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 para-lyzed 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

Hyperasthesia 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 spinalcord 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: Tables Spinalis)

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 AUTO-MATIC 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 spi nal 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 col-umn of Burdach called the root zone. It is also necessarv 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 re straint 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.

## EXPLANATION OF PLATE LII.

The segments of the spinal cord are numbered:  $C_I$  to  $C_{VII}$ ,  $D_I$  to  $D_{XII}$ ,  $L_I$  to  $L_V$ ,  $S_I$  to  $S_V$ ; and these numbers are placed, in the plate, on the region of the skin supplied by the sensory nerves of the corresponding segment. In order to determine, in any given case of spinal-cord disease, the level of the cord affected, it is necessary to test the sensations and to compare the area of anæsthesia with the diagram here given. It is to be remembered, however, that the skin of the body is plentifully supplied with sensory nerves which anastomose freely at their terminations, and the researches of Sherrington have demonstrated that each part of the skin is supplied with sensory nerves from two adjacent segments of the cord. Hence a condition of anæsthesia in the skin indicates a suspension of function of two segments of the cord at least, for if one segment alone were affected the segments above and below it would be capable of supplying the skin with sensation.



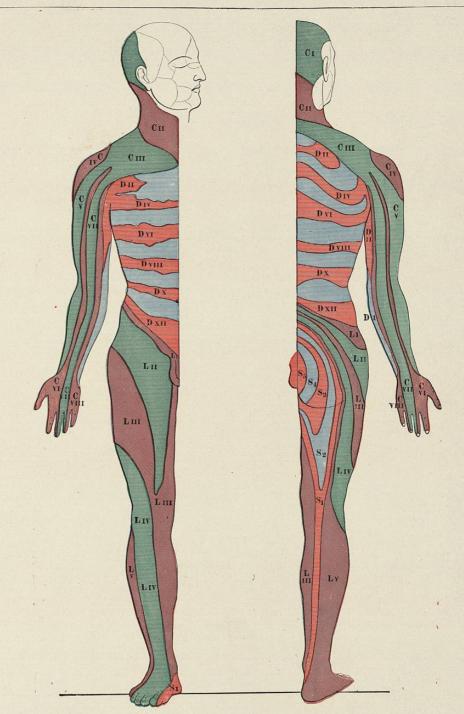
PLATE SHOWING THE AREAS OF THE STATE SOON WHICH ARE

o, cervical: d, dorsal: l, humber: s, sucreit. Then the second of the body is annexthetic

(From Dr. M. Allen Starr)

### EXPLANATION OF PLATE LII.

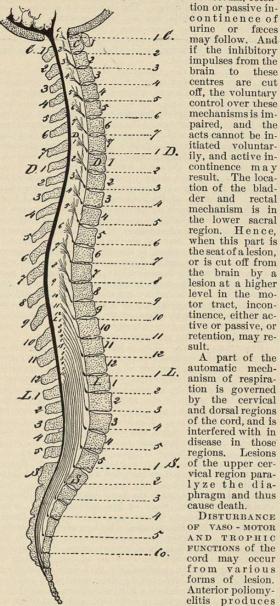
to SY and the second is the part on the region of the skin supplied by the sensory northern the sensations and to compare the area of another the sensations and to compare the area of another the sensations and to compare the area of another the sensations and to compare the area of another the sensations and to compare the area of another the sensations and the remembered, however, that the skin of the body as the sense of the sense of the skin is supplied with sensory never the sense of the cord there a condition of anæsthesia in the skin indicates a sense of the cord at least, for if one segment alone were after the sense of supplying the skin with sensation.



# PLATE SHOWING THE AREAS OF THE SURFACE OF THE BODY WHICH ARE RELATED TO THE VARIOUS SEGMENTS OF THE SPINAL CORD

c, cervical: d, dorsal: l, lumbar: s, sacral. When a segment of the cord is destroyed the surface of the body is anæsthetic in the area corresponding to that segment. (From Dr. M. Allen Starr)

any one of these acts are similar to those underlying the | there is a partial vaso-motor paralysis, indicated by cyasimple spinal reflex, and the same lesions arresting it may arrest these acts. But the result of such arrest is more serious, for, in the case of the bladder or rectum, reten-



A part of the

DISTURBANCE

Fig. 4420.—Relations between the Segments atrophy of the of the Spinal Cord and their Nerves and the Bodies of the Vertebræ. (Gowers.) atrophy of the muscles parabodies of the Vertebræ.

cient affection of the vaso-motor system to cause objective, as well as subjective, coldness in the limb; and when the lesion lies deep in the anterior horn, an arrest of development of the bones of the limb affected. General myelitis is usually associated with a tendency to bedsores upon the parts exposed to pressure, which cannot be avoided by the most scrupulous cleanliness, and to cystitis, and these are ascribed to a disturbance of trophic impulses to the skin and bladder. Pos terior sclerosis is sometimes associated with trophic changes, such as perforating ulcers, joint affections (Charcot's arthropathies), and eruptions on the skin. In a few cases of leprosy serious lesions of the posterior gray horns have been observed. In general myelitis [1861, and a detailed account published by him in 1863, with three autopsies. In 1876 he published a further paper on the subject, with a description of three new

nosis, sluggish circulation, edema, and coldness, with abnormal sweating in the paralyzed parts. But any definite statement regarding the exact localization of vaso-motor or trophic functions in the spinal cord cannot be made as yet. And recently many vaso-motor and trophic symptoms, formerly supposed to be due to spinal lesions, have been found to be produced by disease in the peripheral nerves. It is, however, established that trophic lesions are most frequently observed when the gray matter of the spinal cord in the vicinity of the central canal, including the vesicular column of Clarke, is the part diseased; or when all sensation is cut off from the paralyzed limbs by a transverse lesion.

The regulation of urinary excretion is presided over by a centre in the medulla, and the nerve tract thence to the liver and kidneys is traced through the cervical region of the spinal cord to the first dorsal segment, where it enters the sympathetic chain of ganglia. A lesion in the lateral column of the cervical cord, by involving this tract, may cause a vaso-motor paralysis of either the liver or the kidneys. In the former case diabetes mellitus is produced; in the latter, diabetes insipidus results. It is therefore necessary, in lesions of the spinal cord, to examine the amount and constituents of the urine.

In any case of spinal disease in which it is desirable to localize accurately the lesion, it is suggested that a written summary of the symptoms be compared with the table of localization of the functions of the cord, when it will become evident, by contrasting the normal with the abnormal conditions, what part of the cord is affected. As Bramwell justly observes, "the essence of the clinical examination of the spinal cord consists in the systematic and separate examination of each spinal segment, by observing the motor, sensory, reflex, vaso-motor, and tro-phic conditions of its body area." Such an examination will lead to accurate diagnosis of local lesions.

But one point remains to be mentioned, that is, the re-lation of the various segments of the cord to the bodies and spines of the various vertebræ. As the cord extends only to the level of the second lumbar vertebra, its various segments do not lie opposite to the vertebræ from which they are named. The accompanying diagram of Gowers (Fig. 4420) displays the mutual relation between the segments and their nerves, and the bodies of the vertebræ, and no further description is needed.

#### REFERENCES.

- <sup>1</sup> For a full account of the grouping of these cells, see Localization of the Functions of the Spinal Cord, by M. A. Starr, American Journal of Neurology and Psychiatry, vol. iii., pp. 443 et seq.—Ross: Diseases of the Nervous System, p. 829.

  <sup>2</sup> Gaskell: Journal of Physiology, 1886.

  <sup>3</sup> See article on Brain, Diagnosis of Local Lesions in the.—The Motor Tract, in Vol. II. of this HANDBOOK.

  <sup>4</sup> Schultze, F.: Ueber Secondäre Degeneration im Rückenmarke. Arch. f. Psych., xiv., from which article the figures are taken.

  <sup>6</sup> For the anatomy of this motor tract, see the article referred to above, in Vol. II. of this HANDBOOK.

### AUTHORITIES.

Gowers: Diseases of the Spinal Cord. Lüderitz; Ueber das Rückenmark-Segment. Arch. f. Anat. u. Phys., 1881.

1881.
Ferrier: Brain, vol. iv., p. 223.
Ferrier: Brain, vol. iv., p. 223.
Beevor: Med. Chirurg. Transactions, vol. lxxviii.
Starr: Localization of the Functions of the Spinal Cord. Amer. Jour. Neurol. and Psych., August and November, 1884.—Localization of Lesions in the Lumbar Cord. Amer. Jour. Med. Sci., 1893.—Localization of Lesions in the Cervical Cord. Brain, 1895.
Thorburn: A Contribution to the Surgery of the Spinal Cord, 1893.
T. Kocher: Die Verletzungen der Wirbelsäule, Jena, 1896.
H. Schlesinger: Beiträge zur Klinik der Rückenmarks- und Wirbelstumoren, Jena, 1898.
R. Wichmann: Die Rückenmarksnerven und ihre Segment-Bezüge, Berlin, 1900.

SPINAL-CORD DISEASES: FRIEDREICH'S (HERED-ITARY) ATAXIA.—The condition known as Friedreich's or hereditary ataxia was first described by Friedreich in