrium. Abdominal, seventh to eleventh dorsal. Stroking side of abdomen causes retraction of belly.	Skin of chest and abdomen, in bands running around and downward, corresponding to spinal nerves.
First lumbar.....	Quadratus lumborum. Transversalis obliqui. Ilio-psoas. Sartorius.	Cremasteric, first to third lumbar. Stroking inner thigh causes retraction of scrotum.	Skin over groin and in front of scrotum.
Second lumbar.....	Ilio-psoas, sartorius. Flexors of knee (Remak). Quadriceps femoris.	Patella tendon. Striking tendon causes extension of leg.	Outer side of hip. Front of thigh.
Third lumbar.....	Quadriceps femoris. Inner rotators of thigh. Obturator. Adductors of thigh.	Bladder centre. Second to fourth lumbar.	Front of thigh. Inner side of leg.
Fourth lumbar.....	Abductors of thigh. Abductors of thigh. Flexors of knee (Ferrier).	Rectal centre. Fourth lumbar to second sacral. Gluteal. Fourth to fifth lumbar. Stroking buttock causes dimpling in fold of buttock.	Outer and back side of thigh and front of leg to ankle. Dorsum of foot.
Fifth lumbar.....	Glutei. Biceps femoris. Semitendinosus. Popliteus. Outward rotators of thigh. Flexors of knee (Ferrier).	Achilles tendon. Over-extension causes rapid flexion of ankle, called ankle clonus. Babinski reflex. Scratching sole of foot causes extension of great toe. Fifth lumbar to first sacral.	Leg and foot, outer part.
First and second sacral...	Biceps femoris. Semimembranosus. Extensor longus digitorum. Gastric. Tibialis posticus. Tibialis anticus.	Plantar. Tickling sole of foot causes flexion of toes and retraction of leg.	Back of thigh and leg in saddle-shaped area. Inner side of foot.
Third sacral.....	Peronei. Intrinsic muscles of foot.	.....	Back of buttock, seat.
Fourth and fifth sacral...	Sphincter ani et vesicæ. Perineal muscles.	.....	Perineum, anus. Back of scrotum.

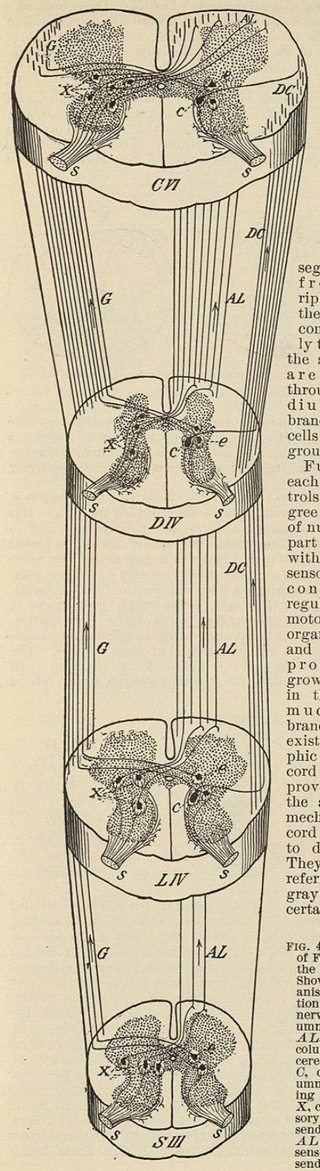

FIG. 4414. — Diagram of Four Segments of the Spinal Cord to Show Sensory Mechanism and Conduction. *S*, Sensory nerve root; *G*, column of Gowers; *AL*, antero-lateral column; *DC*, direct cerebellar column; *C*, cells of the column of Clarke sending fibres to *DC*; *X*, cells of the sensory gray matter sending fibres to *AL*; *e*, cells of the sensory gray matter sending fibres to *G*.

The function of the gray matter of the individual segments is shown in the preceding table. The facts upon which this table has been prepared are gathered from comparative anatomy, from physiological experiment, and from pathological observation. The level of the segment is given; the muscles governed by the groups of cells in it are mentioned so far as they are at present known; the reflex and automatic mechanisms governed by each segment are recorded, and the manner of producing the reflex acts; and the area of skin which sends its sensory nerves to the individual segments is described.

B. The White Tracts. — The gray matter of the spinal segment is surrounded by white nerve tracts, whose function is the transmission of impulses between adjacent and distant segments, and between the various segments and the brain. These tracts are quite numerous, and although in the normal adult cord they cannot be distinguished from one another, there are several means by which their lim-

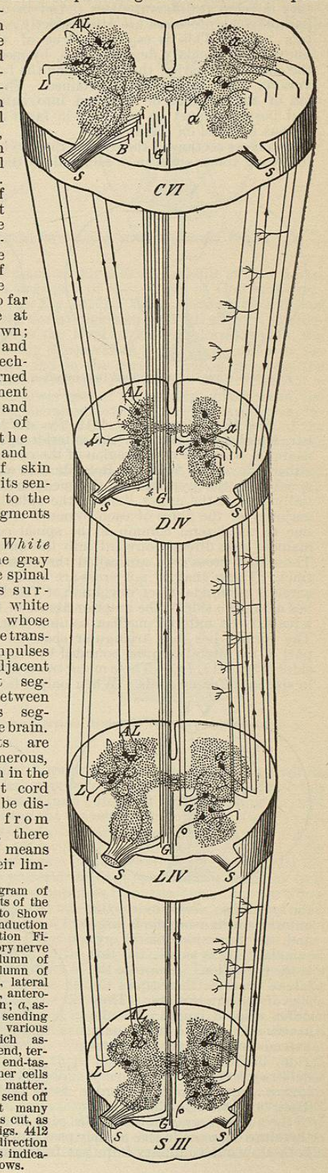

FIG. 4415.—Diagram of Four Segments of the Spinal Cord to Show Sensory Conduction and Association Fibres. *S*, Sensory nerve root; *G*, column of Goll; *B*, column of Burdach; *L*, lateral column; *AL*, antero-lateral column; *a*, association cells sending axones into various columns which ascend or descend, terminating in end-tassels about other cells in the gray matter. These axones send off collaterals at many levels. In this cut, as well as in Figs. 4412 and 4414, the direction of impulses is indicated by the arrows.