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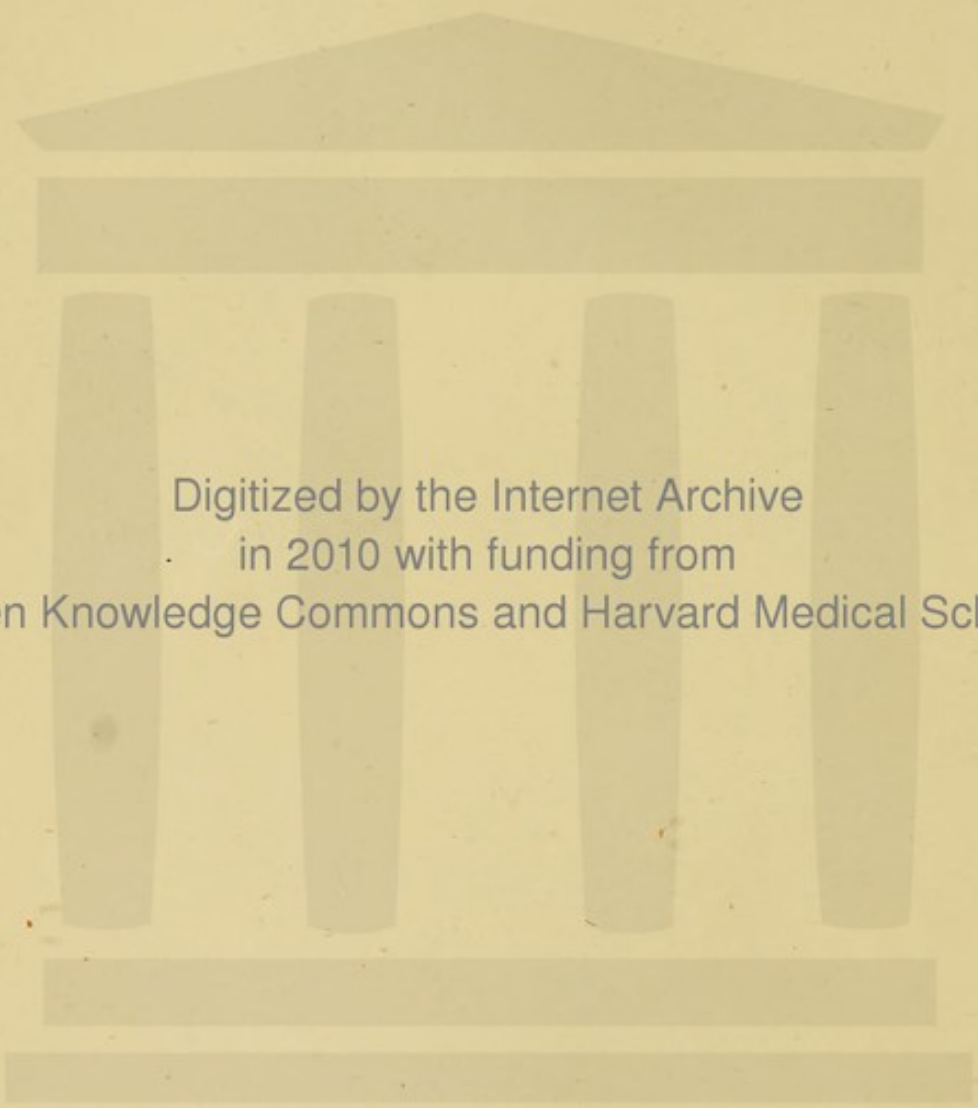
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THE DISEASES OF THE SPINAL CORD


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THE

DISEASES OF THE SPINAL CORD

BY

BYROM BRAMWELL

M.D. F.R.C.P. (EDIN.)

LECTURER ON THE PRINCIPLES AND PRACTICE OF MEDICINE, AND ON MEDICAL DIAGNOSIS, IN THE EXTRA-ACADEMICAL SCHOOL OF MEDICINE, EDINBURGH;

ADDITIONAL EXAMINER IN CLINICAL MEDICINE IN THE UNIVERSITY OF EDINBURGH

LATE PHYSICIAN AND PATHOLOGIST TO THE NEWCASTLE-ON-TYNE INFIRMARY;

LATE LECTURER ON PATHOLOGY, AND ON CLINICAL MEDICINE IN THE UNIVERSITY OF DURHAM COLLEGE OF MEDICINE, NEWCASTLE-ON-TYNE;

FORMERLY MEDICAL OFFICER TO THE TYNEMOUTH UNION WORKHOUSE HOSPITAL, AND TO THE PRUDHOE MEMORIAL CONVALESCENT HOME, ETC., ETC.

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7088

TO

PROFESSOR CHARCOT OF PARIS,

AND

PROFESSOR ERB OF LEIPZIG,

TO WHOSE WRITINGS I AM LARGELY INDEBTED FOR MY KNOWLEDGE OF DISEASES OF THE SPINAL CORD,

THE FOLLOWING PAGES ARE RESPECTFULLY DEDICATED

BY

THE AUTHOR.

PREFACE.

In the following pages, which are based on a portion of my course of Lectures on Medicine, I have endeavoured to give a concise description of the more important points relating to the Diseases of the Spinal Cord.

In some places the original form is retained, but for the most part I have, for the sake of condensation, abandoned the colloquial style of the lecture-room, and in many places arranged the matter in the form of headings.

Believing that one great secret of all successful teaching is to teach by the eye as well as by the ear, I am in the habit of copiously illustrating my lectures by diagrams, drawings, and microscopical preparations. The diagrams and drawings are introduced into the text in the form of woodcuts, the microscopical sections are represented in colours.

The chromo-lithographs are all drawn by myself, first with the camera lucida, and then in lithograph chalk; they are with two exceptions (figures 56 and 151, which are copied from Charcot) representations of my own sections.

I am indebted to Professor Dreschfeld of Manchester, Dr Robertson of Glasgow, Dr Crease of South Shields, Dr Banham of Sheffield, Dr Goyder of Newcastle, and Dr Milner Moore of Coventry, for spinal cords, from which some of the sections were made; and I am particularly indebted to Dr D. J. Hamilton, not only for material, but also for much valuable instruction in the methods of investigating nervous structures.

My thanks are also due to Professor Charcot, Professor Flower, Professor Ferrier, Dr Ross, Dr Gowers, and Dr Herbert Tibbits, for their kindness in allowing me to reproduce some of the figures which have appeared in their respective works.

A considerable portion of the first two chapters was published in the *Transactions* of the Northumberland and Durham Medical Society for November and December 1881.

B. B.

23 Drumsheugh Gardens,
Edinburgh, *March* 1882.

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AN INTRODUCTION

TO

THE DISEASES OF THE SPINAL CORD.

CHAPTER I.

THE ANATOMY AND PHYSIOLOGY OF THE SPINAL SEGMENT.

§ 1. Until quite recently the affections of the spinal cord were generally looked upon as so obscure and difficult as to baffle the understanding of any but the most accomplished specialist. Thanks, however, to the labours of Leyden, Charcot, Erb, Gowers, Buzzard, and others too numerous to mention, the difficulties are rapidly disappearing, and in a short time we shall probably be as well acquainted with the diseases of the spinal cord as with any other affections to which the human economy is liable.

In attacking any complicated clinical question the difficulties are very much reduced if we enter upon our work with a clear idea of the functions of the affected organ, and with a definite plan of study; and to no group of affections does this statement apply more forcibly than to the diseases of the spinal cord.

The spinal cord may be said to consist of a series of segments placed one above another. Each segment comprises the portion of cord to which a pair of spinal nerves is attached (see fig. 1); and each segment may be viewed as a distinct spinal unit, or, to speak somewhat figuratively, as a distinct spinal cord for a definite area of the body, viz., that portion of muscle (muscular area) to which its anterior roots proceed; and that portion of skin, tendon, muscle, mucous membrane, viscus, etc. (sensitive area)

to which the fibres of its posterior nerve root are distributed.[1]

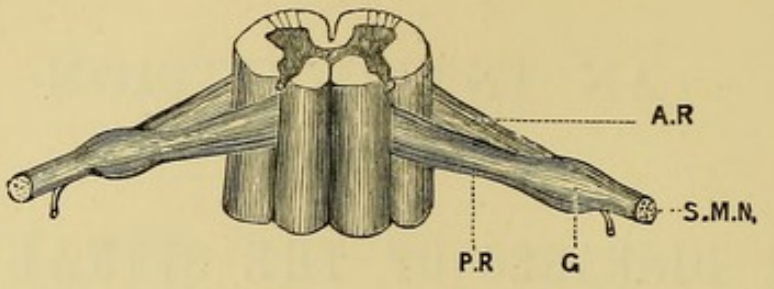


FIG. 1.

A Spinal Segment with its Pair of Spinal Nerves (modified from Quain and Sharpey).

AR—Anterior root. PR—Posterior root. G—Ganglion on posterior root. SMN—Transversely divided common sensory-motor nerve trunk.

Now 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 trophic conditions of its body area; and the comprehension of the diseases of the spinal cord consists essentially in the correct understanding of the structure and functions of the individual parts of these spinal units; of the manner in which they are related to each other; of the pathological changes to which they are liable, and of the derangements in function which result therefrom.

As yet we are not sufficiently well acquainted with the exact distribution and function of each pair of spinal nerves (*i.e.* of each segment) to enable us to examine separately the body area of each individual spinal unit. It must, too, be remembered, that adjacent segments are probably related functionally; and that, in actual practice, it is often necessary or convenient to group several segments together, and to examine, for example, the parts supplied by the lumbar or cervical enlargement as a whole. The more detailed examination of each individual segment (so far as we can at present apply it) should, however, always be employed for scientific purposes, or where we wish to ascertain the exact position and extent of a spinal lesion.

---

[1] It is simpler to consider each lateral half of a spinal segment as a spinal unit and, indeed, I used formerly to do so; but the arrangement adopted in the text, though slightly more complicated, is more correct.

The recent clinical observations of Gowers[1] on the reflexes, of Remak[2] on atrophic spinal paralysis, and the physiological researches of Ferrier and Yeo,[3] and of Paul Bert and Marcacci[4] on the function of the different spinal nerves, have supplied valuable information as to the motor functions of individual segments; and it is probable that before long we shall be sufficiently acquainted with the function of all the more important spinal segments to be able to ascertain their condition by the examination of their respective body areas.

The functions of individual segments, so far as they are at present known, will afterwards be detailed.

I shall now proceed to direct your attention to those points in the anatomy and physiology of the spinal cord which are essential for our present purpose.

THE ANATOMY OF THE SPINAL SEGMENT.

§ 2. Speaking generally, we may describe a segment of the spinal cord as a disc of nervous tissue to which a pair of spinal nerves is attached; each nerve arising by an anterior and a posterior root. (See fig 2.)

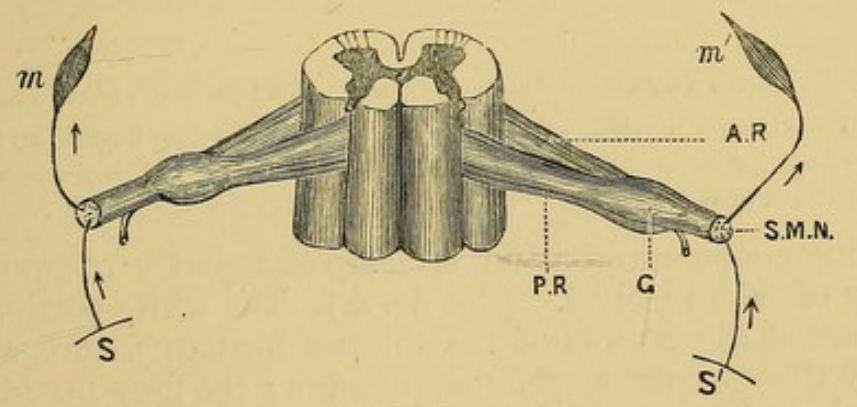


FIG. 2.

A Spinal Segment with its Muscular and Sensitive Body-Areas Attached.

AR—Anterior root. P.R—Posterior root. G—Ganglion on posterior root. S.M.N—Transversely divided sensory-motor nerve trunk. m, m′—Muscular area of the segment. S, S′, Sensitive area of the segment.

Each segment is divided into two symmetrical lateral halves by the anterior and posterior median fissures. The

---

[1] *The Diagnosis of Diseases of the Spinal Cord*, page 15, *et seq.*

[2] *Archiv für Psychiatrie*, 1879, page 510.

[3] *Proceedings of the Royal Society*, No. 212, page 12.

[4] Quoted in the *Lancet*, Oct. 1, 1881, page 598.

division is not quite complete, for a narrow band of nervous tissue remains, towards the centre of the segment, separating the two fissures, and connecting the two lateral halves of which it (the segment) is made up. (See fig. 3.)

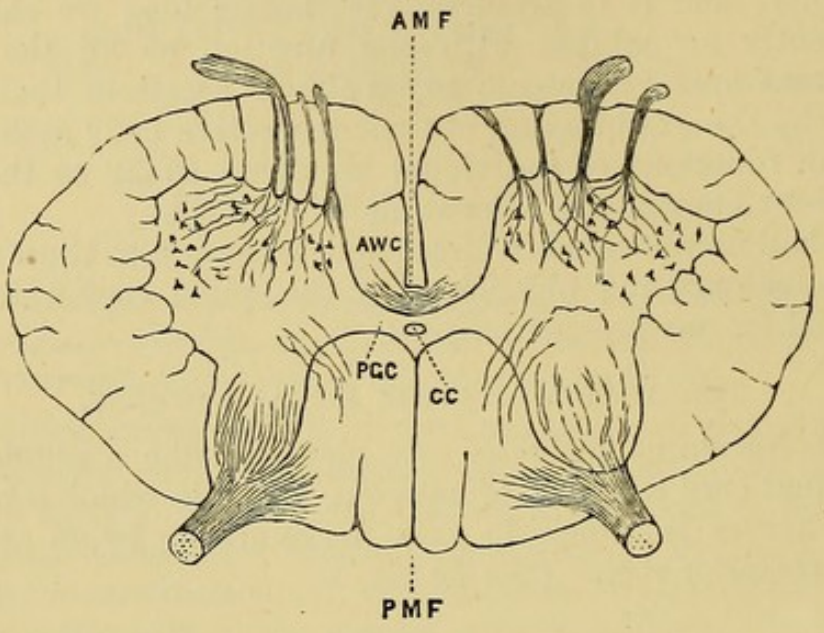


FIG. 3.

Transverse Section of a Spinal Segment in the Cervical region.

AMF—Anterior median fissure. PMF—Posterior median fissure or septum. AWC—Anterior or white commissure. PGC—Posterior or grey commissure. CC—Central Canal.

This commissural band is composed partly of grey and partly of white matter. (See fig. 4.) The white portion lies in front, at the bottom of the anterior median fissure, and is called the *anterior* or *white commissure;* the grey matter lies posteriorly between the anterior white commissure and the posterior median fissure or septum (for the posterior median fissure is seldom a fissure properly so called, but rather a septum of connective tissue, as represented in fig. 3), and is called the *posterior* or *grey commissure.*[1]

In the middle of the posterior or grey commissure the central canal of the spinal cord is placed. (See fig 5.)

[1] Some writers give the term *posterior* grey commissure to that portion of the grey commissure which is situated *behind* the central canal and call the part of the grey commissure which lies *in front* of the central canal the *anterior* grey commissure.

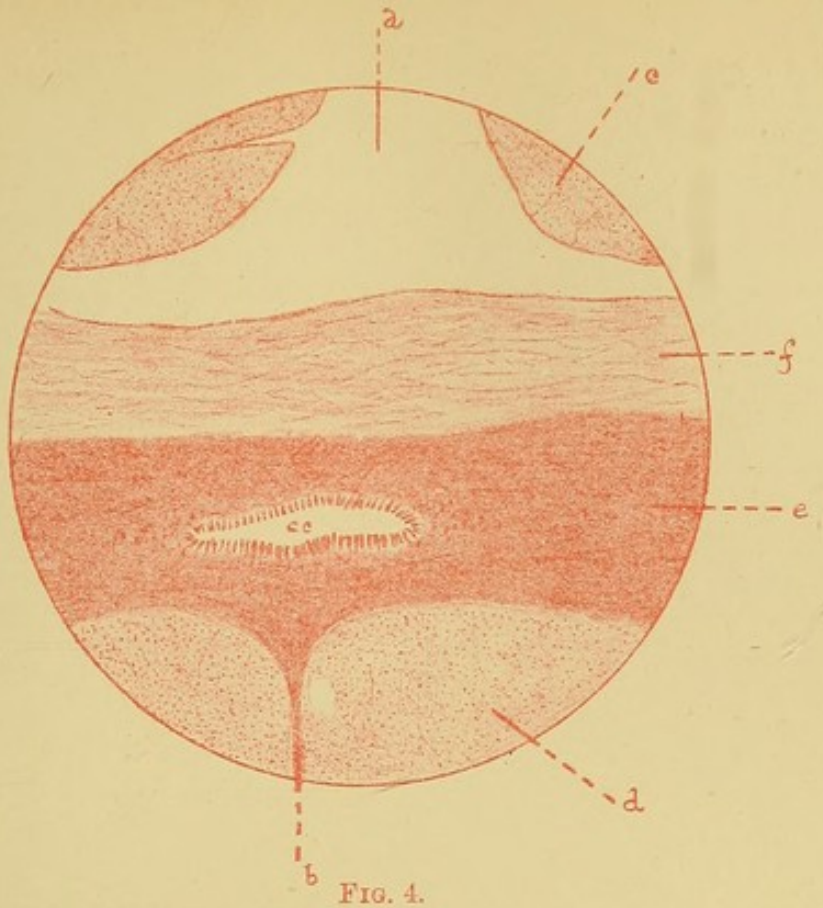


FIG. 4.

Transverse Section of the Spinal Cord, showing the commissures (carmine and dammar), magnified about 70 diameters.

a Anterior median fissure. *b* Posterior median fissure or septum. *c* Right anterior column. *d* Right posterior column. *e* Posterior or grey commissure, with cc, the central canal in its interior. *f* Anterior or white commissure.

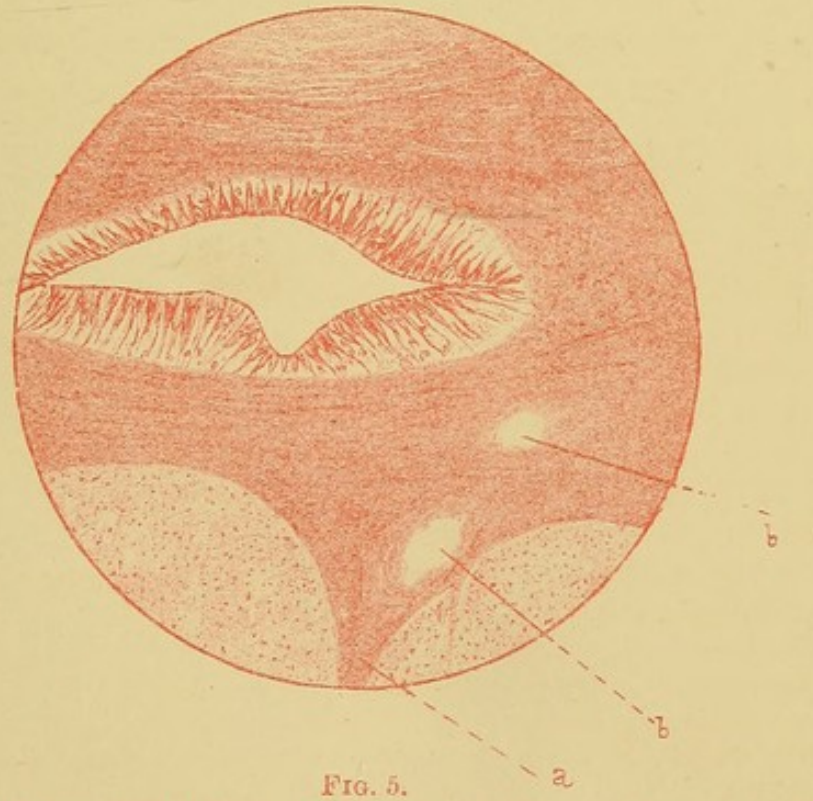


FIG. 5.

Transverse Section through the Spinal Cord of a Child, showing the central canal with its ciliated epithelial lining (carmine and dammar) magnified about 250 diameters.

a Posterior median septum. b, b Openings for blood vessels in the grey matter.

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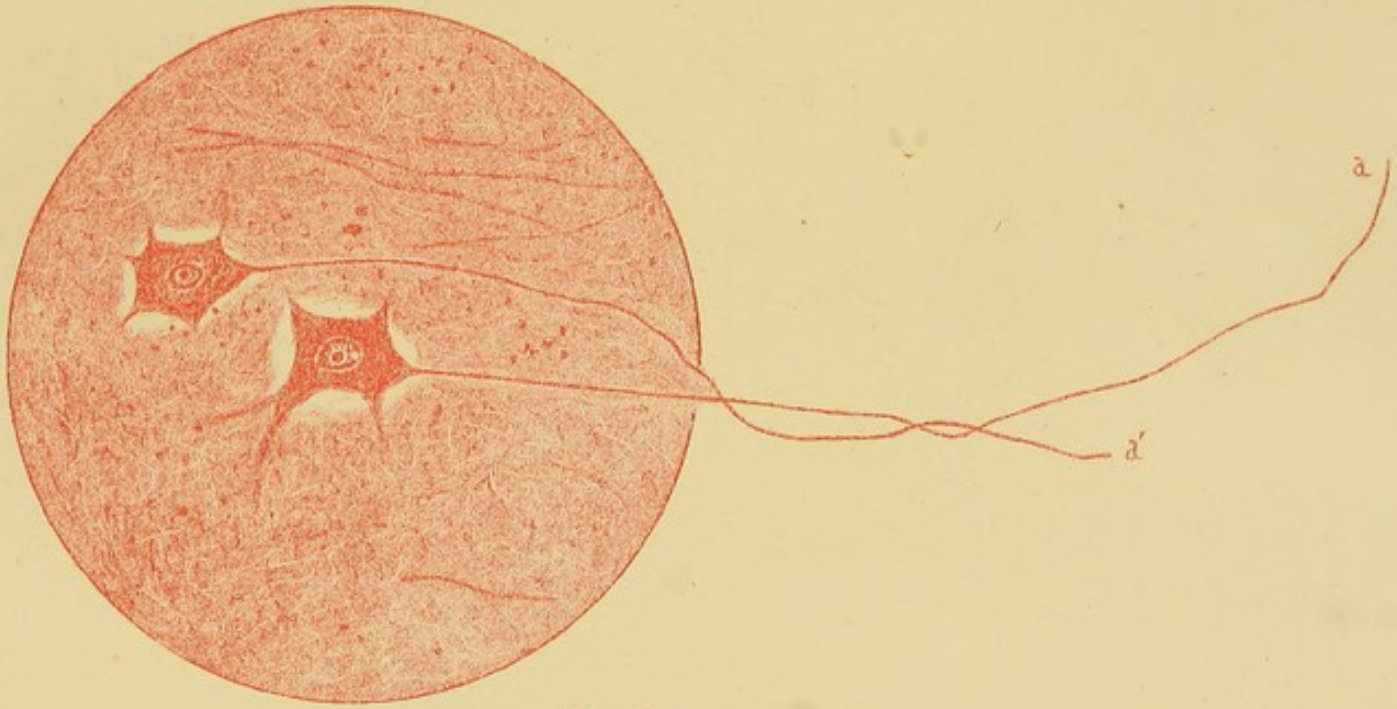


FIG. 7.

Two Multipolar Nerve Cells from the Lumbar Enlargement of Man, with long axis cylinder processes a, a', (carmine and dammar), magnified about 200 diameters.

The axis cylinder process of the lower cell can be traced as far as the point *a*. The circle represents one field of the microscope.

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Each half segment consists partly of grey and partly of white matter. The grey matter (see fig. 6) is placed in the

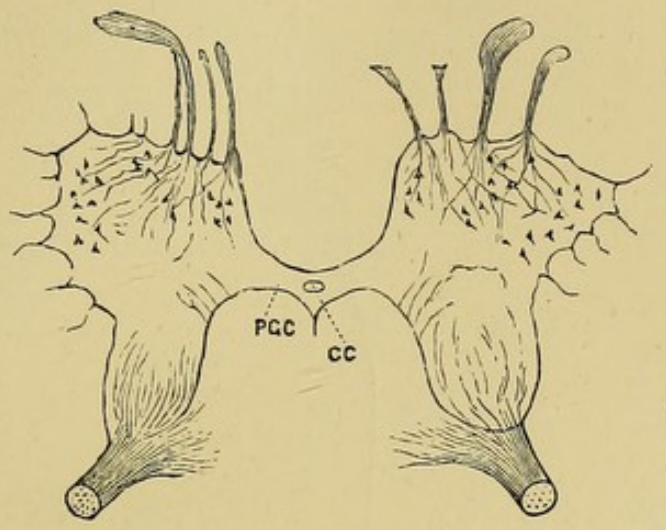


FIG. 6.

The grey matter of the Spinal Segment.

PGC—Posterior or grey commissure. CC—Central canal.

centre of the half segment, and is completely surrounded by white matter except at one point—about the middle of its inner border—where it is prolonged towards the middle line, forming the corresponding half of the posterior or grey commissure. This mass of grey matter consists of two extremities or horns (cornua), as they are termed, joined by an intermediate connecting portion. The *anterior cornu* is much larger than the posterior, and contains the motor nerve cells, which are of the greatest physiological and pathological importance. The posterior horn also contains nerve cells. They are smaller than those in the anterior cornu, and are more spindle-shaped. Their function is probably sensory.

The motor cells are of large size, and are multipolar. (See fig. 7.) One of the poles, which is slender and unbranched, is termed the axis cylinder process. (See figs. 7 and 8.) In very favourable sections it may be traced forwards into the anterior root-fibres, which in leaving the cord pass through the anterior column in several separate bundles. (See figs. 3 and 21.) The other poles of the cell divide almost immediately after leaving the cell body into numerous branches, which are supposed to terminate in the

delicate network of nerve fibrils which runs through the grey matter. (See figs. 8 and 19.)

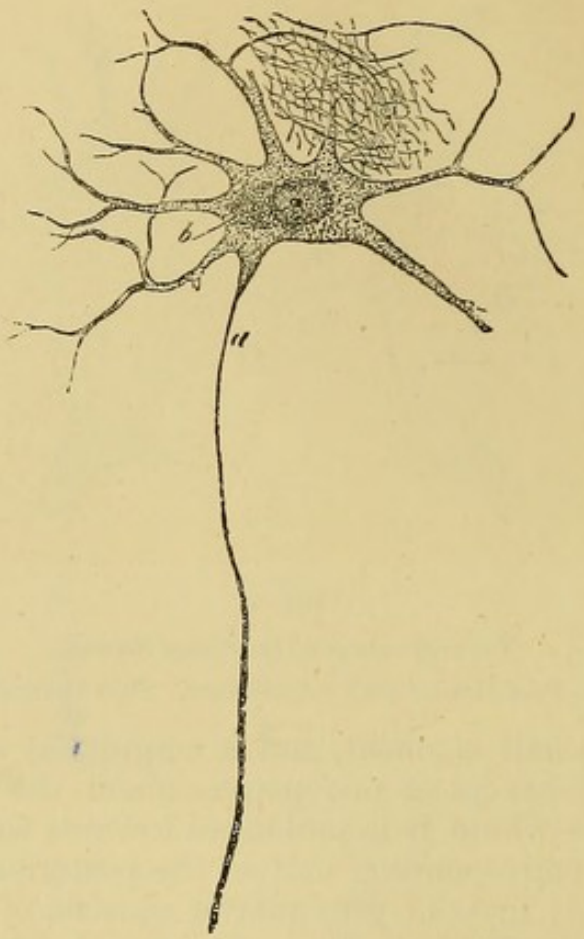


FIG. 8.

Nerve Cell from the Anterior Horn of the Spinal Cord; and Gerlach's nerve network. (*After Gerlach*). *a*—Axis cylinder process. *b*—pigment granules in the cell.

The white matter of the segment has been divided into the following parts or columns:—

1. *The posterior column:* That portion of the white matter which is situated between the posterior median fissure and the posterior cornu of grey matter. (See figs. 9 and 10.)

2. *The lateral column:* That portion of white matter which is situated between the posterior and anterior nerve roots.

3. *The anterior column:* That portion of the white matter which is situated between the anterior median fissure and

the lateral column, *i.e.*, the outermost of the anterior root bundles.

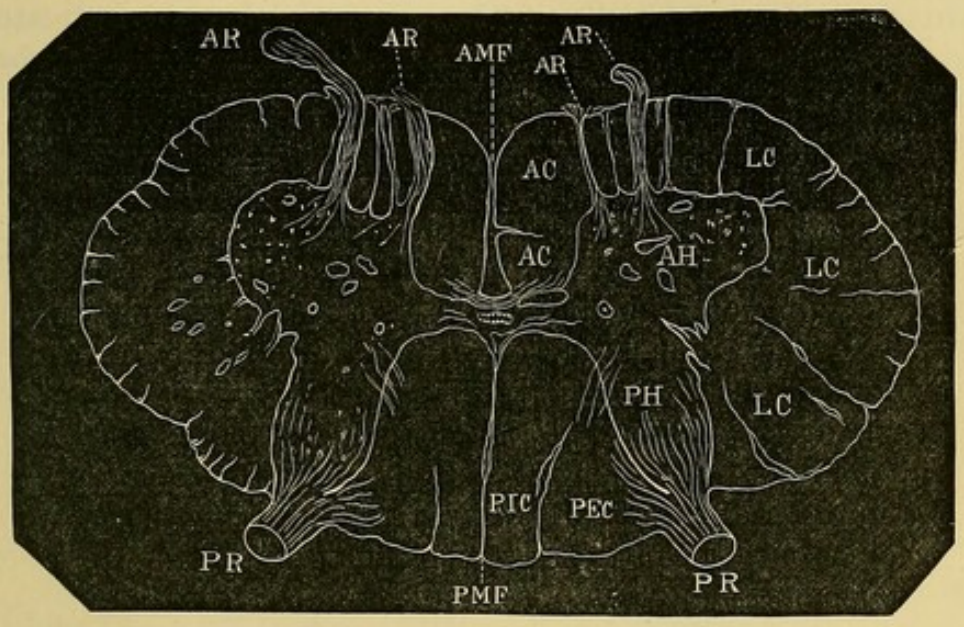


FIG. 9.

Transverse Section of the Spinal Cord in the Cervical Region, from a camera lucida drawing, magnified about six diameters.

AC—Anterior column. LC—Lateral column. PEC—Postero-external column. PIC—Postero-internal column. AH—Anterior horn of grey matter. PH—Posterior horn of grey matter. AR—Anterior nerve roots emerging in separate bundles. PR—Posterior nerve roots entering in a single bundle. AMF—Anterior median fissure. PMF—Posterior median fissure. The circular spaces in the grey matter are openings for blood vessels; they are abnormally large in this specimen. Numerous nerve cells are seen in the anterior cornua.

§ 3. But in addition to this, which we may term the anatomical sub-division of the white matter, pathological observations, and the recent most important researches of Flechsig[1] on the development of the spinal cord, show that each of these columns consists of certain sub-divisions or tracts, all[2] having a distinct physiological function, and, therefore, of the greatest possible clinical importance.

The posterior column is divided into two parts, viz., (1) an internal tract, the *postero-median column* of anatomists, the *postero-internal column*, as Dr Gowers proposes to term it, or the *column of Goll*, as it is often called (see PIC fig. 10); and (2) an external tract, the *postero-external column* of

[1] Die Leitungsbahnen im Gehirn und Rückenmark des Menschen.

[2] The exact physiological function of some of these tracts is still unknown.

Gowers, the root-zone column of Charcot, the wedge-shaped tract (*funiculus cuneatus*) of Flechsig, or the column of Burdach. (See PEC, fig. 10.) The division is indicated anatomically by a depression on the surface of the cord and a septum of connective tissue passing inwards from it.

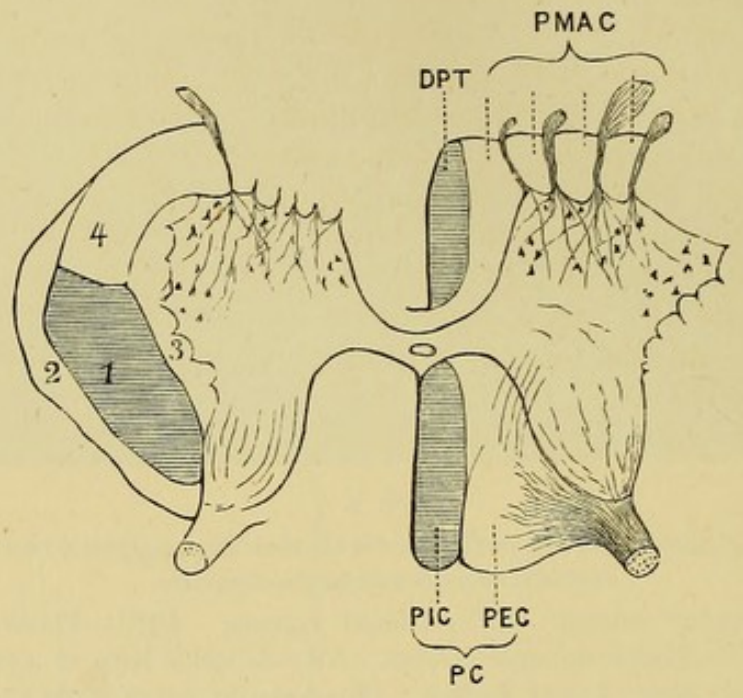


FIG. 10.

Diagrammatic Section of a Spinal Segment showing the Physiological division of the White Matter.

The lateral column of the right lateral half and the anterior and posterior columns of the left lateral half have been cut away. PIC — Postero-internal column. PEC — Postero-external column. DPT — Direct pyramidal tract. PMAC—Principal mass of the anterior column. 1. Crossed pyramidal tract. 2. Direct cerebellar tract. 3. Lateral boundary layer of the grey substance. 4. The anterior mixed region of the lateral column.

The *postero-internal column* is chiefly composed of fibres coming from the posterior grey cornu and from the posterior or grey commissure. Their function is not yet definitely known, but they probably conduct upwards sensory impressions.

The *postero-external column* is composed of fibres of the posterior root passing inwards to the posterior cornu; and of (?) commissural fibres, which probably connect the posterior cornua of different segments. The fibres of the posterior

root, which pass through the postero-external column, carry into the cord peripheral impressions resulting in the production of sensations (probably of touch and pain), and of reflex movement (probably the deep reflexes).

The *lateral column* is divided by Flechsig into four areas or tracts, the most important of which (indeed the only one whose function is as yet definitely known) is termed the *crossed pyramidal tract.* (See 1, fig. 10.) This important area varies in size in different segments. (See fig. 11.) In the cervical enlargement it is large and triangular, and occupies the greater part of the posterior half of the lateral column, being separated by narrow bands of white matter from the grey matter and the periphery of the cord respectively (see figs. 10 and 11). It diminishes in size in passing downwards, and ends in the lumbar enlargement, where it touches the periphery. (See 7, fig. 11.)

The crossed pyramidal tract contains the greater number of the fibres of the main pyramidal or motor tract, viz., all those fibres which have decussated at the lower end of the medulla. Its function, therefore, is essentially to conduct voluntary motor impulses.

In addition to the crossed pyramidal tract, Flechsig distinguishes three other subdivisions of the lateral column, viz.:—

(*a*). The *lateral boundary* or *limiting layer of the grey substance* (see 4, fig. 11), the connection and function of which are unknown.

(*b*). The *direct cerebellar tract* (see 6, fig. 11). This tract appears in the upper part of the lumbar enlargement, and increases in passing upwards. It receives fibres from Clarke's group of cells (see VC, fig. 12). Above, it passes into the cerebellum.

(*c*). The *anterior mixed region of the lateral column* (see 3, fig. 11), the function of which is still unknown.

The *anterior column* is, like the posterior, subdivided into two parts:—(1). An inner sub-division, the *direct pyramidal tract* or column of Türck (see 7, fig. 11), which contains the motor fibres of the pyramidal tract which have not decussated in the medulla. (2). The *principal mass of the anterior column* (see 1, fig. 11), which seems to be composed chiefly of fibres of the anterior roots and of (?) commissural fibres connecting the anterior cornua of different segments.

The order of development of these different tracts is shown in fig. 11,[1] and is, according to Flechsig, as follows :—

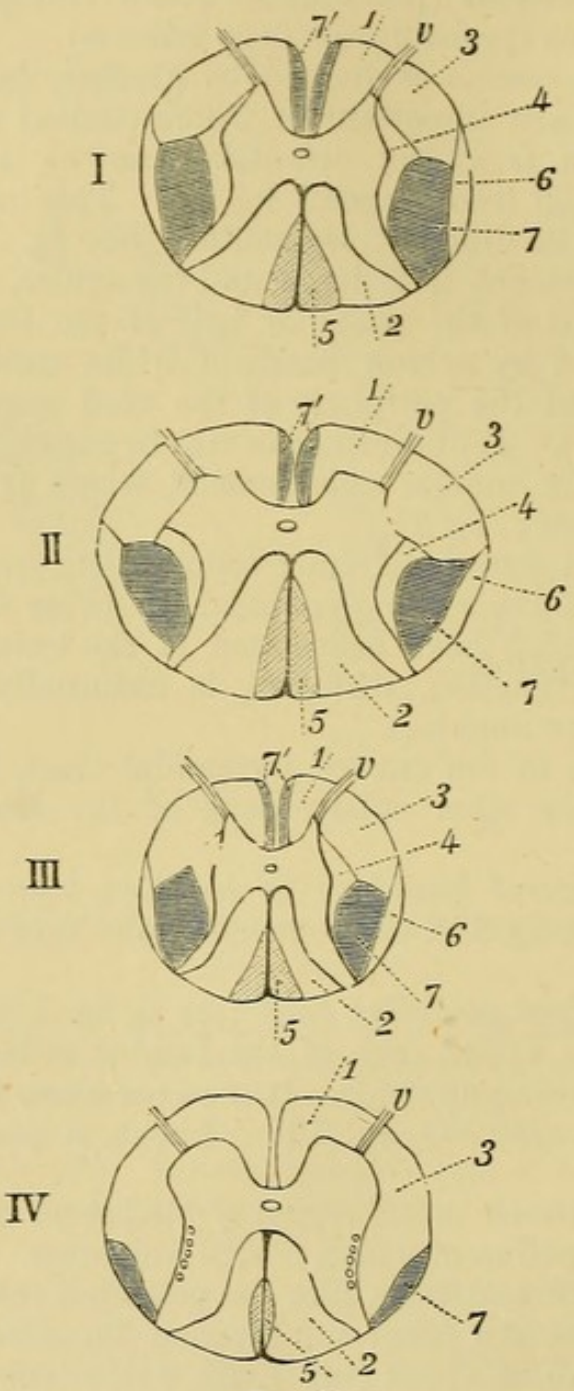


FIG. 11.

Diagram showing the different physiological tracts at various levels of the cord; and the order in which they are developed. (After Flechsig).

I. Section at the height of the 3rd cervical; II. at the height of the 5th cervical; III. at the height of the 6th dorsal; and IV. at the height of the 4th lumbar nerves.

1. Principal mass of the anterior column; 2. Wedge shaped tract; 3. Anterior mixed region of the lateral column; 4. Lateral boundary layer of the grey substance; 5. Column of Goll; 6. Direct cerebellar tract; 7. Pyramidal tract of the lateral columns; 7′. Pyramidal tract of the anterior column; *v*. Anterior root.

[1] Copied from the supplement to *Ziemssen's Cyclopædia*, page 568.

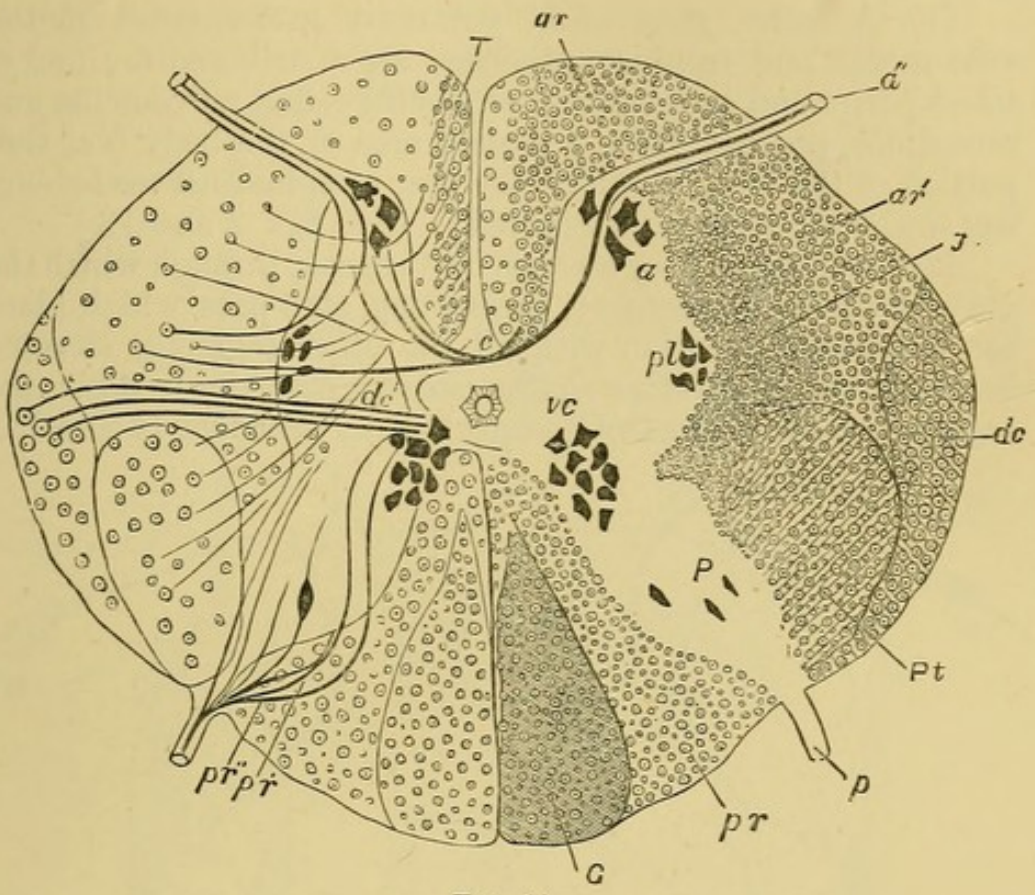


FIG. 12.

Diagram of transverse section of the Spinal Cord in the upper half of the dorsal region. (After Flechsig).

VC—Vesicular column of Clarke. *dc'*—Fibres which pass from the cells of the column of Clarke to the direct cerebellar tract. *dc*—Direct cerebellar tract.

First. The principal mass of the anterior column and the wedge-shaped column. *Second.* The anterior mixed region of the lateral column. *Third.* The lateral boundary layer of the grey substance. *Fourth.* The column of Goll. *Fifth.* The direct cerebellar tract. *Sixth.* The direct and crossed pyramidal tracts.

THE PHYSIOLOGY OF THE SPINAL SEGMENT.

§ 4. Such, then, is a brief statement of the most important points in the anatomy of the spinal segment. Passing now to physiology you will find, that the functional relations of its various parts are much more easily understood if you get into the habit of regarding each segment as—

(1). A *centre, from which* voluntary motor, reflex motor, vaso-motor, and trophic influences are distributed *to;* and *to which* peripheral impressions, producing reflex movements and sensations, proceed *from*, a limited area of the body (*i.e.*, that portion of the body to which the pair of spinal nerves belonging to the segment is distributed.) (See figs. 2 and 13.)

(2). As a *conducting medium*, so to speak, through which the great tracts (motor, sensory, controlling, etc.) pass, which place the brain in connection with all the other segments situated below, or, as comparative anatomists say, posterior to it (*i.e.*, the segment we are at present dealing with). (See figs. 14 and 15.)

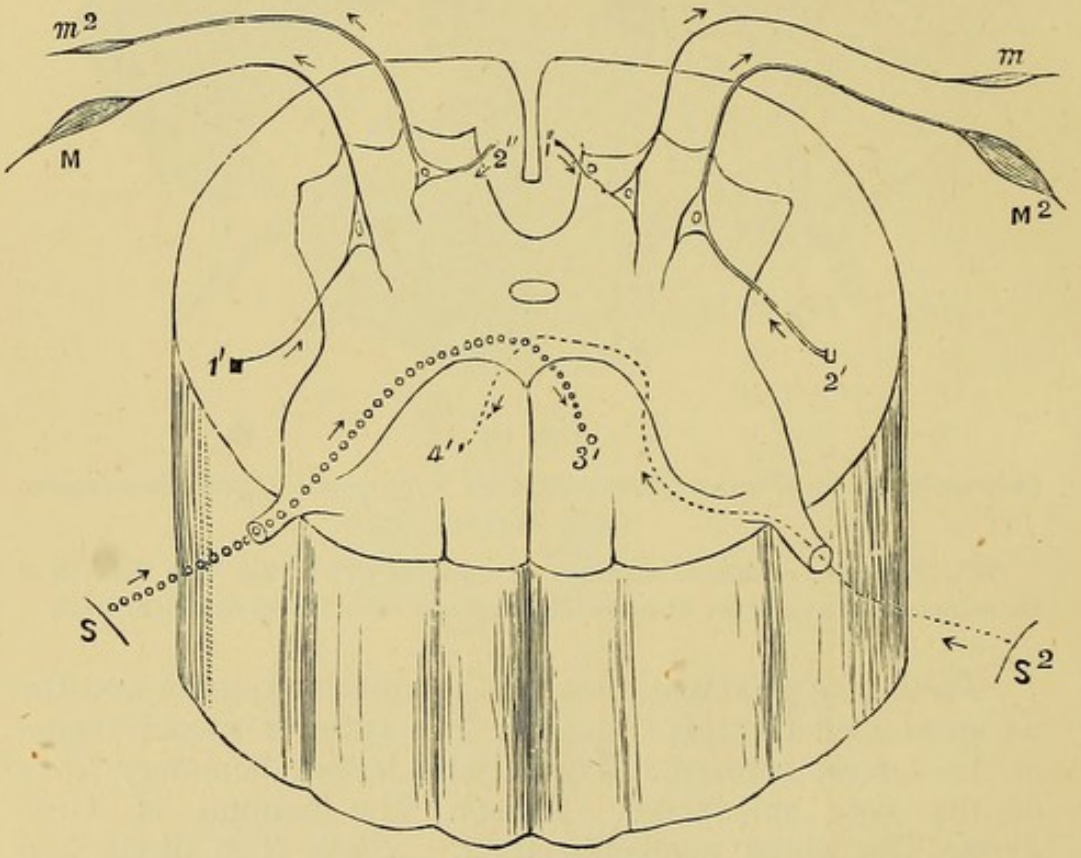


FIG. 13.

Diagrammatic representation of the Spinal Segment as a Spinal Centre.

1′ 2″ Motor fibres proceeding from the left crossed and direct pyramidal tracts, to supply the muscular area, (M,m²,) of the left lateral half of the segment. 2′ 1″ Motor fibres proceeding from the right crossed and direct pyramidal tracts to supply the muscular area, (M²,m,) of the right lateral half of the segment. S, Sensory fibre proceeding from the left sensitive area of the segment to join the main sensory tract (3′) in the right postero-internal column. S², Sensory fibre proceeding from the right sensitive area of the segment to join the main sensory tract, (4′) in the left postero-internal column.

The arrows indicate the direction of the nerve 'currents.'

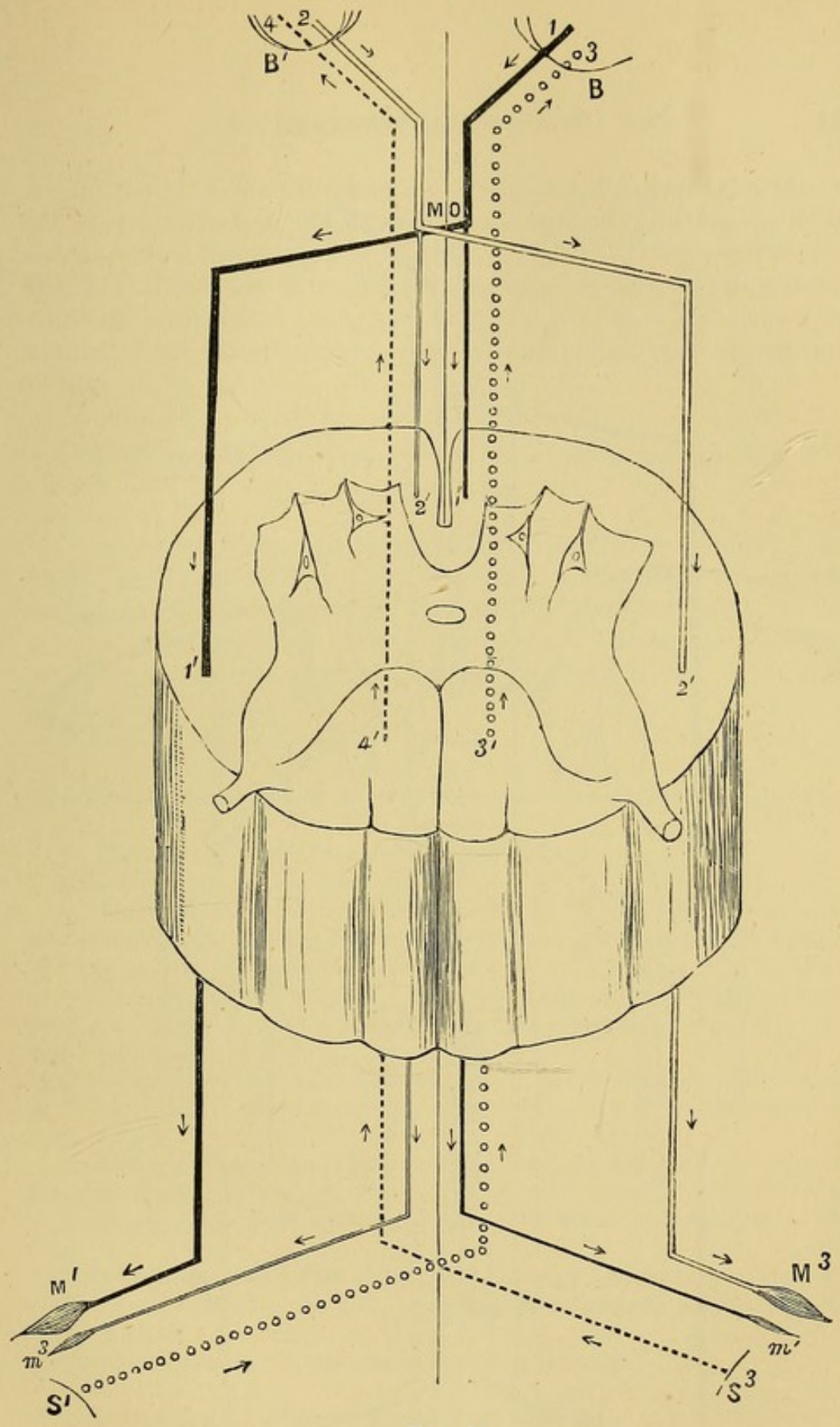


FIG. 14.

Diagrammatic representation of the Spinal Segment as a conducting medium, showing the passage of the main motor and sensory tracts through it. The muscular and sensory areas of the segment have been cut away.

B, Right, and B′, Left hemispheres of the brain. MO, Lower end of medulla oblongata. 1, Pyramidal (motor) tract from the right hemisphere passing through the segment in the lateral column of the opposite, and in the anterior column of the same side. 2, Pyramidal tract from the left hemisphere. 3′, 3 and 4′, 4, Main sensory tracts passing up to the brain in the postero-internal columns. The arrows indicate the direction of the nerve force.

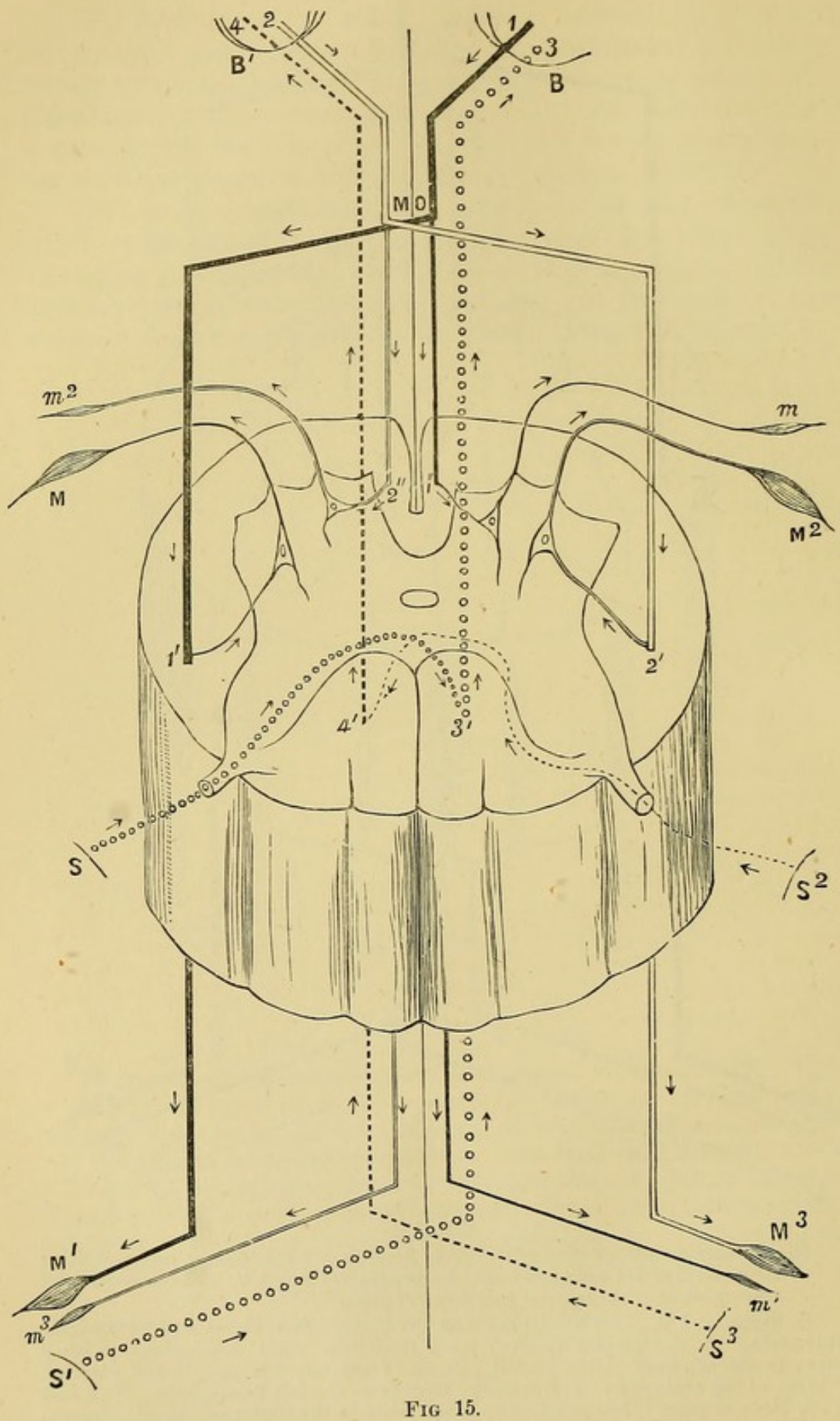


Fig 15.

Further, it is convenient to consider the motor, sensory, reflex, trophic, and vaso-motor functions of the segment in detail; and since each segment consists of two symmetrical lateral halves, it is only necessary to describe the functions of one lateral half, and the manner in which the two symmetrical lateral halves are (functionally) related to each other.

§ 5. THE MOTOR FUNCTIONS OF A SEGMENT OF THE SPINAL CORD.

The nerve fibres, which conduct voluntary motor impulses from the brain to the muscular area supplied by the segment we are dealing with, and to the muscles supplied by all the other segments which are situate below it, enter our segment by the crossed and direct pyramidal tracts. (See figs. 15 and 18.)

DESCRIPTION OF FIG. 15.

Diagrammatic representation of the Spinal Segment as a Spinal Centre and Conducting Medium.

B—Right, and B′—Left, hemispheres of the brain.

MO—Lower end of medulla oblongata.

1—Motor tract from the right hemisphere. At MO it divides. The larger sub-division decussates, passes down the lateral column of the opposite side of the cord, and supplies the muscular fibres M and M′ on the left side of the body. At 1′ the supply to M. is given off. The smaller subdivision does not decussate, but passes down the anterior column, and supplies the muscles *m* and *m′* on the same (right) side of the body.

2, The motor tract from the left hemisphere. It supplies the muscles M^2 and M^3 on the right side of the body, and the muscles m^2 and m^3 on the left side of the body.

S, S′, Sensitive areas on the left side of the body. 3′, 3 the main sensory tract from the left side of the body. It passes up the right (opposite) side of the cord in the (?) postero-internal column, and proceeds to the right hemisphere of the brain.

S^2, $S,^3$ Sensitive areas of the right side of the body. 4′, 4, The main sensory tract from the right side of the body, proceeding up the left side of the cord to the left hemisphere of the brain.

The arrows indicate the direction of the nerve 'currents.'

Note to Figs. 13, 14, and 15.

It is doubtful whether a total decussation of the sensory tract, such as is represented in these figures, occurs. Many physiologists believe that the decussation is incomplete, as is represented in fig. 22.

The course of the motor tract from the cerebrum to the spinal cord is as follows:—Motor impulses are discharged in obedience to the command of the will by the generating motor centres in the cerebral cortex (see fig. 16). The motor force thus liberated is conducted by the fibres of the pyramidal tract to the anterior cornua of the spinal cord. There are, of course, two (main) pyramidal tracts, one proceeding from each hemisphere. Following the course of either, say that arising from the right hemisphere, we find that after leaving the generating motor

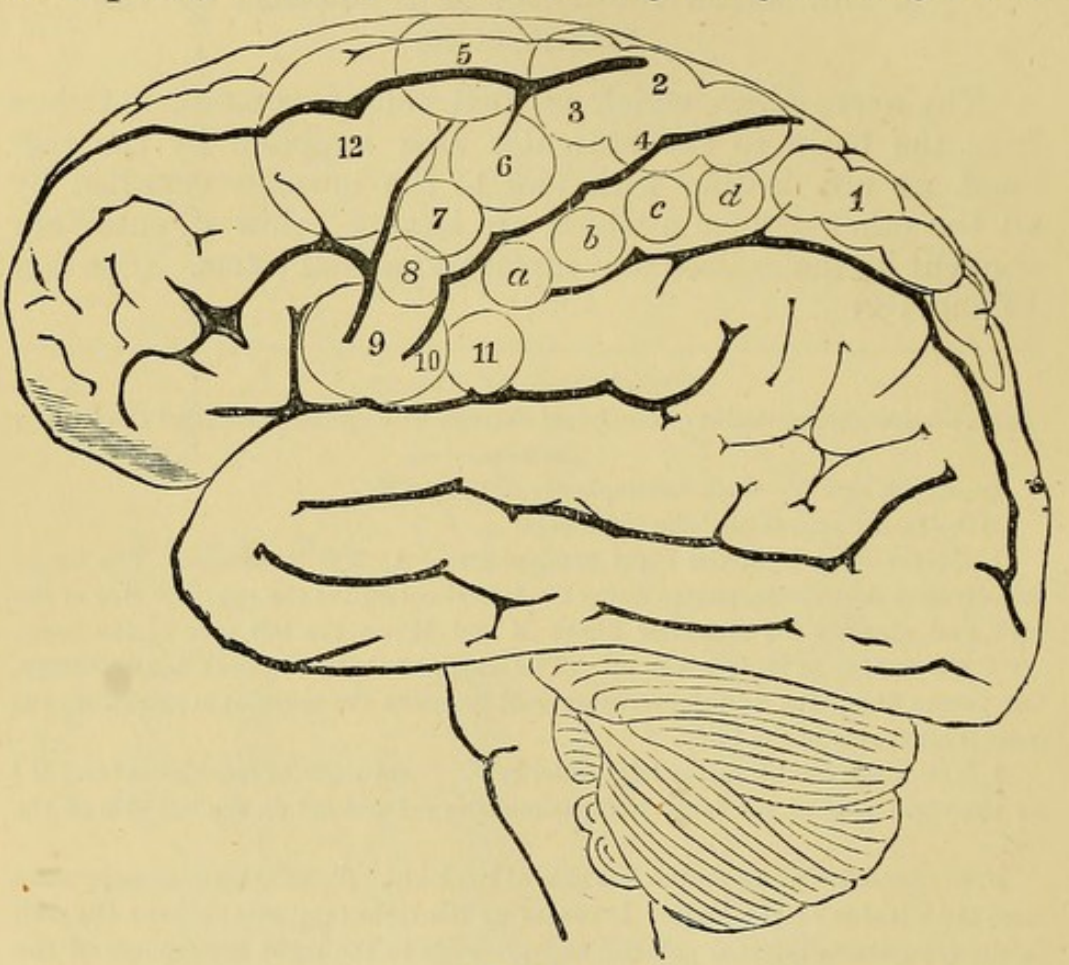


FIG. 16.

Lateral view of the human brain, showing the position of the motor centres. (*After Ferrier.*)

(1.) Centre for opposite leg and foot. (2, 3, 4.) Centres for movements of arms and legs, such as are concerned in climbing, swimming, etc. (5.) Centre for extension forwards of the arm and hand. (6.) Centre for supination of the hand and flexion of the forearm. (7 and 8.) Centres for elevators and depressors of mouth respectively. (9 and 10.) Centre for the movements of the lips and tongue in articulation. (11.) Centre for the platysma; retraction of the angle of the mouth. (12.) Centre for lateral movements of the head and eyes, with elevation of the eyelids and dilatation of the pupil. (*a*, *b*, *c*, *d*.) Centre for the movements of the hand and wrist.

centres it passes through the corona radiata, the anterior two-thirds of the posterior division of the internal capsule (see IK, fig. 17), crus cerebri, right side of the pons, and right side

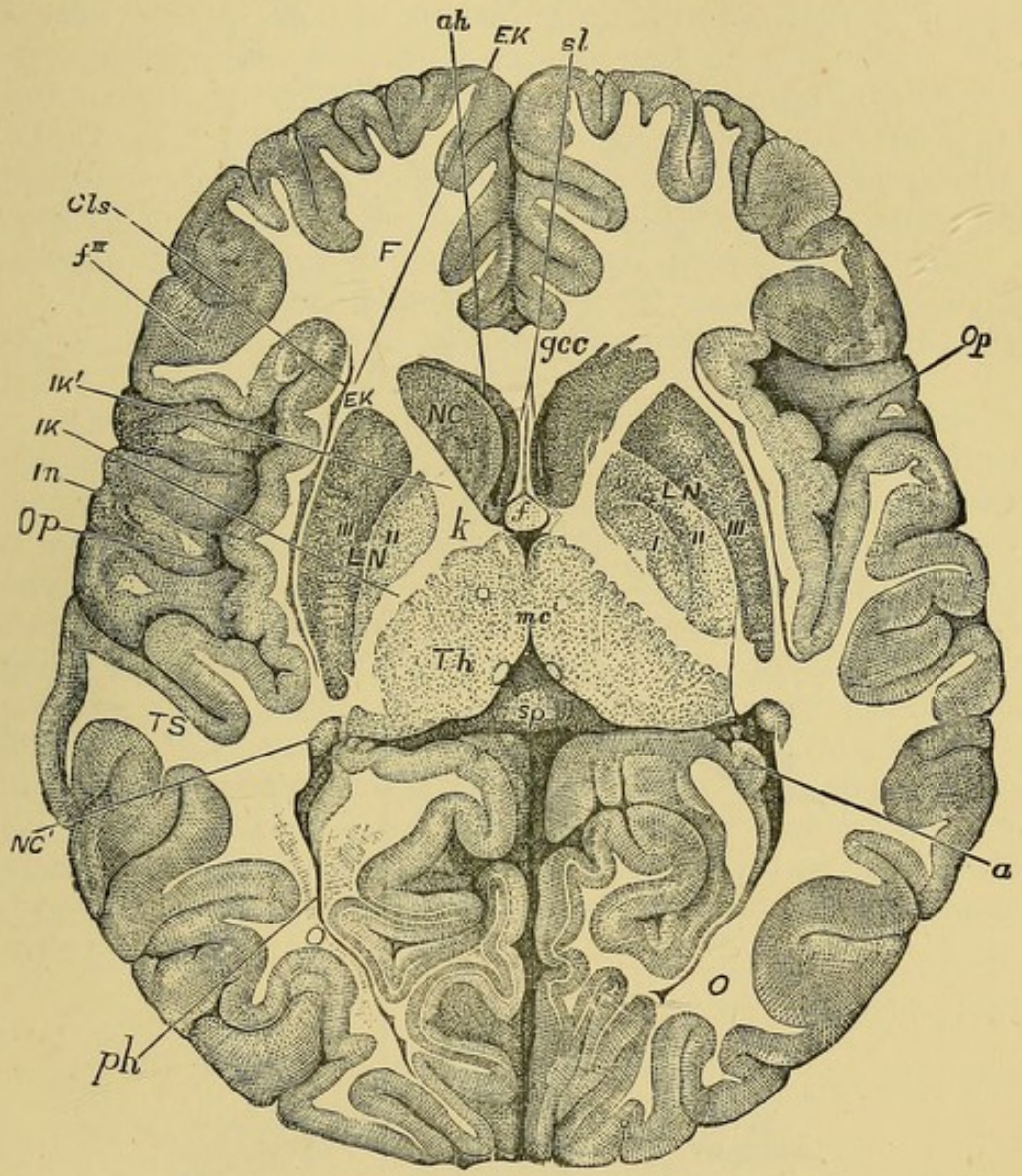


FIG. 17.

Horizontal section of the brain of a child nine months of age, the right side being at a somewhat lower level than the left half, showing the position of the internal capsule (IK, K, and IK'). (*From Flechsig.*)

F, Frontal, TS, Tempero-Sphenoidal, and O, Occipital lobes. Op, Operculum. In, Island of Reil. Cls, Claustrum. F''', Third frontal convolution. Th, Optic thalamus. NC, Caudate nucleus. NC', Tail of caudate nucleus. LN, Lenticular nucleus. I, II, III, First, second, and third divisions of the lenticular nucleus. EK, External capsule. IK, Posterior division, IK', Anterior division, and K, Knee of the internal capsule. ah, ph, Anterior and posterior horns respectively of the lateral ventricles. gcc, Knee of the corpus callosum. Sp, Splenium. mc, Middle commissure. f, Fornix. Sl, Septum lucidum. a, Cornu ammonis.

of the medulla. At the lower end of the right side of the medulla it divides into two parts (see figs. 15 and 18)—

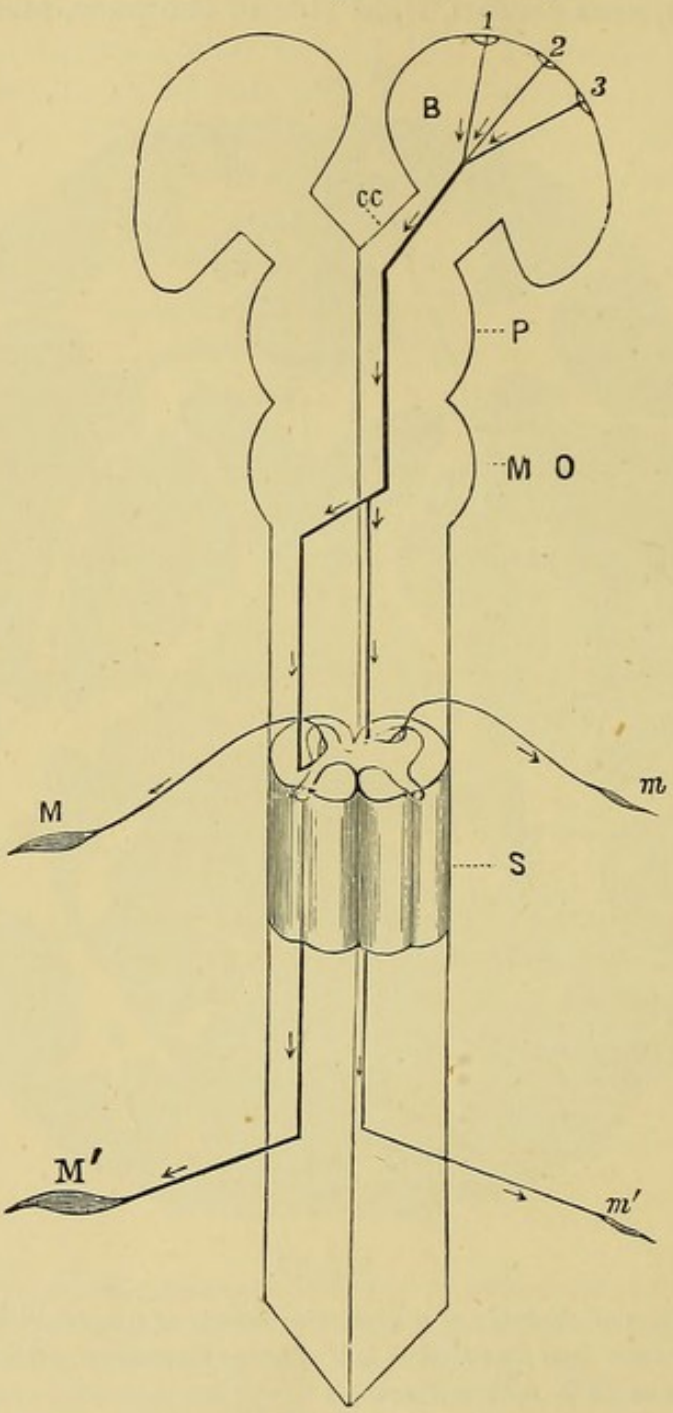


FIG. 18.

Diagrammatic representation of the course of the pyramidal tract arising from the motor centres in the right hemisphere of the brain.

B, Right hemisphere of brain. 1 2 3, Generating motor centres in the right hemisphere. CC, Crus cerebri. P, Pons varolii. MO Medulla oblongata. S, Segment of spinal cord. At the lower end of the medulla the motor tract divides. The larger subdivision decussates and passes down the opposite side of the cord in the lateral column; it is distributed to the muscles M,M′ on the left side of the body. The smaller subdivision passes down in the anterior column of the same side of the cord, and is distributed to muscles m, m′ on the right side of the body.

the larger division (which, according to Flechsig, usually contains from 91 to 97 per cent. of the whole) crosses over to the left side of the spinal cord, and proceeds down the left side of the cord in the lateral column, constituting the crossed pyramidal tract of the left lateral half of the cord. The smaller sub-division (which, according to Flechsig, usually contains from 3 to 9 per cent. of the whole) does not decussate, but passes down the same (right) side of the cord in the anterior column, constituting the direct pyramidal tract (or column of Türck), the position of which has been already described.

Voluntary motor impulses, then, are chiefly carried to our segment by fibres which pass to it from the crossed pyramidal tracts in the lateral columns.

The fibres carrying motor impulses to inferior segments simply pass through our segment. (See fig. 14.) The fibres destined for our segment itself leave the main motor tracts (direct and crossed pyramidal tracts) as represented in fig. 15; enter the grey matter; and pass to the anterior cornu. Some of them probably become connected with the multipolar nerve cells. Others probably pass directly into the anterior nerve roots. After entering the grey matter, the fibres of the pyramidal tract are supposed to divide and subdivide, and finally to form connections with the delicate fibrils of Gerlach's nerve network, in which, as I have previously stated, the branches of the multipolar nerve cells (except the axis cylinder process) are supposed to terminate, as is represented in figure 19.

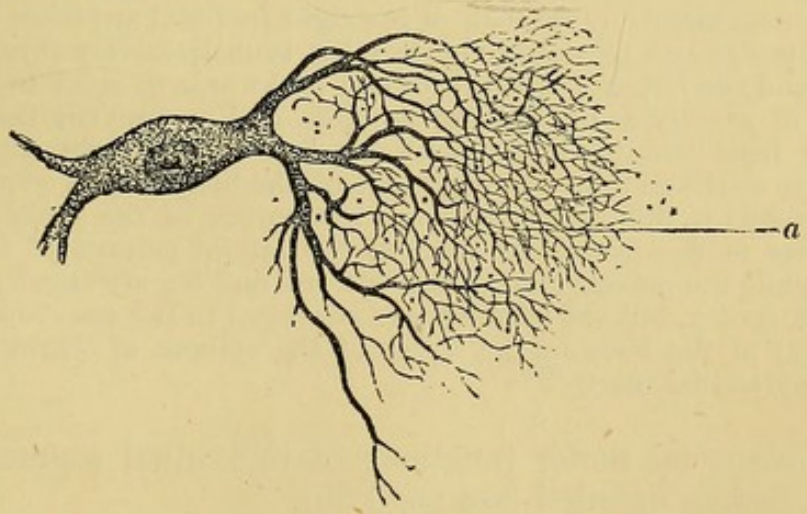


FIG. 19.
Supposed union of a nerve fibre with a nerve cell.
The processes of the cell subdivide into a minute network (Gerlach's nerve network), in which the fibre, *a*, also looses itself.

Voluntary motor impulses leave the multipolar nerve cells of the anterior cornu *per* their axis cylinder processes, and pass out of the cord through the anterior roots. The anterior root-fibres pass through the anterior column in several bundles, as is shown in fig. 20.

The passage of the motor fibres from the crossed pyramidal tract to the anterior nerve root is diagrammatically represented in fig. 15. The exact manner in which the fibres from the direct pyramidal tract pass into the anterior root is not yet definitely decided. Some authorities think that they decussate in the cord and pass out through the anterior nerve roots of the opposite side. According to this view there is a total decussation of the main pyramidal tract proceeding from each hemisphere—the greater number of the fibres of which it is composed passing to the opposite side of the cord at the lower end of the medulla, and constituting the crossed pyramidal tract; the smaller number (which pass down on the same side of the cord as the *direct* pyramidal tract) decussating in the cord itself, through the anterior white commissure.

Others suppose that the fibres of the direct pyramidal tract pass through the anterior horn of grey matter to the anterior nerve roots on the same side, as shown in fig. 15. According to this theory, the main pyramidal tract from *one* hemisphere conducts motor impulses to *both* sides of the body. This view is supported by the great authority of Dr Hughlings Jackson. His, very ingenious explanation of the mechanism, as quoted by Dr Ross,[1] is as follows:—"The crossed and direct connection which this (the main pyramidal) tract forms between the cortex of the brain, and the grey anterior horn is rendered necessary by the fact that every movement of one side of the body alters the centre of gravity, and necessitates a new adjustment of the opposite side. I obtained this idea (says Dr Ross) in a conversation with Dr Hughlings Jackson, and he illustrated his meaning by showing that when a man stands on the ball of the right foot and stretches his right arm upwards and forwards, the left leg is instinctively thrust backwards and the left arm downwards and backwards in order to keep the centre of gravity as far back as possible, and so prevent the line of gravity from passing in front of the ball of the right foot. The muscular contraction of the right side of the body may be supposed to be regulated in this action from the left cortex of the brain through the fibres of the pyramidal tract of the lateral column of the right side; while the movements of the left arm and leg are regulated from the left cortex, but the impulses are conveyed to the same side of the cord and of the body by the fibres of the column of Türck (*i.e.*, the direct pyramidal tract)."

The special motor functions of individual segments will be afterwards detailed (see page 86).

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[1] *Diseases of the Nervous System*, vol. ii., page 83.

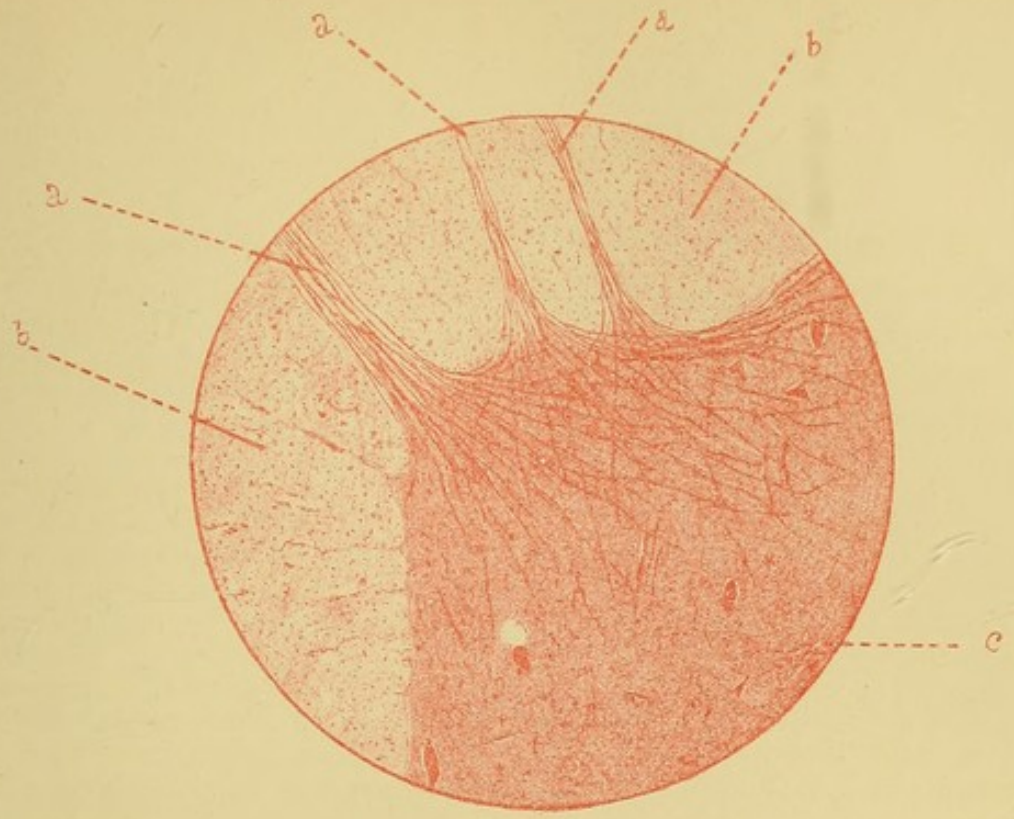


FIG. 20.

Transverse section through the Spinal Cord, showing the passage of the anterior root-bundles through the anterior column. (Carmine and dammar), magnified about 50 diameters.

a, a, a, Anterior root-bundles: b, b, anterior column; c, anterior horn of grey matter.

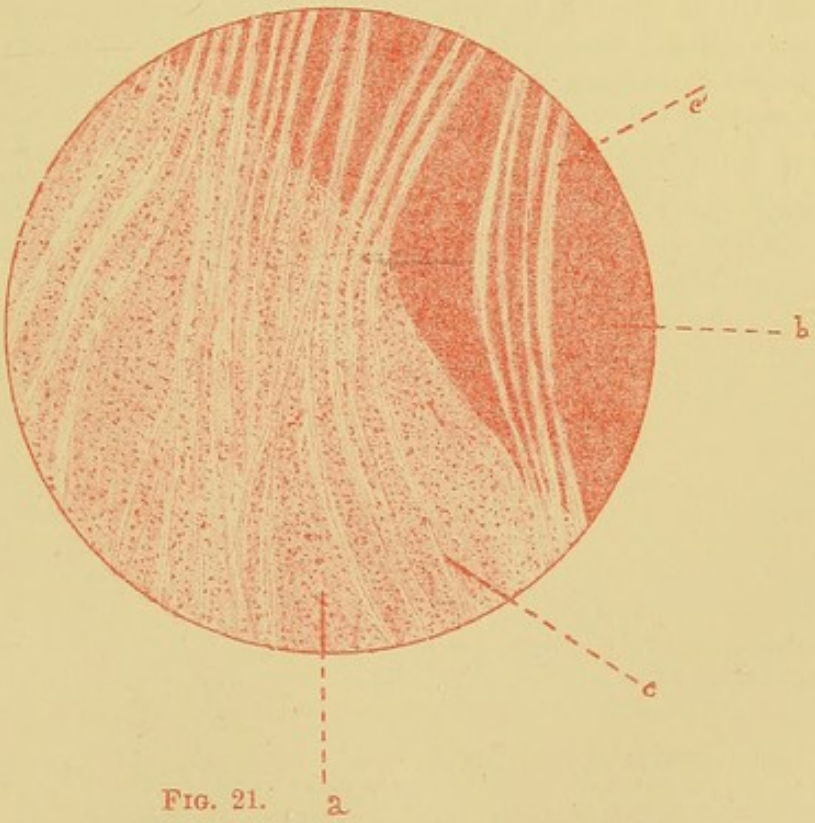


FIG. 21.

Transverse section through the Spinal Cord, showing the passage of the posterior root-bundles through the postero-external column. (Carmine and dammar), magnified about 50 diameters.

a, Postero-external column; b, posterior horn of grey matter; c, posterior root-bundle passing through the postero-external column; c′, posterior root-bundle after it has entered the grey matter.

B.B.delt. ad. natm.

McLagan & Cumming, Lith. Edin.

§ 6. THE SENSORY FUNCTIONS OF THE SPINAL SEGMENT.

The fibres which conduct sensory impressions from all inferior segments (*i.e.*, from all parts of the body below our segment) enter and pass through our segment either in the lateral or in the posterior columns. (See figs. 14, 15, and 22.) The exact position in the transverse section which these fibres occupy has not yet been definitely ascertained,[1] but it is generally believed that each lateral half contains the greater number, if not all, of the sensory fibres proceeding from all inferior half segments on the opposite side of the body; that is to say, that there is a more or less complete decussation of the sensory fibres at (or about) *their* point of entrance into the cord, as we have already seen that there is a more or less complete decussation of the motor fibres at *their* point of entrance into the cord, *i.e.*, at the top of the cord. (See fig. 23.)

The fibres which conduct sensory impressions from the body area corresponding to our segment itself, enter our segment through its posterior nerve roots. The posterior nerve root joins the cord as a single bundle of fibres (see fig. 3). After entering the cord its component fibres diverge. Some of them pass directly into the posterior horn of grey matter, as is shown in figs. 3 and 25. Others traverse the postero-external column, and then pass into the grey matter of the posterior horn (see fig. 21). In some parts of the cord the fibres of the posterior roots pass obliquely through the postero-external column; they consequently enter the grey matter at a slightly different level from their point of entrance into the cord. Having passed into the posterior horn of grey matter, they, then, in great part or *in toto*, decussate, pass through the

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[1] From the experiments of Woroschiloff and Ludwig it would appear that sensory impulses in the rabbit are conducted through the lateral columns. These observers think that each lateral column contains sensory fibres for both legs.

In support of these experiments on the rabbit, Gowers brings forward a case of crushed cord in man, in which he found secondary ascending degeneration of the postero-internal columns, and a symmetrical patch of the ascending degeneration in that part of the lateral column which lies in front of the crossed pyramidal tract. He thinks that this fact, taken in conjunction with the experiments on animals, favours the view that some sensation is conducted in this region in man. —*Diagnosis of Diseases of the Spinal Cord*, page 14.

It may, perhaps, be not uninteresting in this connection, to direct attention to figure 34, which represents the section through the cervical region of the cord in a case of disseminated myelitis. The usual secondary ascending degeneration of the postero-internal columns is well shown, and there is also a patch of degeneration in the lateral column. I am not prepared to say whether the patch in the laternal column was primary or a secondary ascending lesion.

commissure of the cord, and join the main sensory tract, which passes up the opposite side of the cord to the brain. (See

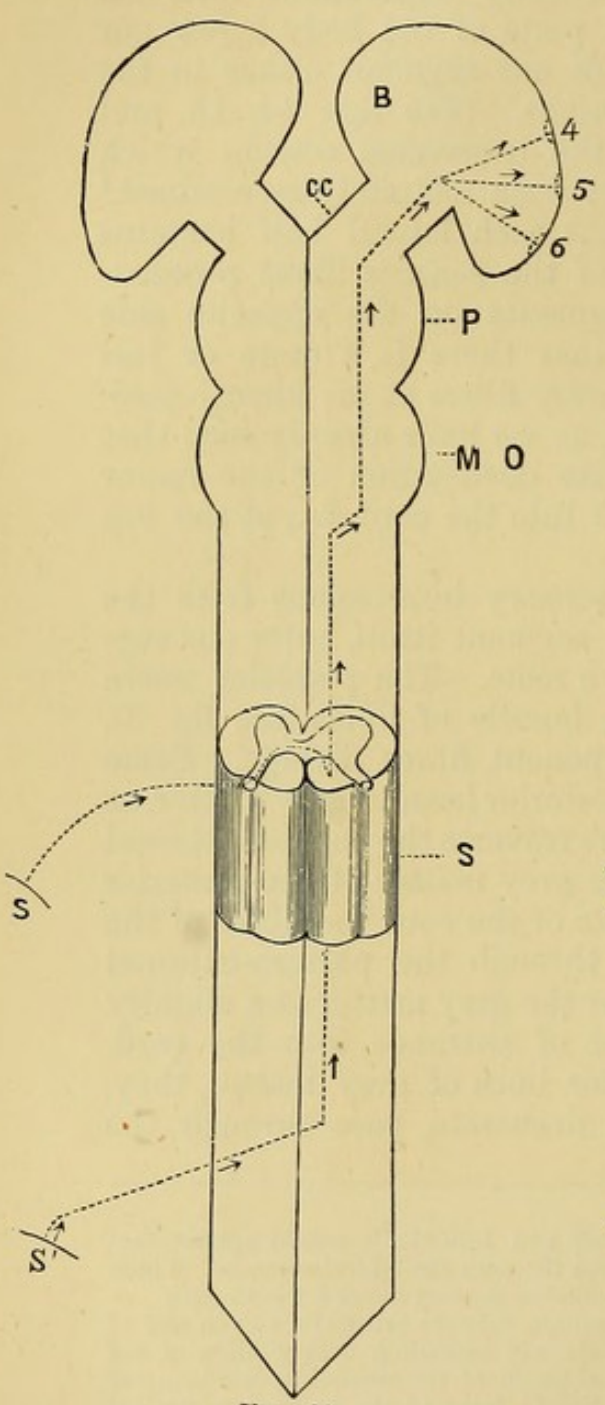


FIG. 22.

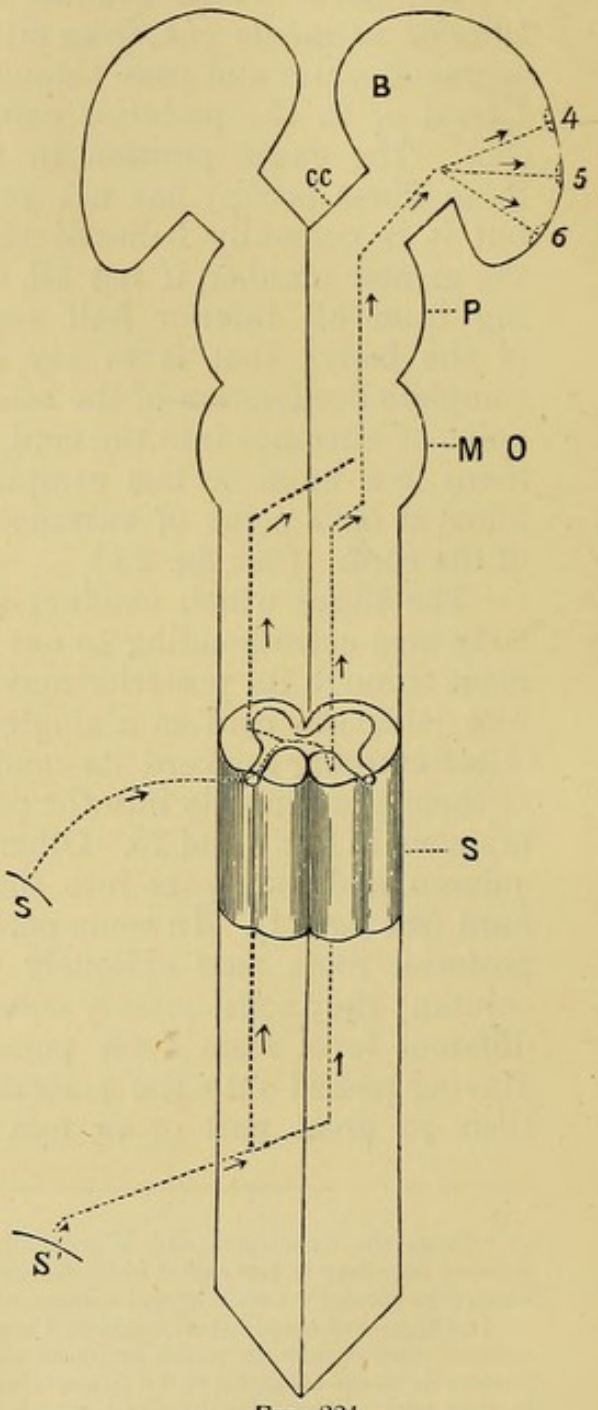


FIG. 22'.

Diagrams of the course of the Sensory Tract proceeding from the left side of the Body.

Fig. 22.—B, The right hemisphere of the brain. 4 5 6, Perceptive (sensitive) centres in the right hemisphere. S S.', Sensitive areas (skin, muscles, tendon, etc.) of the left side of the body from which sensory impressions proceed in the direction indicated by the arrows.

Fig. 22'.—In this figure the sensory decussation in the cord is represented as partial only. Those fibres, which do not pass to the opposite side of the cord, are made to ascend in the lateral column, and to decussate at the lower end of the medulla.

figs. 13, 22, and 23.) The decussation of the sensory fibres in the cord is not accepted by all authorities; Meynert, for example, describes a sensory decussation in the medulla; while Landois, in his scheme of the nerve centres, makes the whole of the sensory tract pass up the same side of the cord and decussate in the medulla. (See his *Physiologie des Menschen*, page 717.) A partial decussation is represented in fig. 22′.

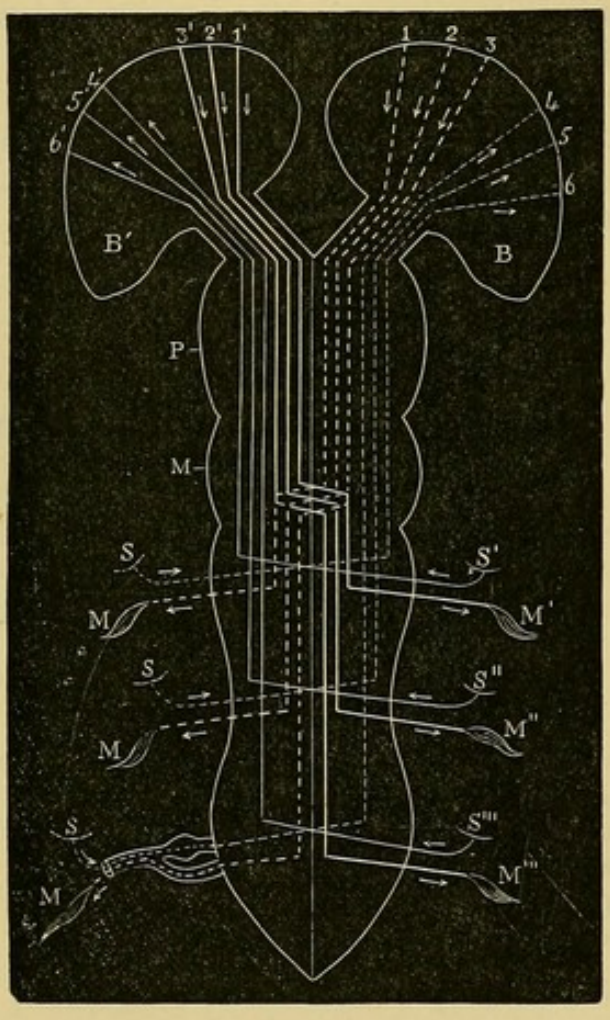


FIG. 23.

Diagrammatic representation of the course of the sensory and motor nerve tracts for both sides of the body.

The *thick* lines=Motor fibres. The *fine* lines=Sensory fibres. The *continuous* lines=Motor (thick) and sensory (fine) fibres, supplying the left side of the body. The *dotted* lines=Motor (thick) and sensory (fine) fibres, supplying the right side of the body. B=Left side of brain. B′=Right side of brain. 1 2 3=Motor centres for *right* arm, trunk, and leg muscles respectively, in the *left* cerebral cortex. 1′ 2′ 3′=Motor centres for *left* arm, trunk, and leg muscles respectively, in the *right* cerebral cortex. P=The pons varolii M=The medulla oblongata. S=Sensory nerves from skin, etc., of *right* upper extremity. S′=Sensory nerves from skin, etc., of *left* upper extremity. S″=Sensory nerves from skin, etc., of *left* side of trunk. S‴=Sensory nerves from skin, etc., of *left* lower extremity. M=Muscles of *right* upper extremity. M′=Muscles of *left* upper extremity. M″=Muscles of *left* side of trunk. M‴=Muscles of *left* lower extremity.

After leaving the cord, the main sensory tract passes through the medulla, pons, crus cerebri, and posterior third of the posterior division of the internal capsule (see figs. 17 and 24) in order to reach the sensory perceptive centres, which are situated in the posterior parts of the hemispheres. There is great difficulty in establishing the exact position of the tactile centre. Ferrier, as the result of numerous experiments, places it in the hippocampus major and uncinate convolution, a view which is confirmed by clinical observation, for many cases have been reported in which rupture or disorganisation of that part of the internal capsule, which lies external to the optic thalamus, has caused hemianæsthesia of the opposite side of the body.[1]

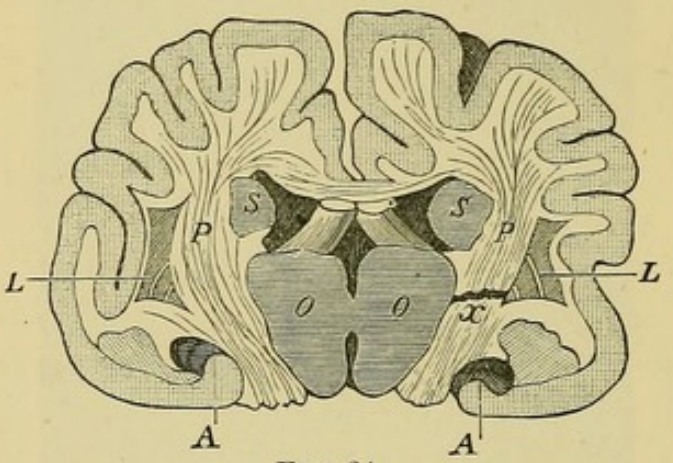


FIG. 24.

Vertical transverse section through the brain of the dog on a level with the corpora mamillaria. (*After Carville and Duret.*)

OO. The optic thalami. SS. The nuclei caudati of the corpora striata on each side. LL. The lenticular nuclei of the corpora striata. PP. The internal capsule, or peduncular expansion. AA. The hippocampi. X. Section of the posterior part of the peduncular expansion, causing *hemianæsthesia.*

The special sensory functions of individual segments of the cord will be afterwards described (see page 137).

§ 7. THE REFLEX FUNCTIONS OF THE SPINAL SEGMENT.

Every half segment of the spinal cord with its sensory and motor nerve roots is, in theory and probably also in fact, a perfect reflex arc. Peripheral irritation applied to the portion of skin, muscle, tendon, etc., to which the fibres of its posterior nerve root are distributed, may result in the production of

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[1] *The Functions of the Brain*, page 175.

(reflex) movements in the muscular area to which the fibres of its anterior nerve root proceed.

As a matter of fact, peripheral irritation applied to the body area of one segment often produces (reflex) movements in the muscular areas of other segments as well as in its own muscular area. This is owing to the free inter-communication between the nerve fibrils in the grey matter of the cord.

The multipolar nerve cells of the anterior cornua probably constitute the centre of this reflex arc. The superficial reflexes are probably conducted by those fibres of the posterior root which pass directly into the posterior horn of grey matter; while deep reflex impulses are probably carried by those fibres which pass through the postero-external column, as is shown in fig. 25.

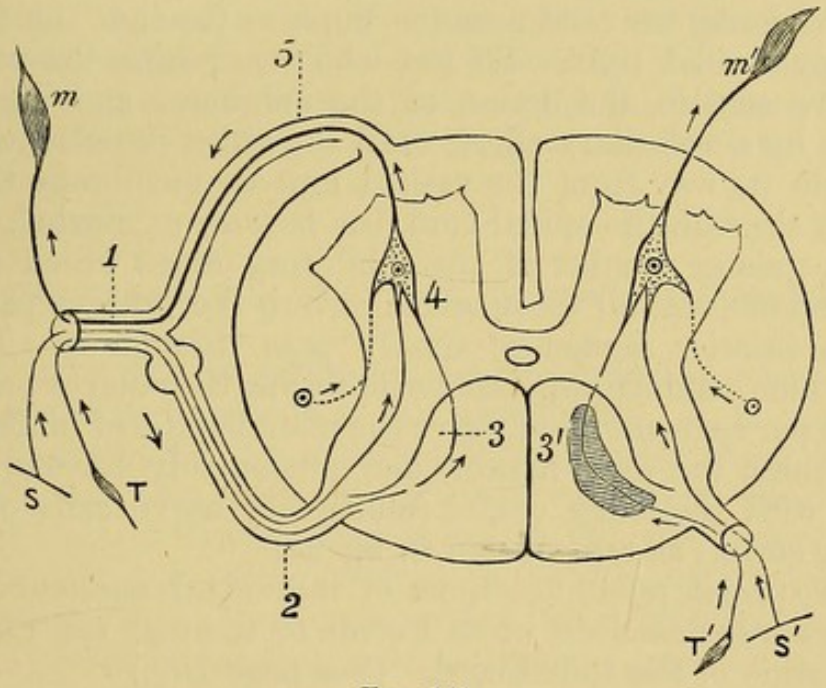


FIG. 141.

Diagrammatic representation of the reflex functions of the Spinal Segment.

The left half segment is normal. The right half represents the early stage of locomotor ataxia, the position of the lesion being shaded dark.

S, Skin from which sensory fibres pass through the common sensory motor nerve trunk, posterior root, and posterior horn of grey matter to the reflex centre (4). T, Tendon, muscle, and fascia, from which the sensory fibres pass which conduct the deep reflex movements. These fibres traverse the postero-external column.

m, Muscle supplied by the anterior root of the left half segment. The dotted line passing from the crossed pyramidal tract in the lateral column represents the inhibitory fibre.

The arrows show the course of the reflex impulse.

On the right side the deep reflex movements are seen to be arrested by a lesion in the postero-external column.

Clinical and pathological observations go to show that reflex movement may be inhibited by an effort of the will. The fibres which conduct inhibitory reflex impulses probably pass down the cord in the crossed pyramidal tract.

Whether there are distinct fibres set apart for the conduction of inhibitory impulses is doubtful. It is, I think, quite possible that the same fibres which conduct voluntary motor impulses may conduct inhibitory impulses too. When we inhibit or prevent a reflex movement; when, for example, we resist the reflex jerking of the leg, which, under ordinary circumstances, follows tickling of the sole, we do so by placing the muscles of the limb—probably the antagonistic muscles to those concerned in the production of the reflex movement—in a condition of tonic, but not obvious (naked eye) contraction. In other words, we send a motor impulse through the fibres of the pyramidal tract. So, too, when we inhibit the vesical reflex we sustain the action of the sphincter and urethral muscles by a voluntary effort, until the reflex impulse, which has made its way from the vesical mucous membrane to the detrusor through the spinal cord, has become exhausted.

The greater number of the inhibitory fibres which enter our segment (viz., all those which govern the reflexes passing through inferior segments) simply pass through it. Those fibres which conduct impressions inhibiting the reflexes passing through our segment itself, leave the main bundle of inhibitory fibres, enter the grey matter, and presumably become connected with the reflex centre (multipolar nerve cells of the anterior cornu) as represented in fig. 25.

The special reflex functions of individual segments will be afterwards described when I come to treat of the clinical examination of the spinal cord. (See page 130.)

§ 8 THE TROPHIC FUNCTIONS OF THE SPINAL SEGMENT.

The multipolar nerve cells of the anterior cornu exert a trophic influence upon the motor nerve fibres passing out of the segment, and upon the muscular fibres to which they are distributed. When the multipolar nerve cells are acutely destroyed, the axis cylinder processes arising from them, the fibres of the anterior roots, and the muscular fibres to which they are distributed, undergo rapid atrophy.

But, in addition to the trophic influence of the multipolar nerve cells of the anterior cornu, the grey matter seems to

exert a trophic influence upon the portion of skin from which the fibres of its posterior nerve root proceed. After certain injuries of the central grey substance, for example, acute bed sores have been observed.

Further, it must be remembered that the fibres of the pyramidal tracts (direct and crossed) have their trophic centre in the motor area of the cerebral cortex. Lesions, therefore, in our segment which sever this connection will be followed by descending degeneration of the pyramidal tracts in all the segments which are situated below it. (See p. 34.)

Again, the fibres of the posterior nerve roots and of their upward prolongations have their trophic centre in the posterior root ganglia. Lesions of our segment, therefore, which sever this connection will be followed by secondary ascending degeneration; and as a matter of fact we know that lesions of the postero-external column are followed by secondary ascending degeneration of the postero-internal column, and of the direct cerebellar tract of Flechsig, above the level of the lesion.[1]

§ 9. THE VASO-MOTOR FUNCTIONS OF THE SPINAL SEGMENT.

We have not as yet much definite knowledge regarding the vaso-motor functions of the spinal cord. It is probable that *vaso-constrictor* and *vaso-dilator* fibres pass through each segment, and that there are distinct vaso-motor centres in some, at least, of the segments of which the cord is composed. The exact position of the conductors (vaso-motor dilators and constrictors) in the transverse section is not definitely known, but it is probable that they pass down some part of the lateral columns.

§ 10. THE FUNCTIONAL CONNECTION OF THE TWO LATERAL HALVES OF A SPINAL SEGMENT.

Each half segment is, to a large extent, functionally independent of its fellow on the opposite side. In the segments

[1] This is to my mind a strong argument in favour of the postero-internal columns being the part of the transverse section through which sensory conductors pass *en route* to the cerebrum.

composing the cervical region this independent action is most highly developed, but even there the two lateral halves are in intimate connection.

Numerous illustrations of their relationship might be given, but the following will suffice: In case of hemiplegia voluntary movements in the sound (non-paralysed) hand are sometimes attended by corresponding movements in the paralysed one. External irritation applied to one limb not unfrequently produces reflex muscular movements on both sides of the body. If one hand be plunged into cold water an alteration in the temperature of the other hand occurs, etc., etc.

In the segments composing the dorsal region of the cord, this independent action, as regards the motor functions at all events, is much less developed. The muscles supplied by the half segments of one side (in this the dorsal region) are in the habit of acting in connection with the muscles supplied by the half segments of the other. And to provide for this associated action there is probably, as Dr Broadbent has so well theorised, a free communication between their respective nerve nuclei, the multipolar nerve cells of the anterior cornua.

§ 11. THE CONNECTIONS OF DIFFERENT SEGMENTS WITH ONE ANOTHER. *Spinal co-ordination of movement.*

Adjacent segments are, of course, structurally continuous. But, in addition, different segments are structurally and functionally connected with and related to one another. Such a combination is required for the co-ordination and grouping of motor impulses. The co-ordination of muscular movement, especially of the muscular movements of the lower extremities, is to a large extent reflex, *i.e.*, under the direction of peripheral impressions proceeding from the skin, muscles, tendons, joints, etc. And we may theoretically suppose that the peripheral impressions of this description, which enter by one posterior root, may require to be distributed to several adjacent segments. This distribution is probably, in part at least, provided for by the commissural fibres which pass (?) from the posterior horn of one segment to the posterior horn of other segments, through the postero-external columns. For the same reason, we may theoretically suppose that the multipolar nerve cells of one segment are connected with the multipolar nerve cells of other segments

—that there are are, in short, commissural fibres which pass from the anterior horn of one segment to the anterior horns of other segments, possibly through the anterior columns.

But in addition to these, which we may term the more highly specialised commissural connections, every segment is connected with every other segment by means of the vast network of delicate nerve fibrils which constitute such a large and important part of the grey matter.

Motor impulses and sensory impressions, or, more correctly speaking, the nerve vibrations which produce sensory impressions and motor impulses (or nerve currents, as they are less accurately, though very expressively called) follow the path of the least resistance; and the more highly specialised the sensation or movement, the more sharply defined is the path through which that impulse travels (*i.e.*, the greater is the difference in the degree of resistance between the path through which it must pass to produce the desired result, and all other paths.) The nerve currents which produce the more highly specialised movements have little or no tendency to diffuse themselves, and in passing through the grey matter of their respective segments they make use of very definite channels, while the nerve currents, which produce the least specialised sensations and movements, have a considerable tendency to spread from segment to segment, and so through the whole cord, *per* this delicate nerve network in the grey matter. Conversely, in cases of disease where the accustomed path (the path of least resistance) is 'blocked' by a lesion, the more sharply defined that path, the greater difficulty will the nerve vibration have in finding its way by any other. Hence it follows, that the more highly specialised sensations and movements are most easily deranged, and, when deranged, are with the greatest difficulty restored.

Where the accustomed path is 'blocked,' the nerve current will always tend to pass by some side or collateral channel. In some cases the position of the 'block' prevents the possibility of such passage. Section of a sensory-motor nerve, or of its posterior root (the position of such lesions is marked in fig. 25 by the numbers 1 and 2), will, of necessity, prevent any reflex movement being excited by irritation of its sensitive body area; but where the 'block' affects the reflex arc after the posterior root has entered the grey matter, say, for example, where the reflex centre (4, fig. 25) is destroyed, though the

reflex impulse cannot pass out, *per* its accustomed channel, it may make its way through the delicate nerve network in the grey matter to other and more distant segments, and so pass out to other and more distant muscles.

A very striking illustration of this fact came under my notice a short time ago. The case was one of *polio-myelitis anterior acuta* in an adult. A woman was suddenly attacked with paralysis of all four extremities. The attack was ushered in with fever. The paralysis was followed by rapid atrophy. When I saw the case, some months after the onset of the disease, the paralysis was almost complete in the lower extremities, and very considerable in the upper. The paralysed muscles were atrophied to an extreme degree. Sensibility was quite perfect, indeed abnormally acute. There was not, and never had been, any affection of the bladder or rectum. *Tickling the soles produced no reflex movement in the lower extremities*, for the reflex impulse could not pass out owing to the lesion in the reflex centre, *but was followed by strong contraction of the muscles supplied by the upper segments of the cord*, indeed, the jerking movement which resulted was sufficiently powerful to produce a violent start of the whole body.

CHAPTER II.

THE PATHOLOGY OF THE SPINAL SEGMENT—THE ALTERATIONS IN FUNCTION WHICH RESULT FROM LESIONS OF ITS DIFFERENT PARTS.

§ 12. PASSING from normal to diseased function, I will now describe some of the more important points with regard to the pathology of our spinal segment. I must beg of you, however, to remember that, in actual disease, the morbid process is very seldom, even at its commencement, limited to a single segment. In the majority of cases met with in practice, several segments, or even long portions of the cord, are involved. The somewhat artificial arrangement which I propose to follow is, however, useful—it greatly simplifies the subject,—and, with the caution I have just given, may be safely adopted for teaching purposes.

Some of the morbid conditions affecting the spinal cord (*i.e.*, our segment) are *primarily nervous;* others originate in the surrounding parts (*i.e.*, bones and membranes). In the latter case, the cord lesion is *secondary.* Now, in describing the pathology of the spinal segment and the symptoms which result from derangement of its different parts, it is convenient to separate these two groups of lesions, which may be termed *intra-medullary* and *extra-medullary* respectively.

INTRA-MEDULLARY LESIONS.

The affections of the cord which are primarily nervous are either acute or chronic. In some the diseased process is strictly limited to definite physiological tracts. These affections are called *system diseases.* In others the morbid process

has no such physiological limitation, but involves at haphazard, as it were, a greater or smaller portion of the transverse section. To these lesions the term *indiscriminate* may be applied. In a third group of cases, as we shall afterwards see, these two forms of lesions are combined.

§ 13. THE SYSTEM DISEASES OF THE SPINAL CORD.

The great characteristic of a *system disease* is, as I have already mentioned, the sharp limitation of the lesion to a definite physiological tract. The lesion usually extends through several segments, and is generally bilateral and symmetrical.

The great vertical extent is due in some cases, as in *polio-myelitis anterior acuta,* to the circumstance that the same physiological areas in many different segments are affected *simultaneously.* In others it results from the extension of the morbid process from segment to segment along the fibres of the affected tract. This extension (always?) takes place in the direction of the physiological activity of the affected fibres, and is very generally of the nature of a 'secondary degeneration.'

The system lesions of the spinal cord are either *primary* (*i.e.*, arising independently of any previous lesion), or *secondary* (*i.e.*, resulting from some previous morbid condition). To the secondary system lesions the term '*secondary degenerations*' is usually applied.

§ 14. *Primary system lesions.*—The physiological tracts in the transverse section, which are liable to be affected by primary system lesions are:—

1. The region of the anterior cornu (1, 1′ fig. 26).
2. The region of the crossed pyramidal tract (3, 3′ fig. 26).
3. The region of the postero-external column (2, 2′ fig. 26).

Professor Pierret of Lyons has described a case in which the postero-internal columns were affected by what seemed to be a primary system lesion; but so far as I am aware no other case has yet been recorded.[1]

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[1] *Archives de Physiologie*, 1873, p. 74.

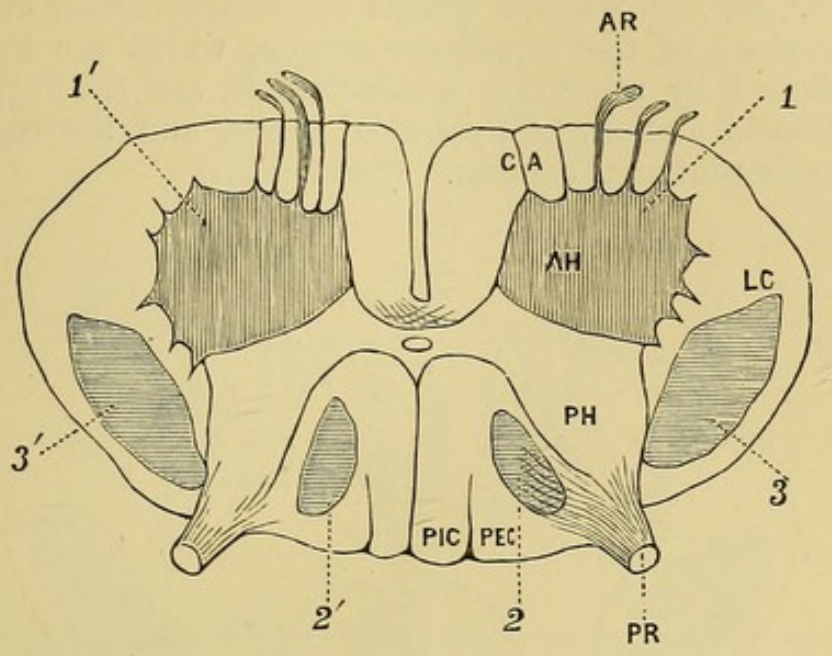


FIG. 26.

Diagrammatic representation of the primary system lesions of the spinal cord.

1, 1′ Region of the anterior horn. 2, 2′ Region of the postero-external column. 3, 3′ Region of the crossed pyramidal tract.

In the majority of cases one system only is involved, but in some cases two physiological tracts in the same half segment may be affected. In combined cases the lesion is, at the outset, limited to one tract, and subsequently spreads to another. The mode of extension is usually along the fibres which connect the two systems functionally, and is (always ?) in the direction of the physiological activity of the connection. Thus, a sclerosis of the postero-external column may extend to the anterior cornu, presumably along the fibres, which pass from the postero-external column to the posterior cornu, and thence to the anterior horn; or a sclerosis of the crossed pyramidal tract may extend to the anterior cornu, presumably along the fibres which leave the main motor tract and pass into the grey matter.

Occasionally it would appear that the morbid process is propagated from one physiological tract to another by direct extension (continuity of tissue), and that a sclerosis of the postero-external column (and posterior cornu) may cause a sclerosis of the pyramidal tract in the lateral column; a lesion of the anterior cornu may pass to the crossed pyramidal tract, and so on.

§ 15. *Secondary system lesions (secondary degenerations).*—The regions of the transverse section, which are liable to be affected by secondary degenerations, are the regions of the crossed and direct pyramidal tracts, and the region of the postero-internal column. (See fig. 27.)

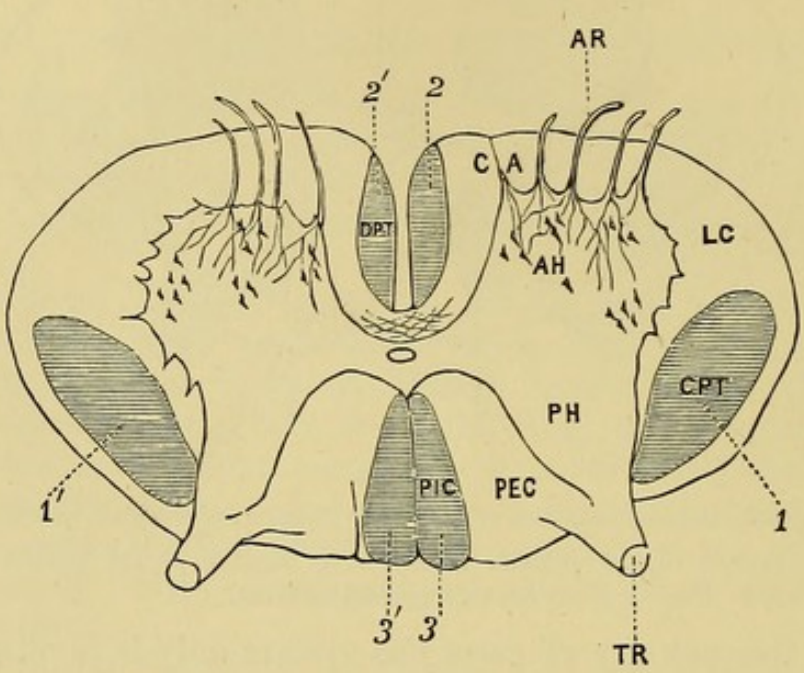


Fig. 27.

Diagrammatic representation of the secondary system lesions of the Spinal Cord.

1, 1′ Region of the crossed pyramidal tract. 2, 2′ Region of the direct pyramidal tract. 3, 3′ Region of the postero-internal column.

§ 16. *The secondary degenerations of the crossed and direct pyramidal tracts (secondary descending degenerations).*—The primary lesion, which produces secondary degeneration of the motor tracts in the cord, may be situated either in the brain or in the cord itself, but it must be so placed as to separate the fibres of the pyramidal tract from their trophic centre, *i.e.*, the nerve cells of the cortical motor centres. When the lesion is in the brain, where, for example, there is hæmorrhagic destruction of the *internal capsule*, the degenerative process extends downwards along the course of the pyramidal tract. In the cord, therefore, it involves the direct pyramidal tract on the same side, and the crossed pyramidal tract on the opposite side to the brain lesion. (See fig. 28.)

Where the primary lesion is situated in the cord itself the

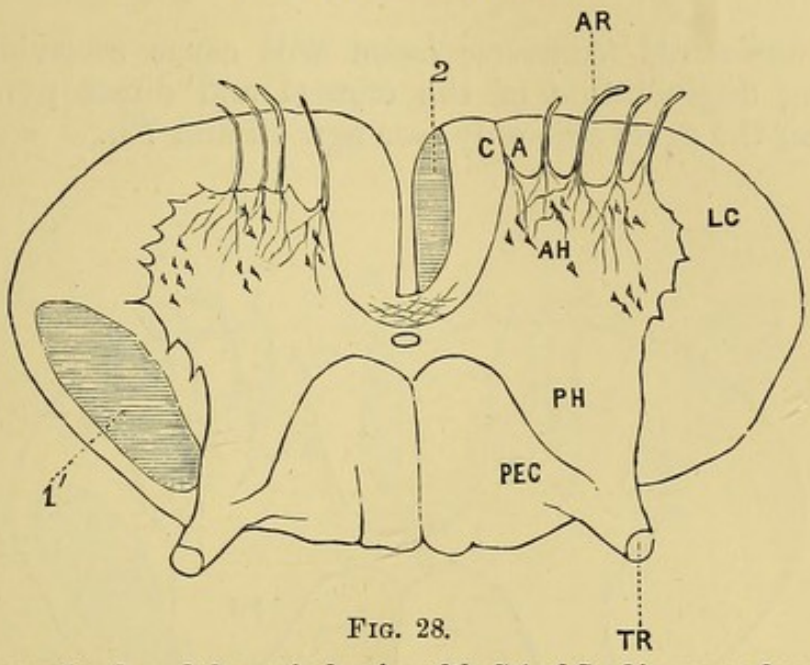


FIG. 28.

Transrerse section through the cervical region of the Spinal Cord in a case of secondary descending degeneration, the result of a lesion of the right pyramidal tract above the cord.
The crossed pyramidal tract (1) is degenerated on the left (the opposite) side to the brain lesion. The direct pyramidal tract (2) is degenerated on the right (the same side) as the brain lesion.

degenerative process may involve one or all of the pyramidal tracts below the lesion.

A total transverse lesion will produce secondary descending degeneration of the direct and crossed pyramidal tracts on both sides of the cord. (See figs. 29 and 71.)

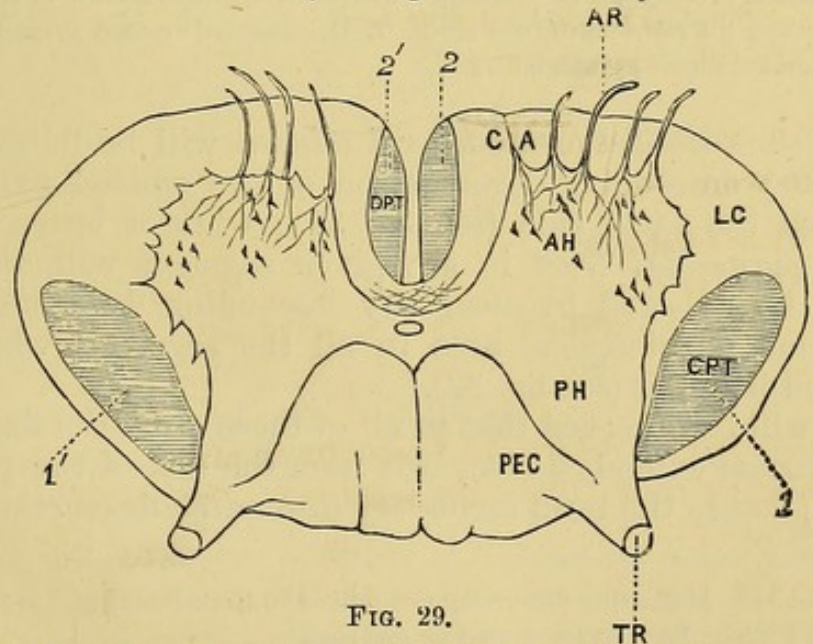


FIG. 29.

Secondary descending degeneration of the Spinal Cord after a total transverse lesion.
The lesion is supposed to be situated above the level of the section shown in the figure. 1, 1′ Degenerated crossed pyramidal tracts. 2, 2′ Degenerated direct pyramidal tracts.

A unilateral transverse lesion will cause secondary descending degeneration of the crossed and direct pyramidal tracts on the same side only (see figs. 30 and 73).

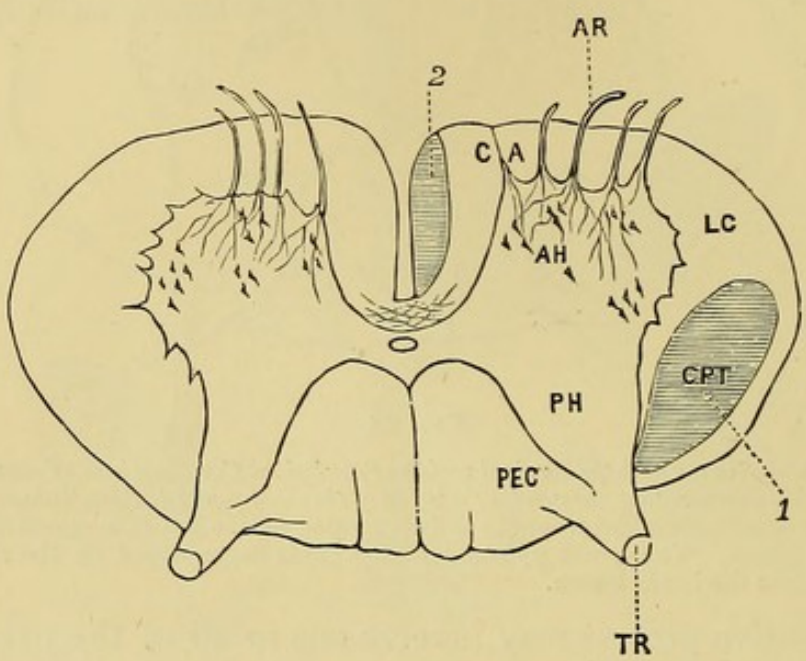


FIG. 30.

Secondary descending degeneration of the Spinal Cord after an unilateral lesion.

The lesion is supposed to be situated above the segment shown in the figure, and to occupy the right half of the cord. 1. Degenerated crossed pyramidal tract. 2. Degenerated direct pyramidal tract.

A lesion limited to the lateral column will be followed by secondary descending degeneration of the crossed pyramidal tract on the same side (see fig. 31); while a lesion of the *direct pyramidal tract* in any given segment will, theoretically,[1] be followed by secondary descending degeneration of the same physiological area in all the segments which are situated below it (see fig. 32).

It will be observed that in all of these cases the degenerative process extends downwards from the seat of the primary lesion; hence the term secondary *descending* degenerations.[2]

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[1] Secondary descending degeneration confined to the columns of Türck has not, so far as I know, been demonstrated *post mortem*.

[2] The fibres of the pyramidal tract above the lesion do not, of course, degenerate for their connection with the trophic centre is unimpaired.

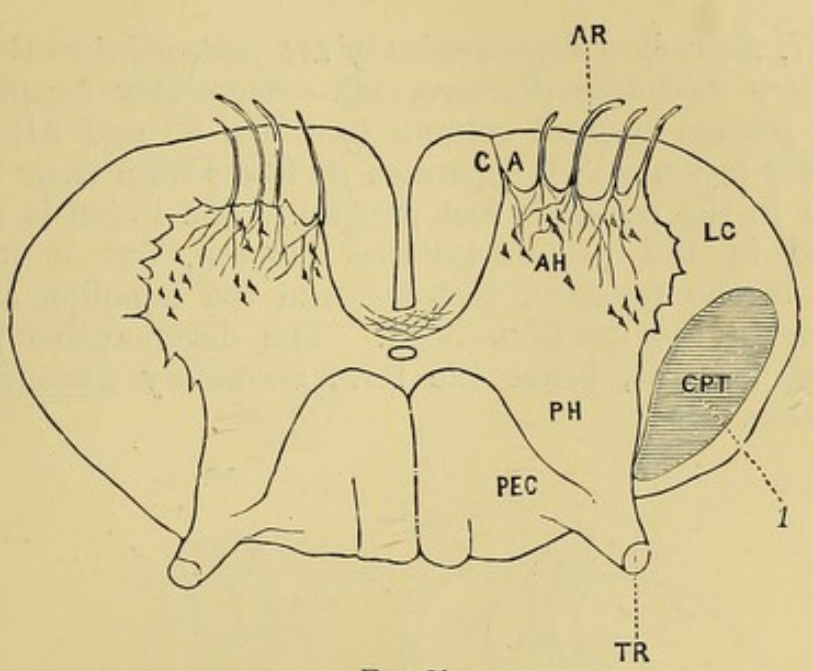


FIG. 31.

Secondary descending degeneration of the crossed pyramidal tract after a lesion of the lateral column on one side.

The lesion is supposed to be situated above the level of the section shown in the figure, and to involve the lateral column of the right side. 1. Degenerated crossed pyramidal tract of the right side.

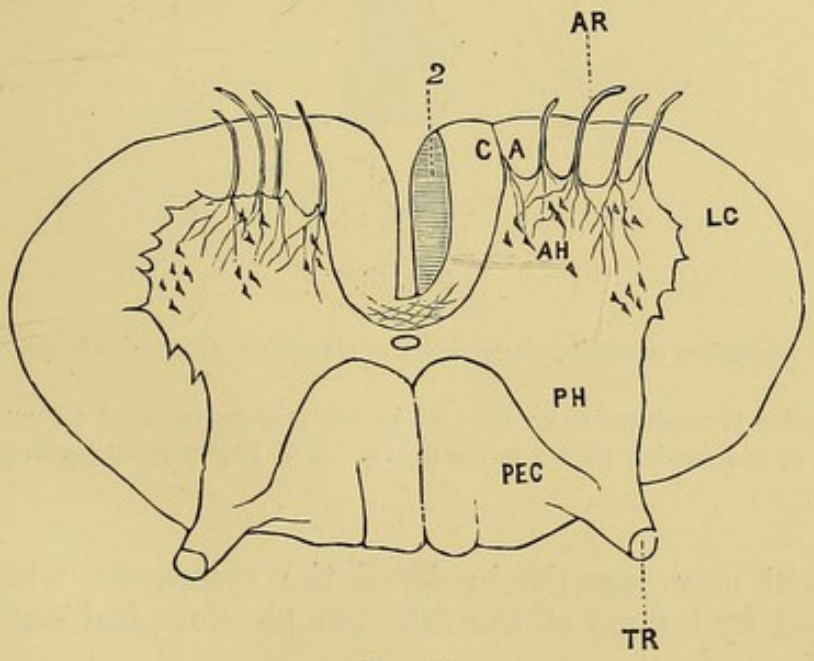


FIG. 32.

Secondary descending degeneration after a lesion confined to the anterior column of the cord.

The lesion is supposed to be situated above the level of the section shown in the figure, and to involve the anterior column of the right side. 2. Degenerated direct pyramidal tract of the right side.

§ 17. *Secondary degeneration of the postero-internal column (secondary ascending degeneration).*—Secondary degeneration of the postero-internal column (see figs. 33 and 34) results from any lesion which separates its fibres from their trophic centres in the posterior root ganglia. The lesion is usually situated in the postero-external column, but it may be placed in the posterior root between the ganglion and the cord, or in the ganglion itself. The degenerative process extends upwards, hence the term secondary *ascending* degeneration.

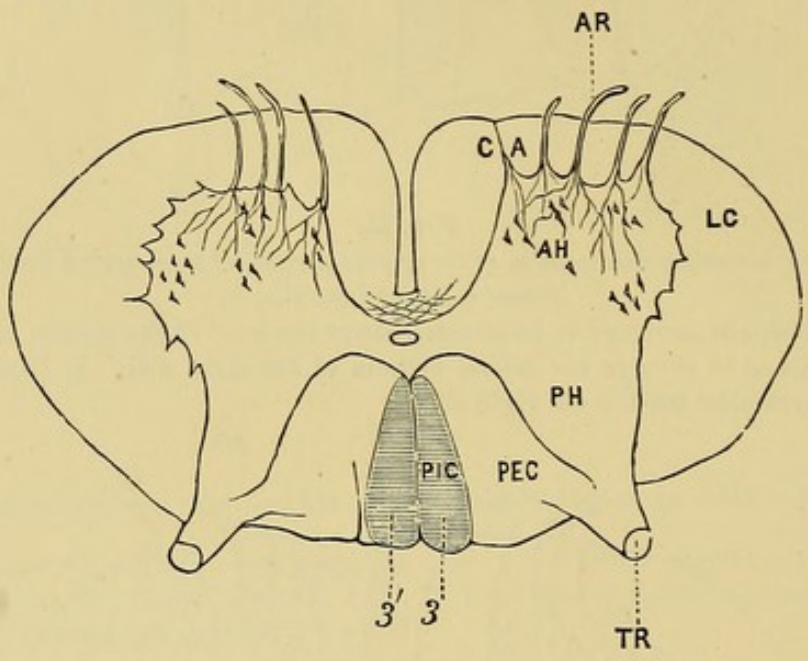


FIG. 33.

Secondary ascending degeneration of the postero-internal columns.

The lesion is supposed to be situated in the postero-external columns below the level of the section shown in the figure. 3, 3′ Degenerated postero-internal columns.

I will now describe in detail the symptoms which are produced by lesions of the different physiological regions or tracts.

§ 18. LESIONS OF THE ANTERIOR CORNU.

The ultimate effect of all lesions of the anterior cornu (see

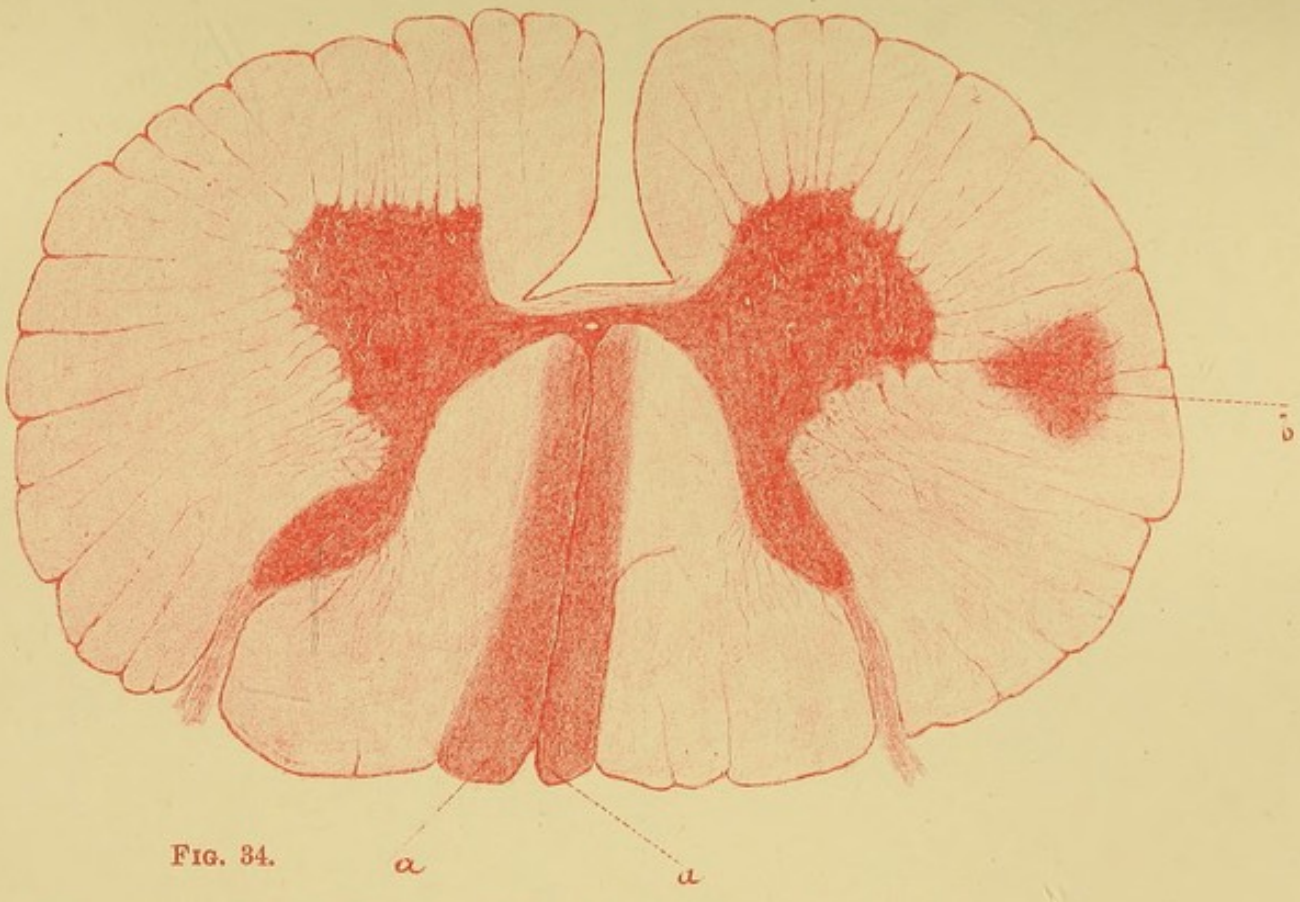


FIG. 34.

Transverse section through the cervical region of the spinal cord, showing secondary ascending degeneration of the columns of Goll. (Carmine and dammar), magnified about 10 *diameters.*

a, a, Degenerated postero-internal columns; b, a patch of degeneration in the lateral column.

I am indebted to Dr Robertson of Glasgow for the cord from which this preparation was made. The case was one of myelitis.

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fig. 35) is to cause destruction of the multipolar nerve cells which are its essential constituents. The effects of the lesion,

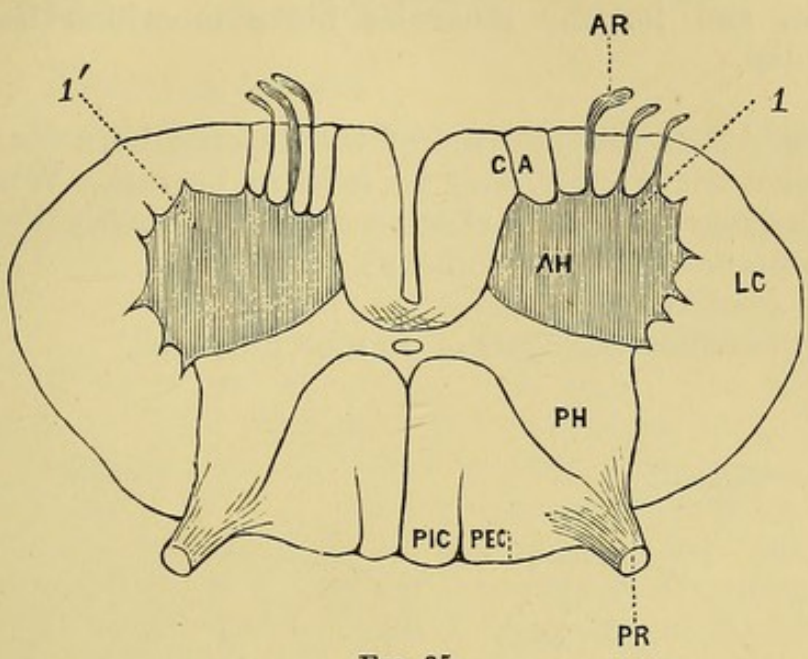


FIG. 35.

Transverse section of the Spinal Cord, showing the region of the anterior cornu (1, 1′) *shaded dark.*

i.e., the symptoms, in any given case, depend upon two elements, viz., the rapidity of the destruction, and the number of nerve cells which are destroyed. In order that you may be perfectly clear as to the *rationale* of the symptoms, allow me to recapitulate the functions of the multipolar nerve cells of the anterior cornu, and at the same time to direct your attention to figure 36.

§ 19. *The physiological function of the multipolar nerve cells of the anterior cornu.*—1. To the multipolar nerve cells of the anterior cornu pass:—(*a*) Voluntary motor impulses from the brain, *per* the fibres of the crossed and direct pyramidal tracts (see fig. 24); (*b*) reflex impulses from the 'sensitive area' of the segment, *per* the fibres of its posterior nerve roots; (*c*) controlling[1] impulses for the reflexes of the segment, *per* the fibres of the crossed pyramidal tract.

2. From the multipolar nerve cells—(*a*) voluntary and (*b*) reflex motor impulses pass to the muscular area of the segment, *per* their axis cylinder processes.

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[1] With regard to the question of separate controlling fibres, see page 26.

3. The multipolar nerve cells of the anterior cornu are the trophic centres for the motor nerve fibres arising from the segment, and for the muscular fibres to which they are distributed.

§ 20. ACUTE DESTRUCTION of the anterior horn (*i.e.*, of its multipolar nerve cells) may be *total* or *partial*. Where all the multipolar nerve cells of our segment are *acutely* destroyed, the symptoms will be as follows:

(a.) Positive symptoms.—

1. Immediate paralysis of its muscular area.

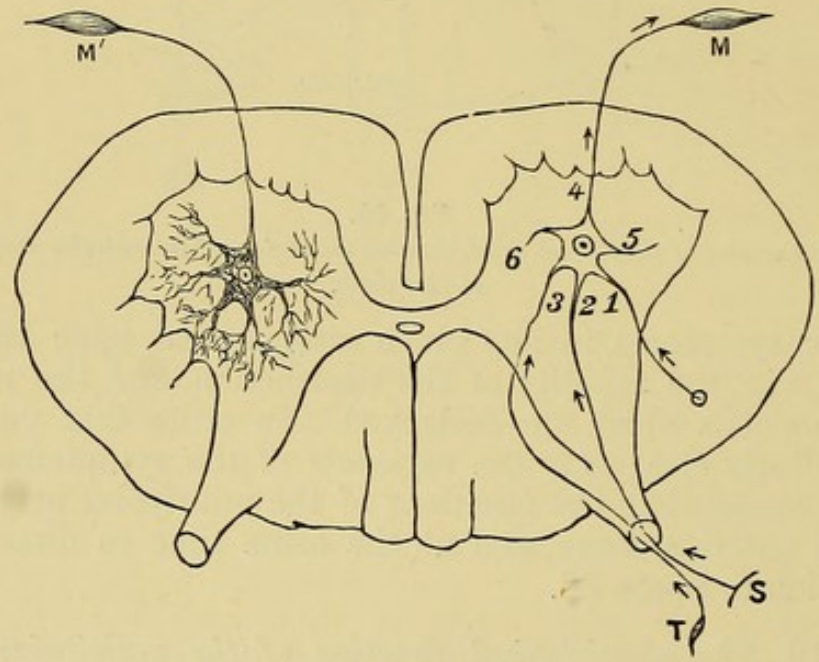


FIG. 36.

Diagrammatic representation of the connections of the motor nerve cells of the anterior cornu.

1. Branch which conveys to the cell, voluntary-motor and controlling-reflex impulses. 2. Branch which conducts to the cell the superficial reflexes. 3. Branch which conducts to the cell the deep reflexes. 4. Axis cylinder process, which transmits voluntary motor, reflex motor, and trophic impulses, from the cell to the muscle M. 5. Branch which places the cell in communication with other cells in other segments. 6. Branch which places the cell in connection with other cells in the opposite anterior horn of the same segment. On the left side the division of the cell processes, which terminate in Gerlach's nerve net work is shown.

2. Absence of all reflex movements in its muscular area.

3. Rapid atrophy, and the 'reaction of degeneration,'[1] in the paralysed muscles.

(b.) Negative symptoms.—

Lesions limited to the anterior cornu do not disturb the sensory functions of the segment, nor its functions as a conducting medium. There is, therefore, no derangement of sensibility in the parts supplied by its posterior roots. The motor, sensory, and other functions of all inferior segments are intact.

The extent of the paralysis and of the muscular atrophy varies directly with the number of nerve cells which are destroyed. Where the destruction is incomplete the paralysis is of course partial. In such cases the reflex impulses may still be obtained. In cases of partial destruction it may be difficult or impossible to demonstrate the 'reaction of degeneration,' for the healthy muscular fibres respond in a normal manner to both forms of current, and so obscure the reactions of disease.

In many cases of acute inflammation of the anterior cornu (polio-myelitis anterior acuta), in which there is at the outset total paralysis, a certain amount of power is subsequently regained. In such cases, the inflammatory process temporarily arrests the function of all the nerve cells of the affected segments. When the acute stage passes off, some of the cells have been absolutely destroyed, and the muscular fibres which they supply are permanently atrophied. Other nerve cells, which have been less seriously damaged, gradually recover, and the muscular fibres which they innervate regain bulk and power.

As a matter of experience, we know that, in *polio-myelitis anterior acuta* (the primary system disease of the anterior cornua) the functions of the bladder and rectum are not affected, a fact which seems to show either that the centres for the vesical and rectal reflexes are not situated in the anterior cornua, or that the multipolar nerve cells in the lower segments, through which the vesical and rectal reflexes pass, are uninjured by the lesion.

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[1] The term 'reaction of degeneration' has been given by Erb to certain *qualitative* electrical alterations, which are fully described on page 105.

In Fig. 37 I have diagrammatically represented the effects of *acute* destruction of the anterior cornua.

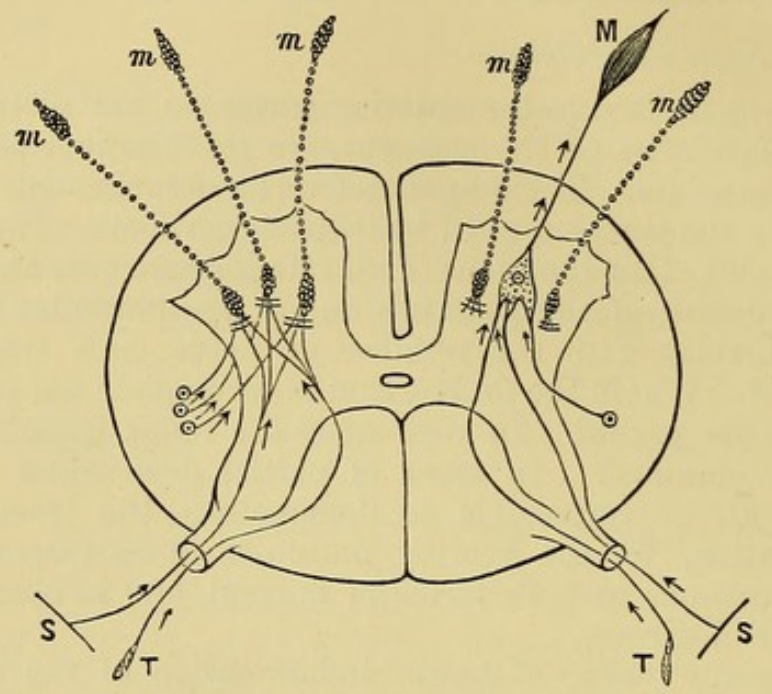


FIG. 37.

Diagrammatic representation of the symptoms which result from acute destruction of the anterior cornua of the spinal cord.

On the left side the destruction of the nerve cells is complete. The anterior nerve roots, motor nerve fibres, and muscles, which they supply, are all degenerated. There is a total 'block' to the passage of voluntary motor and reflex motor impulses. On the right side two-thirds of the motor cells are destroyed; two-thirds of the muscular area connected with the right anterior cornu are degenerated and atrophied; one-third (M) remains healthy, and can be made to contract by voluntary or reflex motor impulses.

Acute destruction of the multipolar nerve cells of the anterior cornu often occurs as a primary system disease (the morbid process commencing in, and being confined, to the anterior cornu). It then constitutes the affection which is termed *polio-myelitis anterior acuta* (acute inflammation of the anterior grey matter).

Acute destruction of the anterior horn of grey matter may also be part and parcel of an indiscriminate lesion, as, for example, a transverse myelitis. In such cases the symptoms, which result from acute destruction of the anterior cornu, are present, together with the symptoms which result from destruction of the other tracts which happen to be affected.

§ 21. *The morbid histology of polio-myelitis anterior acuta.* —Almost all authorities are at one in thinking that the lesion is inflammatory. Opinions differ as to whether the inflammation commences in the nerve elements or in the connective tissue, but all authorities are agreed that the ultimate effect of the inflammatory process is to cause acute destruction of the multipolar nerve cells. Atrophy of their axis cylinder processes, of the anterior root fibres, and of the muscular fibres to which they are distributed necessarily follows. The disease is rarely fatal; it is seldom, therefore, that an opportunity of observing the condition of the cord in the early stages occurs. In the few cases which have been examined, soon after the onset of the affection, an area of inflammatory softening was found in the lumbar or cervical regions of the cord.

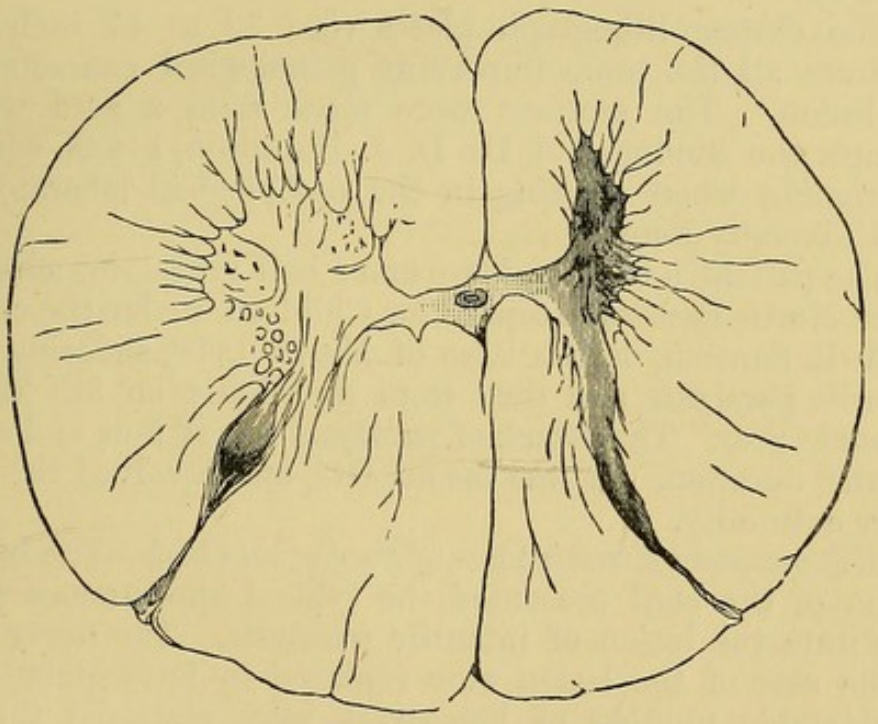


FIG. 38.

Transverse section of the cervical region of the Spinal Cord in long standing infantile paralysis. (*After Charcot.*)

The patient, a woman, æt. 50, died in the Salpetriére from *General Paralysis of the Insane.* The right upper extremity had been affected with Infantile paralysis. There was fibroid atrophy of the right anterior cornu, and atrophy of the white columns of the corresponding side.

The microscopical examination, in recent cases, shows the usual appearances of an acute myelitis (see page 52): the nerve elements are more or less disintegrated; many of the nerve cells have entirely disappeared; compound granular corpuscles and oil globules infiltrate the softened tissue and adhere to the outer coats of the vessels; the connective tissue elements in the neighbourhood of the inflamed part are proliferating, nuclei and leucocytes are scattered throughout the surrounding tissue.

At a later stage the softening is less marked, or has entirely disappeared. The nerve cells are in great part destroyed, and are here and there replaced by oil globules. The vessels of the affected part are dilated; and the connective tissue elements increased. Ultimately the affected cornua become atrophied and sunken, as is represented in figure 38.

The chromo-lithograph plates (figs 39 to 45 inclusive) illustrate all the more important pathological characters of the lesion. The sections were made from a cord which, through the kindness of Dr D. J. Hamilton, I was enabled to examine when working in the pathological laboratory of the University here.

The patient, a boy, aged two and a half years, was admitted to the North-Eastern Hospital for Children, under the care of Dr A. E. Sansom, on the 21st of March 1879, suffering from infantile paralysis, and died from diphtheria on 9th May of the same year. The attack of paralysis was of four and a-half months' duration. It was incomplete, and involved the right lower extremity.

Microscopical Examination of the Spinal Cord.—The lumbar region of the cord presented the typical appearances which constitute the lesion of infantile paralysis. The nerve cells at the seat of the lesion were replaced by fatty globules; a considerable number of leucocytes were scattered through the anterior cornua, and, indeed, through the grey matter generally; the connective tissue corpuscles of the grey matter were more numerous than in health; some of the blood-vessels in the anterior cornua seemed abnormally large and dilated; fatty globules, similar to those replacing the nerve cells, were adherent to the outer coat of some of the vessels. The white columns of the cord were perfectly healthy.

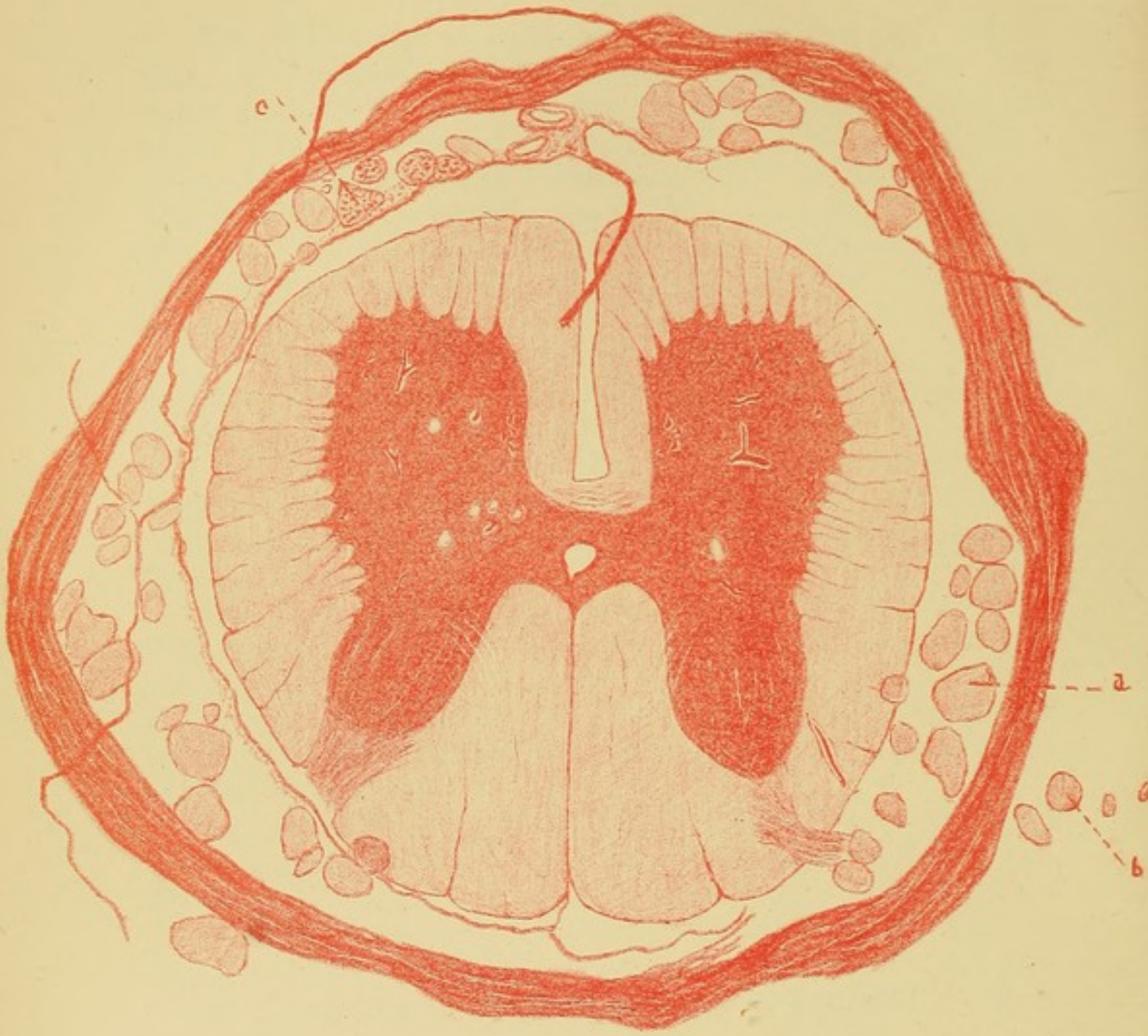


FIG. 39.

Transverse Section of the Spinal Cord in the Lumbar Region, from a case of Infantile Paralysis (carmine and dammar), magnified about 10 diameters.

The membranes are still *in situ*. Numerous transversely divided nerve roots, a, are seen between the dura and the arachnoid. The process of membranes, which passes into the anterior median fissure, is slightly displaced; a few of the transversely divided nerve roots, b, have escaped outside the dura in the process of mounting.

Nearly all the multipolar nerve cells of the left anterior cornu have disappeared. The inner group still remains. Compare with fig. 40. Some of the anterior root bundles (*c*) can be seen to be degenerated even with this low power. The central canal is of large size; under a high power it is seen to be beautifully healthy.

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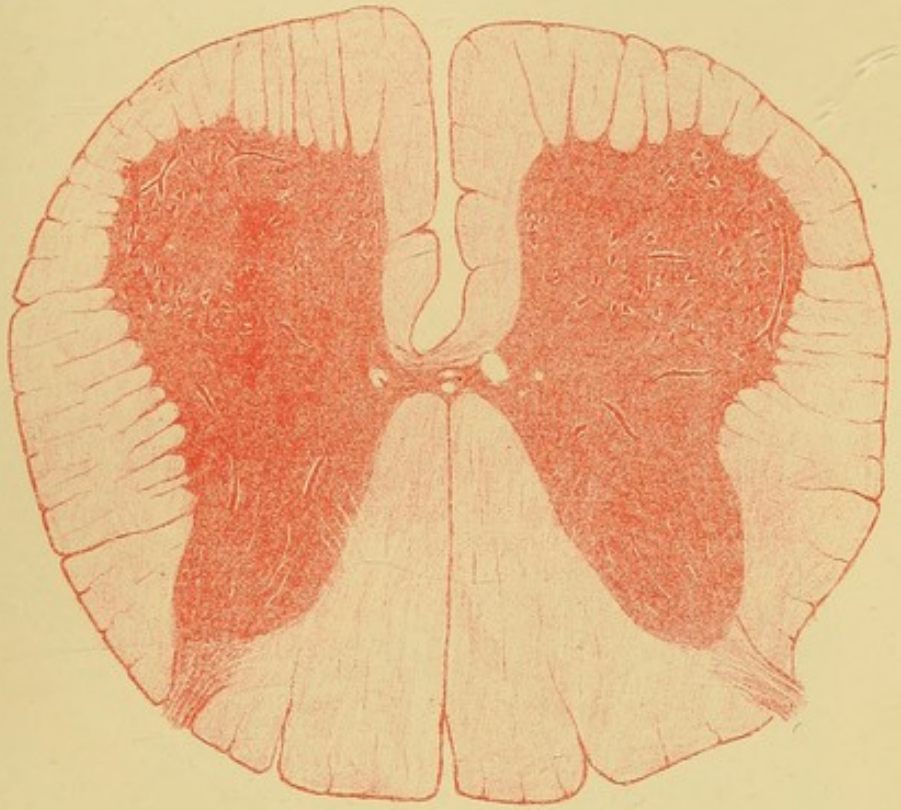


FIG. 40.

Transverse Section through the Lumbar Region of the Spinal Cord of a Child, showing the normal appearance of the Anterior Cornua (carmine and dammar) magnified about 10 diameters.

Numerous multipolar nerve cells are seen in the anterior cornua. The central canal is double. This condition is a rare anatomical variation of no practical importance.

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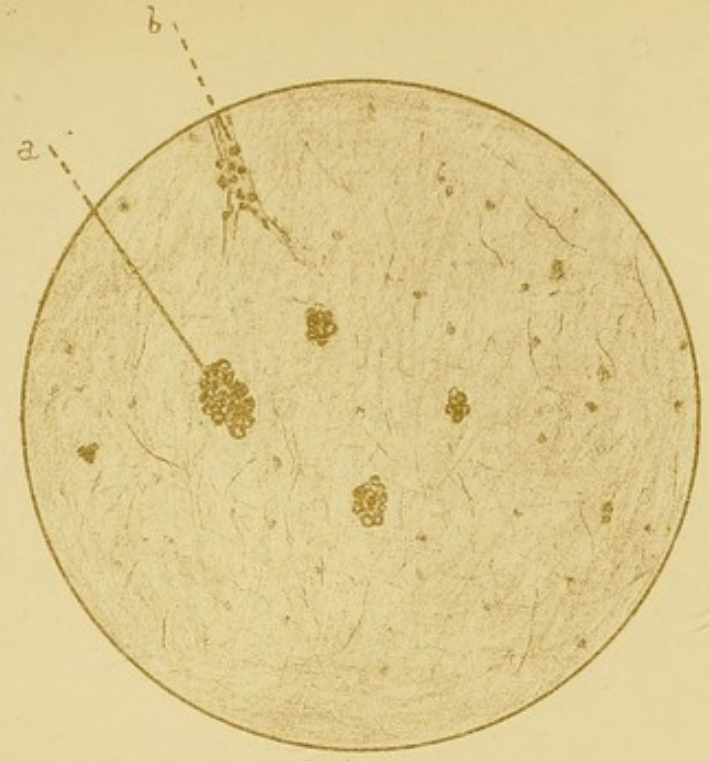


FIG. 42.

Portion of the Anterior Horn of grey matter represented in fig. 41, *showing the minute structure of the lesion (osmic acid and farrant) magnified* 250 *diameters.*

The dark particles seen in fig. 41 are fatty globules stained with osmic acid. They are situated in spaces which have formerly contained nerve cells. A small vessel is seen at the upper part of the section — some fatty globules adhere to its outer coat.

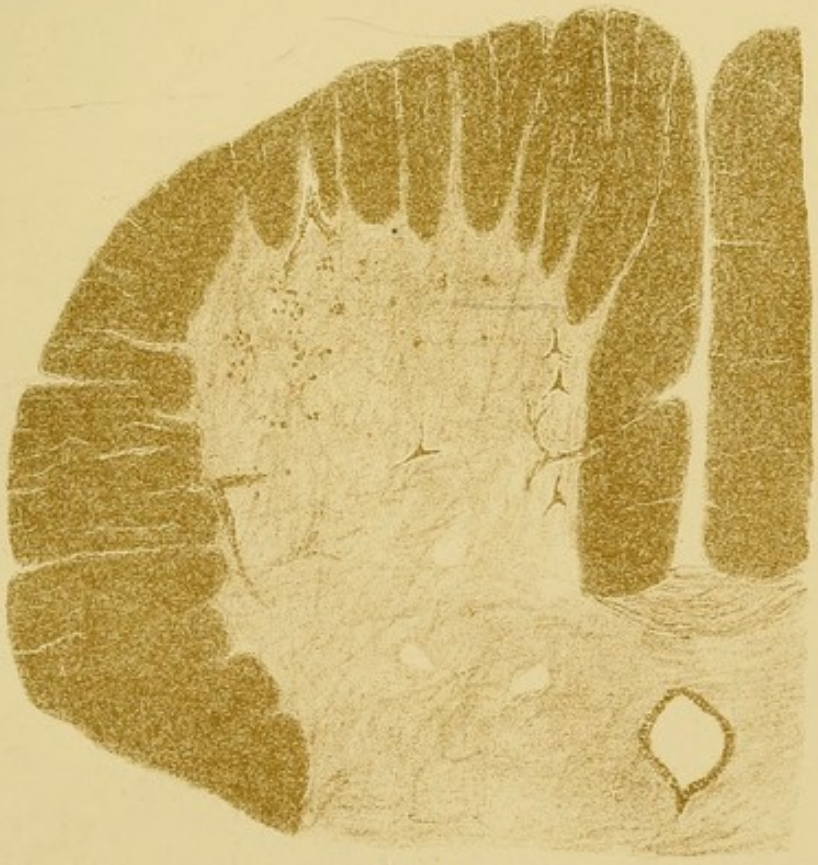


FIG. 41.

Transverse Section through the Anterior Horn of grey matter in a case of Infantile Paralysis (osmic acid and farrant), magnified about 18 *diameters.*

Nearly all the nerve cells have disappeared. Their place is taken by fatty particles which can be easily seen even with this low power.

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FIG. 43.

Transverse Section of an Anterior Nerve Root from a case of Infantile Paralysis, showing marked degeneration. The preparation is one of the anterior nerve roots, c, seen in Fig. 39, more highly magnified. × 200 diameters.

Many of the nerve tubes have disappeared, and are replaced by masses of connective tissue, a, a, which stain deeply with carmine; b, transversely divided blood vessel.

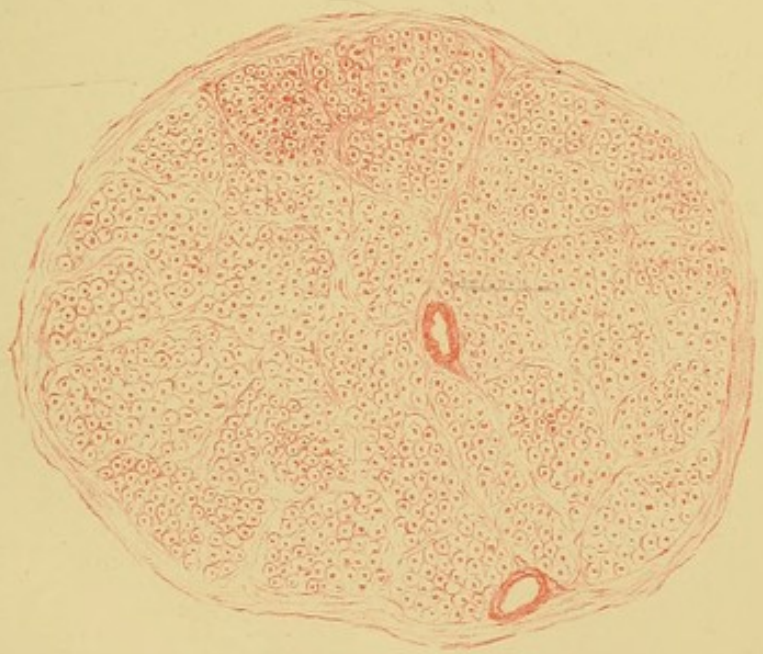


FIG. 44.

Tranverse Section of a healthy Anterior Nerve Root from the preparation shown in Fig. 39. Stained with carmine, cleared with oil of cloves, and mounted in dammar. × 200 *diameters.*

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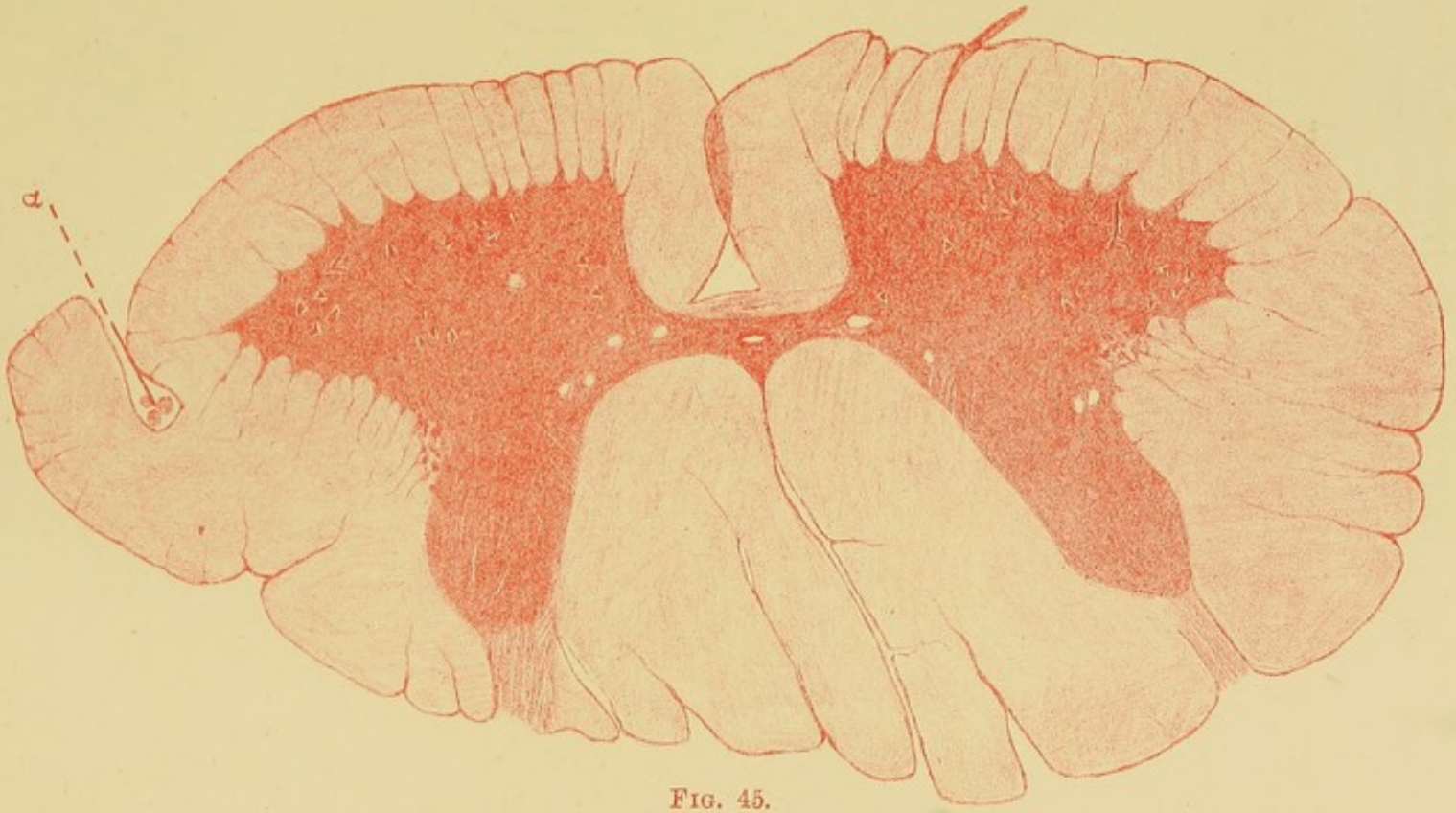


FIG. 45.

Transverse Section of the Cervical Portion of the Spinal Cord in a case of Infantile Paralysis (carmine and dammar), magnified about 10 *diameters.*

The lateral column on the left side is split up by a deep fissure and is curiously misshapen. At the bottom of the fissure a bundle of nerve fibres, *a*, evidently a transversely divided nerve root, is situated. This alteration in the shape of the lateral column is a congenital condition, and probably had nothing to do with the paralysis. The minute structure of the lateral column is perfectly healthy.

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§ 22 Chronic Affections of the Anterior Cornu.—In chronic affections of the anterior cornu the multipolar nerve cells are picked out, as it were, one by one, and are gradually destroyed.

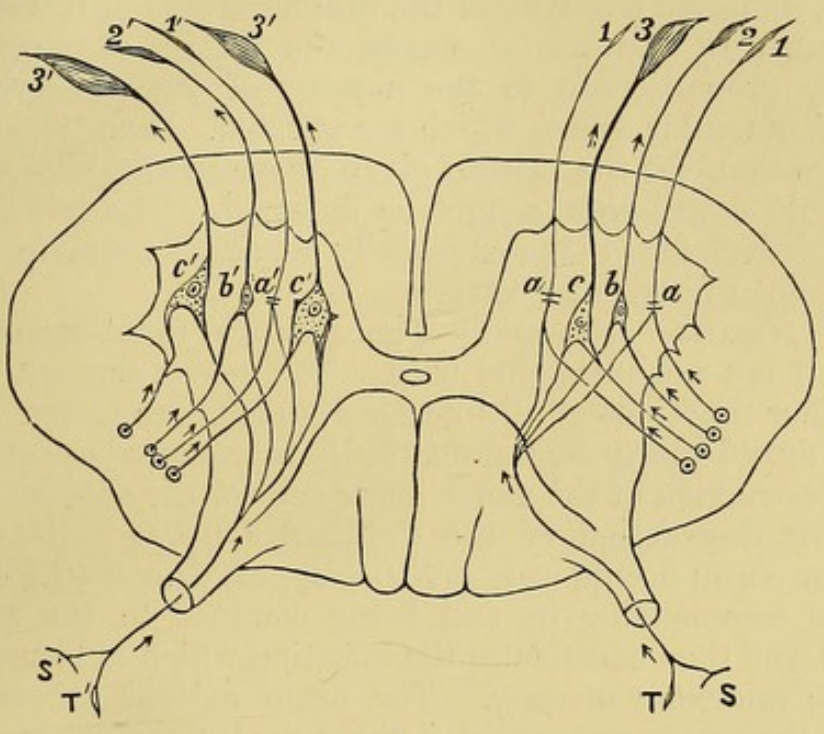


Fig. 46.

Diagrammatic representation of the symptoms which result from slow destruction of the multipolar nerve cells of the anterior cornu.

On the left side the disease is in an early stage. One nerve cell (a′) is completely destroyed. Its muscular fibre (1′) is completely atrophied. Voluntary motor and reflex motor impulses are 'blocked' at the seat of the lesion (a′). One nerve cell (b′) and its muscular fibre 2′ are very much atrophied, but feeble motor and reflex impulses can still pass through the cell to the muscle. Two nerve cells c′ c′ are healthy. Their muscular fibres are of normal bulk, and can be made to contract either by voluntary or reflex impulses.

On the right side the disease is much more advanced. The muscular area is three-fourths degenerated. There is a total 'block' at a and a. This condition represents a late stage of progressive muscular atrophy. The atrophy of the muscular fibres is represented as *simple*. There is not the same fatty change as is shown in figure 37.

The symptoms, which result from slow destruction of the multipolar nerve cells, are as follows:—

Positive symptoms.—(*a*) Slow and gradual atrophy of the muscular fibres to which the axis cylinder processes of the affected cells proceed.

At first, there is no paralysis but only slight muscular weakness. As the disease progresses the muscular debility becomes more marked. In the final stages, *i.e.*, when all the motor nerve cells of the segment are destroyed, there is, of course, complete paralysis of the muscular area of the affected segment. In the earlier stages, the degree of atrophy is directly proportionate to the number of motor nerve cells (*i.e.*, of muscular fibres) which are affected. The 'reaction of degeneration,' for the reasons given above in speaking of partial acute destruction, cannot be detected. The reflexes are not abolished, for so long as healthy nerve cells remain, a path is still open for reflex impulses.

(*b*) *Negative symptoms.*—The sensory functions of the segment are not interfered with. The motor, sensory, reflex, and other functions of all inferior segments are intact.

In fig. 46 I have diagrammatically represented the effects of slow destruction of the motor nerve cells of the anterior cornu.

Slow destruction of the multipolar nerve cells of the anterior cornu occurs as a *primary system disease* (the morbid process commencing in, and being confined to, the anterior cornu), and then constitutes the affection which is termed *progressive muscular atrophy.* The same morbid process also affects the motor nerve nuclei of the medulla, and constitutes the anatomical substratum of the affection called *glosso-labial* or *bulbar paralysis.*

Chronic destruction of the multipolar nerve cells of the anterior horn is occasionally met with as a *secondary system lesion*, as in those cases in which a sclerosis of the postero-external column, or of the crossed pyramidal tract, extends to the anterior cornu. In such cases the symptoms which I have just described as characteristic of slow destruction of the motor nerve cells of the anterior cornu, are added to the symptoms of the original affection.

§ 23. SUB-ACUTE DESTRUCTION OF THE MULTIPOLAR NERVE CELLS also occurs as a secondary system lesion. The original lesion is a sub-acute inflammation of the crossed pyramidal tract in the lateral column. After a short time the morbid process extends to the anterior cornu. The segments of the cervical enlargement are first and most affected. This condition has been called by Professor Charcot, who was the first to direct attention to it, *amyo-trophic lateral sclerosis.*

§ 24. LESIONS OF THE CROSSED PYRAMIDAL TRACT IN THE LATERAL COLUMN.

Lesions of the crossed pyramidal tract (see fig. 47) derange, interrupt, or destroy the conducting power of the nerve fibres of which it is composed. These fibres conduct to our segment, and to all the segments which are situated below it, voluntary motor and controlling reflex impulses. The exact nature of the symptoms, which result from a lesion of the crossed pyramidal tract in our segment, depends upon *(a)* the extent of the lesion, *i.e.*, the number of nerve fibres which are affected by it; *(b)* the manner in which the lesion affects the nerve fibres; and *(c)* the rapidity with which the morbid process is developed.

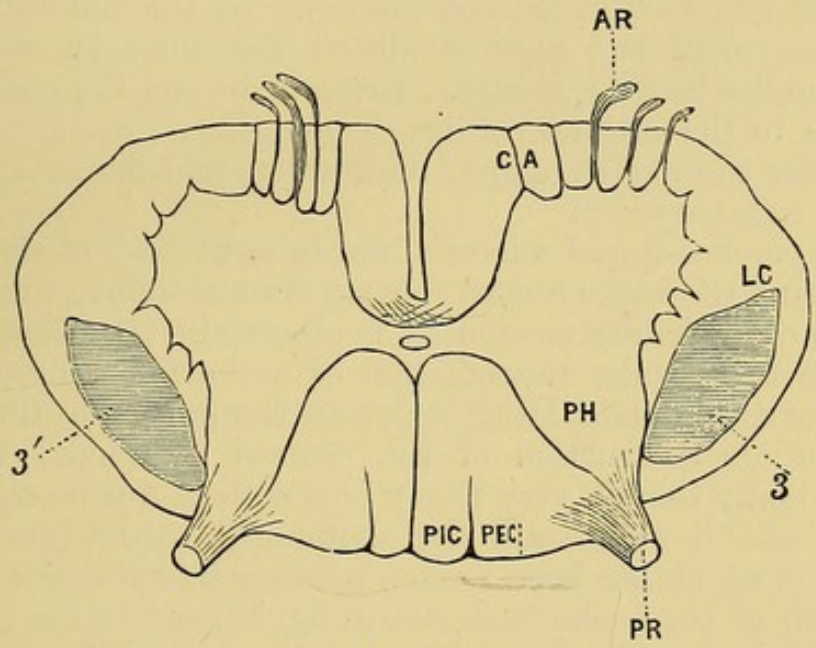


FIG. 47.

Transverse section of the cord showing a symmetrical lesion of the crossed pyramidal tracts. 3, 3′ point to the position of the lesion in each lateral column.

§ 25. *Acute and complete destruction of the crossed pyramidal tracts in the lateral columns* produces the following symptoms: (*a*) *Positive.* — 1. Immediate paralysis of the muscular areas of our segment, and of all the segments which are situated below it. The loss of power is not total, for the muscular fibres supplied by the direct pyramidal tract of course escape. The paralysed muscles are at first flaccid, they do not undergo rapid atrophy. There is 'no reaction of degeneration.' 2. Increase of the reflexes passing through our segment, and through all the inferior segments supervenes, and corresponds to the development of secondary descending degeneration,

which of course occurs. (*b*) *Negative symptoms.*—The sensory functions of the cord are intact. The bladder and rectum are not seriously affected.

Acute destruction of the crossed pyramidal tract only occurs in 'indiscriminate' lesions (myelitis, traumatic injuries, etc.). Other parts of the transverse section of the segment are usually implicated. The clinical picture is proportionately complicated.

§ 26. *Chronic destruction of the crossed pyramidal tracts.*—Where the morbid process is *chronic*, the conducting properties of the crossed pyramidal tract are *gradually* interrupted. Muscular weakness, stiffness, and rigidity slowly develop: the reflexes are increased: sensibility is intact: the bladder and rectum are not affected. As the morbid process becomes more and more advanced, the muscular weakness and rigidity become greater. Towards the end the lesion may extend to the anterior cornu. The rigidity then gives way: muscular atrophy develops: diminution, and finally abolition of the reflexes occur.

In cerebro-spinal sclerosis, and in some cases of slow compression of the cord, a remarkable rhythmical tremor, which only occurs on voluntary movement, is observed. It is supposed to be due to irregular transmission of nerve force through the fibres of the pyramidal tract which are compressed by the lesion.

Chronic destruction of the crossed pyramidal tract is occasionally though very rarely met with as a *primary system lesion*, and it constitutes the anatomical substratum of the affection which has been termed *primary lateral sclerosis.* The position of the lesion is shown in fig. 47, and in the chromo-lithograph, fig. 48. The section shown in the chromo-lithograph was made from a portion of cord which Dr Dreschfeld of Manchester, was good enough to send me. The case is one of great interest, and is recorded by Dr Morgan and Dr Dreschfeld in the *British Medical Journal* for January 1881. According to Professor Charcot, this is the only case in which *post-mortem* examination has demonstrated a lesion confined to the lateral columns, without participation of either the grey matter or the posterior columns.

Chronic destruction of the crossed pyramidal tract frequently results from *indiscriminate lesions* (chronic myelitis, slow compression of the cord, cerebro-spinal sclerosis, etc.). In *secondary descending degenerations* the fibres of the crossed pyramidal tract are, of course, gradually destroyed.

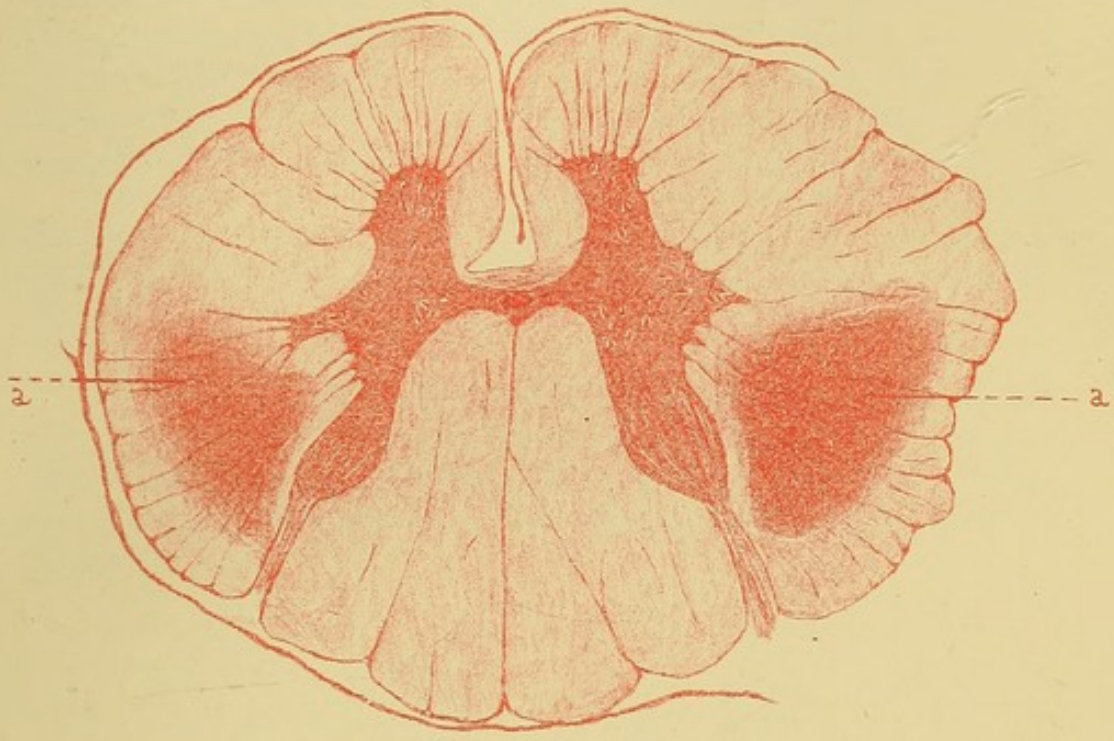


FIG. 48.

Transverse section through the upper dorsal region of the cord in a case of Primary Lateral Sclerosis. Stained with carmine, mounted in dammar, and magnified about 10 *diameters.*

a, a, Sclerosed patches mapping out the region of the crossed pyramidal tracts.

The preparation was made from a portion of cord which was kindly sent me by Dr Dreschfeld of Manchester.

B.B. delt ad. natm

McLagan & Cumming, Lith. Edin.

§ 27. LESIONS OF THE POSTERO-EXTERNAL COLUMN.

The postero-external column is in great part composed of posterior root-fibres passing to the grey matter. These fibres carry inwards impressions which result in the production of pain, touch, and reflex movements (probably deep reflex movements, see page 25). The postero-external column probably also contains commissural fibres which connect the posterior cornua of different segments. Possibly, too, sensory conducting fibres, after they have decussated, pass from the grey matter into the postero-external column *en route* to the cerebrum.

The most common lesion of the postero-external column is the *primary system lesion* which constitutes the anatomical substratum of the affection which is called locomotor ataxia. (See fig. 49.)

§ 28. *The morbid histology of locomotor ataxia.*—The morbid process seems to commence in the nerve elements, and is probably an extremely chronic inflammation. The nerve tubes are gradually destroyed. The fibrous tissue (neuroglia) is increased.

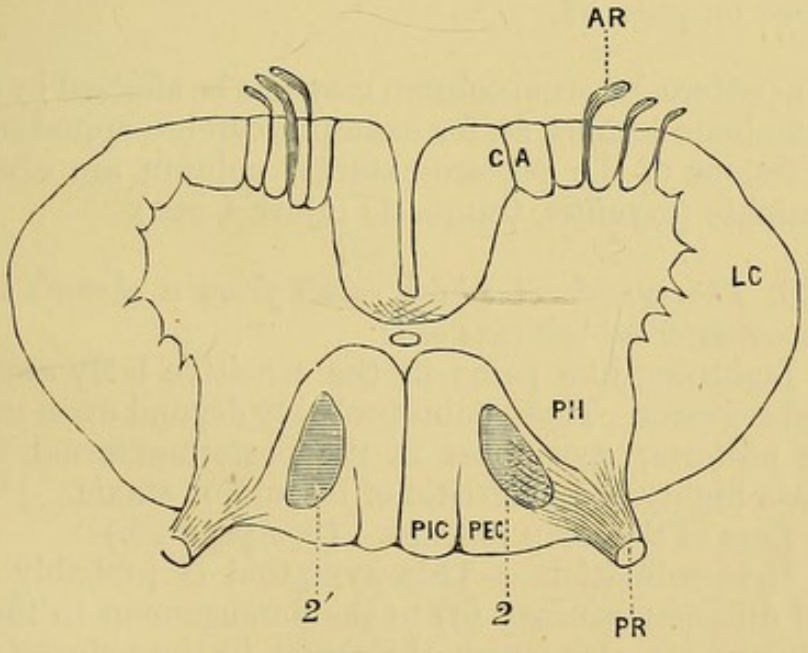


FIG. 49.

Transverse section of the spinal cord, showing the position of the lesion in the early stage of locomotor ataxia (primary sclerosis of the postero-external columns).

The term 'sclerosis' is usually given to this and to all those lesions in which the connective tissue of the cord is increased.

The name is somewhat misleading, for it includes, at least, two distinct morbid conditions. In both forms of sclerosis the ultimate result is the destruction of the nerve elements, and the increase of the neuroglia; but in one (locomotor ataxia) the lesion commences in the nerve elements; in the other (disseminated sclerosis) in the neuroglia; in the latter case the destruction of the nerve elements is a secondary process, the nerve tubes being, as it were, strangled by the connective tissue growth which surrounds them. In locomotor ataxia numerous corpora amylacea are found; in disseminated sclerosis the tissue is filled with compound granule corpuscles, the lymphatic sheaths of the blood vessels are crowded with oil globules, and fatty (margarine?) crystals are sometimes to be seen in the sclerosed tissue. Compound granule cells are also found in locomotor ataxia, but they are, I think, much less numerous than in disseminated sclerosis.

The chromo-lithograph plates (figs. 50 to 54 inclusive) illustrate all the more important histological characters of the lesion in locomotor ataxia.[1] The histological characters of the form of sclerosis which commences in the neuroglia are described on page 56.

The postero-external column may also be affected by chronic indiscriminate lesions, as, for example, cerebro-spinal sclerosis. Acute lesions of the postero-external column are always indiscriminate (myelitis, traumatic injuries, etc.)

§ 29. *The symptoms which result from a chronic lesion of the postero-external columns* are:

1. Lightning-like pains in the sensitive body area of the affected segment. These pains probably depend upon irritation of the posterior root-fibres in the postero-external column. They are highly characteristic of locomotor ataxia.

2. Loss of the deep reflexes. (See page 25.)

3. Inco-ordination. — This symptom is probably due to several different causes: (*a*) to the derangement in the reflex grouping of muscles which the arrest in the reflexes entails; (*b*) to the fact that the 'guiding sensory sensations' which

[1] The full details of the case, which, through the kindness of Dr D. J. Hamilton, I was enabled to examine when working in the pathological laboratory of the Edinburgh University, are recorded in the *Edinburgh Medical Journal, January* 1871.



FIG. 50.

Transverse section through the dorsal region of a healthy Spinal Cord. Stained with osmic acid, mounted in Farrant's solution, and magnified about 10 *diameters.*

All the white columns are deeply stained by the acid. The grey matter is only slightly affected by it.

B.B. delt. ad. natm.

McLagan & Cumming, Lith. Edinr.

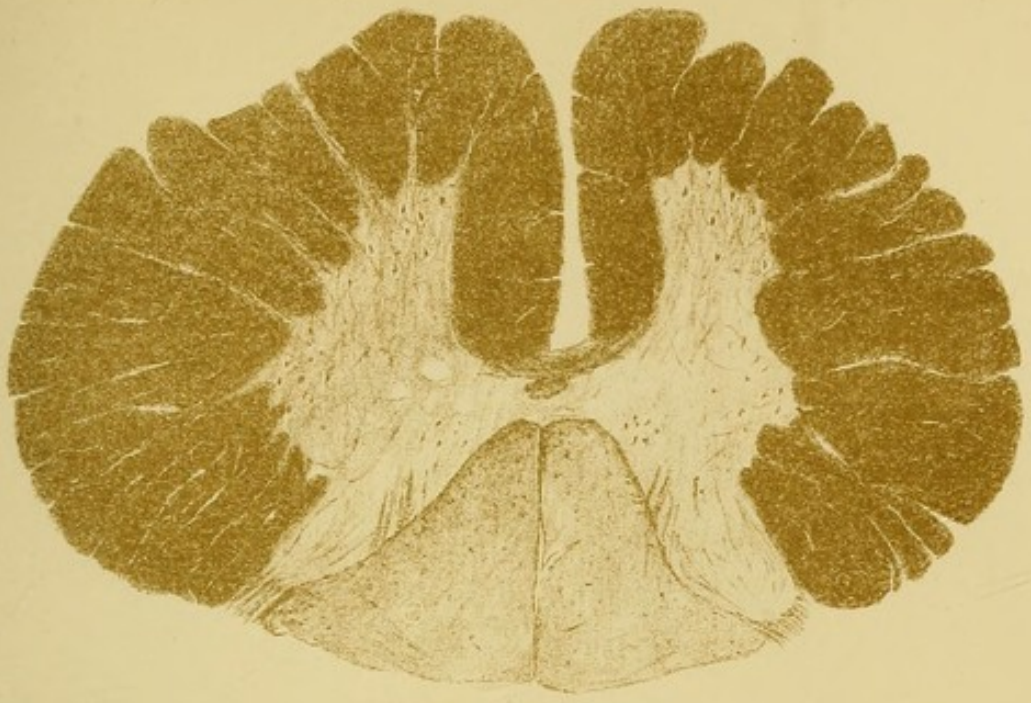


FIG. 52.

Transverse section through the dorsal region of the cord in a case of Locomotor Ataxia. Stained with osmic acid, mounted in Farrant's solution, and magnified about 10 *diameters.*

The lesion involves the whole of the posterior columns, which are seen to be very faintly stained by the acid.



FIG. 51.

Transverse section through the lumbar region of the cord in a case of Locomotor Ataxia. Stained with osmic acid, mounted in Farrant's solution, and magnified about 10 *diameters.*

The greater part of the posterior columns is invaded by the lesion, and is, therefore, unstained by the acid. The part of the posterior columns adjacent to the posterior commissure is still healthy.

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McLagan & Cumming, Lith. Edin.

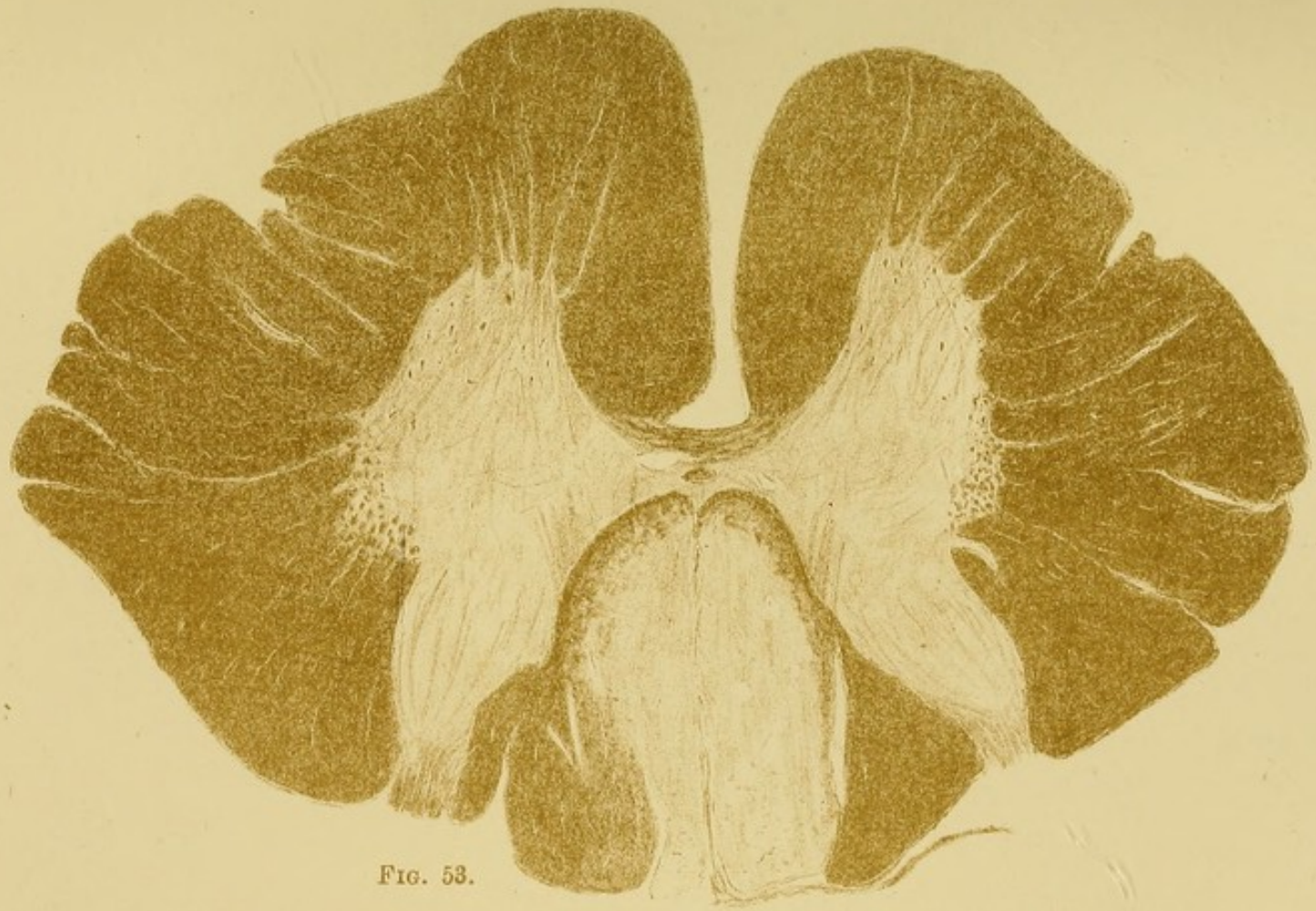


FIG. 53.

Transverse section through the cervical enlargement in a case of Locomotor Ataxia. Stained with osmic acid, mounted in Farrant's solution, and magnified about 10 *diameters.*

The lesion is confined (or almost so) to the postero-internal columns; and presents the usual features of a secondary ascending degeneration.

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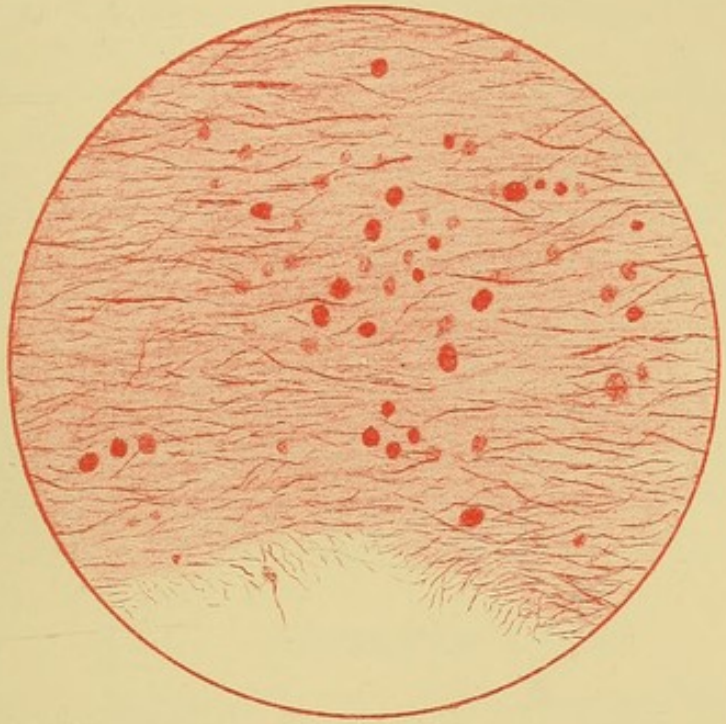


FIG. 54.

Longitudinal section through the posterior column of the cord in Locomotor Ataxia. Stained with carmine, mounted in dammar, and magnified about 250 *diameters.*

The nerve tubes have almost entirely disappeared. Their place is taken by a delicate connective tissue. Numerous corpora amylacea are scattered through the section. These bodies are deeply stained by carmine and logwood, faintly by osmic acid.

B.B.delt. ad. nat.

Mc Lagan & Cumming, Lith. Edin^r.

proceed from the periphery are arrested by the lesion; (*c*) probably also in part to derangement of the commissural connections of different segments which pass through the postero-external columns.

4. More or less anæsthesia and analgesia in the sensitive areas of the affected segments.

5. Some derangement of the vesical and rectal mechanisms (Often, but not always, present.)

6. Trophic disturbances in the skin, such as herpetic and other eruptions. (Sometimes observed.) They probably depend upon implication of the central grey matter.[1]

7. (Negative symptoms.) No paralysis; no muscular atrophy; no qualitative electrical alterations.

Lesions of the postero-external column cut off the trophic influence of the posterior root ganglia, and are therefore followed by secondary *ascending* degeneration.

Complications.—In chronic lesions of the postero-external column the posterior horn of grey matter is often affected. The morbid process occasionally extends to the anterior cornu, or to the lateral column. Other symptoms then, of course, develop.

§ 30. 'INDISCRIMINATE LESIONS.'

The great characteristic of an 'indiscriminate' lesion is, as I have already mentioned, that it is not, *of necessity*, limited to any particular physiological tract, but may affect any part of the transverse section, though it occasionally, but rarely, happens that an indiscriminate lesion may be limited to a definite physiological tract. In cerebro-spinal sclerosis, for example, a patch of sclerosis may chance to be limited to the postero-external columns. Inco-ordination in the lower extremities, identical with the inco-ordination of locomotor

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[1] This description does not comprise all the symptoms of locomotor ataxia, but only those which result from the lesion of the postero-external columns, *i.e.*, the *spinal* portion of the lesion. Some of the most characteristic symptoms, as, for example, the eye symptoms, depend upon derangement of the parts supplied by cranial nerves. The disease is, in short, a cerebro-spinal affection. Trophic alterations in the bones are occasionally met with. The osseous lesions have not been referred to in the text, because they do not result from the lesion of the postero-external columns. Professor Charcot, who first described them, thinks they depend upon implication of the motor nerve cells of the anterior cornu. Dr Buzzard suggests that they are caused by a lesion of the medulla.

ataxia, might thus be produced. The differential diagnosis could only be made by observing the mode of development of the symptoms, the associated nerve derangements, and the general features of the case.

The vertical extent of indiscriminate lesions is, as a rule, small; indeed the morbid process may be limited to a single segment. Many separate foci of disease may be scattered through the cord, but such cases do not constitute exceptions to the general rule, for the vertical extent of individual patches is usually small.

Indiscriminate lesions have no marked tendency to invade symmetrical parts of the two lateral halves of the transverse section. In some cases they are unilateral.

The indiscriminate lesions which are primarily nervous are either acute or chronic. Myelitis is the best example of the former; disseminated cerebro-spinal sclerosis is a good example of the latter. Other indiscriminate lesions are simple (non-inflammatory) softening; hæmorrhage into the substance of the cord; and new formations in the substance of the cord (*intra*-medullary tumours).

§ 31. *The morbid anatomy of acute myelitis.*—Three stages are usually described. The *first* is that of *congestion* or *stasis.* The affected portion of cord is seen on section to be red and hyperæmic. It swells up above the level of the adjacent healthy tissue, and is somewhat softer than natural. On microscopical examination the vessels are found to be distended with blood. The lymphatic sheaths are in places crowded with leucocytes. Capillary hæmorrhages are sometimes present. The axis cylinders and nerve cells are, even in the early stages, hypertrophied (see figs. 55 and 56). The connective tissue elements are commencing to proliferate, the stellate corpuscles (Deiters' cells) are unusually prominent. In the softened parts the nerve elements are breaking down, compound granule corpuscles and oil globules are seen.

In hardened specimens a glassy, colloid-like exudation, is frequently met with. It surrounds the blood vessels and infiltrates the tissues. The nerve cells of the anterior cornu are often greatly swollen and distended by this finely-granular, glistening material. Small, round masses of the same material are sometimes seen in the interior of the blood vessels. This glassy swelling of the nerve cells must be carefully dis-

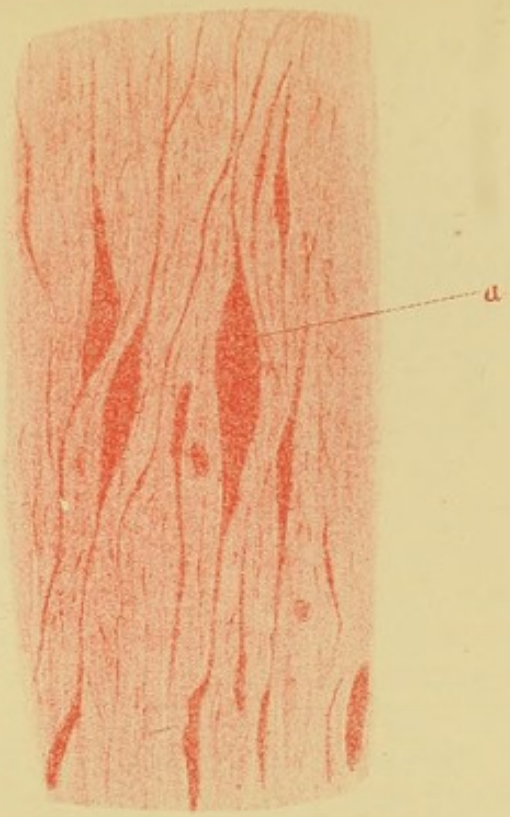


FIG. 55.

Longitudinal section through the Spinal Cord in Myelitis, showing hypertrophy of the axis cylinders. (After Charcot.)

a. Largest axis cylinder.

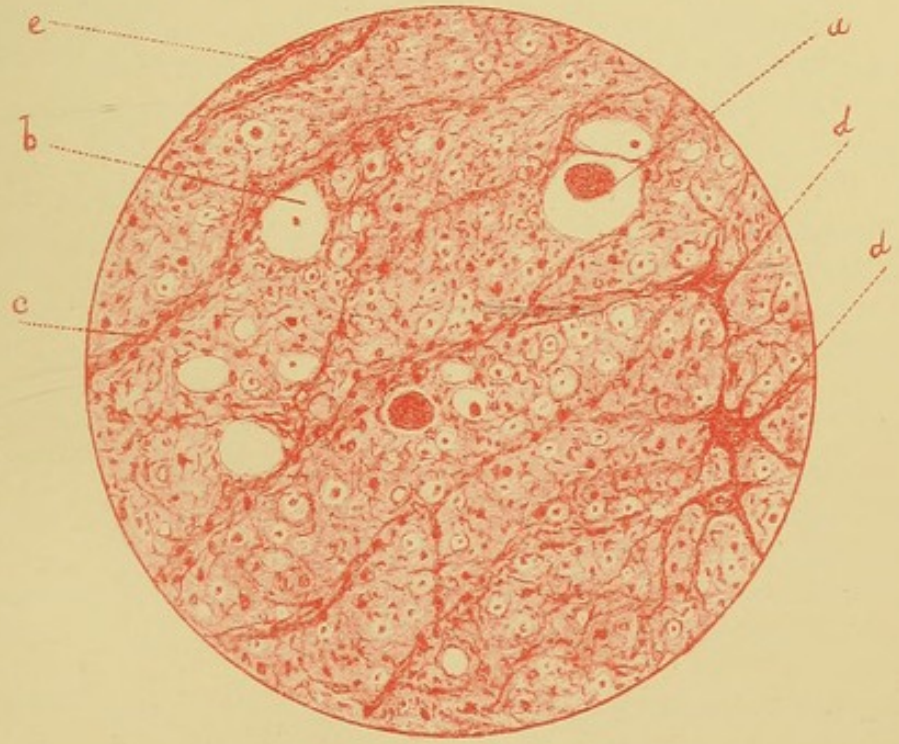


FIG. 56.

Transverse section through the dorsal region of the Spinal Cord in a case of Chronic Myelitis from compression. Stained with carmine, mounted in dammar, and magnified about 250 diameters.

a, Hypertrophied axis cylinder seen in transverse section; b, dilated nerve tube containing a normal axis cylinder; c, sclerotic tissue containing numerous nuclei; d, d, Deiter's cells; e, blood vessel with thickened walls.

B.B.del. ad. nat.

M.Lagan & Cumming, Lith. Edin.

tinguished from the inflammatory hypertrophy which Charcot has described. In the true inflammatory swelling the enlarged nerve cells stain deeply with carmine. In the glassy swelling the swollen cells are unaffected by that reagent. Osmic acid stains the hyaline material a deep blue black; methyl aniline a rose pink colour. In true hypertrophy the enlarged cells are unaffected by these reagents. The exact significance of this hyaline change is doubtful, but so far as I can form a judgment, it occurs in the great majority of cases of myelitis, both acute and chronic. Some good observers think that it results from the mode of preparation, and that it is due to the action of spirit. I have not been able to satisfy myself that it is an artificial product. The condition seems to be identical with that which was described by Professor Rutherford and Dr Batty Tuke under the term '*miliary sclerosis.*'

In the *second* stage, that of *exudation and softening*, the redness and hyperæmia have in great part or entirely disappeared, the softening is much more marked; the affected parts may be completely disorganised and liquified. Under the microscope the nerve elements are seen to be broken down: compound granule cells and oil globules are abundant.

In the *third* stage, that of *absorption and cicatrisation*, the softened material is gradually removed. The affected portions contract and become sclerosed. On microscopical examination, the compound granular corpuscles and oil globules have in great part disappeared: the connective tissue trabeculæ are thickened: the connective tissue cells are large and prominent: the blood vessels in the affected part are sometimes enormously dilated and their walls hypertrophied. Occasionally after the absorption of the softened tissue, a cyst remains.

In *sub-acute* and *chronic* myelitis the softening is much less than in the acute variety. The sclerotic changes are greater. The hypertrophy of the axis cylinders is usually well marked. These appearances are well shown in fig. 61, which represents a transverse section through the white matter of the cord, in an unpublished case of compression-myelitis.

§ 32. *Simple* (*non-inflammatory*) *softening.*—This condition probably depends in the great majority of cases upon defective blood supply. Any alteration of the walls, which narrows the vascular canal, will tend to produce it. In some cases plugging (*embolism* or *thrombosis*) of the vessels going

to the softened part can be demonstrated. But such cases constitute only a small proportion of the whole.

Erb states that simple (non-inflammatory) softening occasionally results from slow compression of the cord.

The microscopical examination of the softened parts is the only means by which we can distinguish simple softening from the inflammatory variety. Professor Erb gives the following concise account of the microscopic appearances in the two conditions:

'Whenever we find a large number of cells containing fat granules, tensely distended blood-vessels, numerous young cells, increase of the interstitial tissue, swollen axis-cylinders, etc., we are justified in assuming positively the inflammatory character of the process. On the other hand, when these characteristics are wanting, and we only find simply swollen and disintegrated nerve-fibres, ganglion-cells in a state of glassy swelling, a few cellular elements and fat-granule cells, and a small quantity of fatty detritus, we must diagnose a simple softening. Further and searching investigations on this point are, however, very much to be desired.' [1]

The softening is usually situated in the lumbar region of the cord, and is due, as Dr Moxon has so ably shown, to the peculiar manner in which the lower end of the cord receives its blood supply. Dr Moxon's explanation is so important, that I make no apology for quoting it in full:—

'Here I must take the liberty of drawing your attention to some very well-known anatomical points by which the curious fact that only the legs suffer in the caisson disease is, I think, quite clearly explained. I need scarcely remind you that whilst the spinal canal extends down into the sacrum, the spinal cord does not extend below the lower border of the first lumbar vertebra; but the spinal cord gives off nerves all the way down on each side, and the spinal canal on its part is pierced on each side with a series of openings all the way down, even to the lowest part of it, at pretty even intervals, the openings serving to let out the nerves that arise from the cord. The consequence of this arrangement is of course that the distance between the place of origin of the nerves of the cord, and their place of exit from the canal is short, as to the upper nerves, but increases much as to the lower nerves, so that the nerves run nearly horizontally to the neck and upper limbs, whereas to the loins and legs they run a long way down before reaching the dura mater, extending indeed for many inches, and forming a bunch of long, loose threads, something like a horse's tail, and known as the cauda equina. Now, the spinal cord is

[1] *Ziemssen's Cyclopædia of Medicine*, vol. xiii. p. 470.

suspended within the spinal canal in subarachnoid fluid, which entirely insulates it. And meantime surrounded by this liquid, and insulated by it, the spinal cord itself is out of reach of any blood-supply except such as can come to it from the brain above or else along the nerve-roots at the sides. And in fact the supply of this important part becomes, if I may so speak, one of Nature's difficulties. Let us see how the difficulty is met. The blood-supply to the spinal cord is carried out by slender vessels which come from the vertebral arteries within the cranium. There are three of these arteries—one on the front and two on the back of the cord; they are very slender, and yet have to run along its whole length. No arteries so small as these run so great a length elsewhere in the body, and pressure falls rapidly in minute arteries as the length of pipe increases, so that it becomes necessary to reinforce these slender vessels wherever possible; and advantage is taken of the nerve-roots to send up little reinforcing arteries along these. In the part of the cord corresponding to the neck, upper extremities, and trunk, where the nerve-roots are short, the reinforcing arteries are also short, and they reach and join and furnish blood to the spinal arteries—so that in this part of the cord every segment of it is supplied with blood from two directions, the anterior spinal artery bringing blood from above and the reinforcing artery from below. But when you approach the tip of the cord the supply from below becomes exceedingly precarious, and even apt to fail entirely, because upon the long strands of the cauda equina the small arteries are too narrow and too long to reinforce the cord with any certainty. But, at the same time, the supply from above has to be furnished with greater difficulty than in the upper regions of the cord, because the original anterior spinal artery is very far away, and the reinforcing arteries even in the lumbar region have to run considerably longer courses than they had in the cervical region.

Thus the tip of the cord has its blood-supply only from above, and deficiently even there, whilst the upper parts of the cord have a better sustained supply both from above and below; and this becomes especially the case upon the cauda equina itself, for here the arteries are exceedingly minute and uncertain in size on the several nerves. Hence we see that the tip of the spinal cord corresponding to the lower limbs and sphincters is much more weakly organised as to its circulation than are the upper parts of the cord.'[1]

§ 33. *The morbid histology of cerebro-spinal sclerosis.*—In this rare condition, which has been so ably described by Charcot, nodules or patches of degeneration (sclerosis) are scattered through the nerve centres (the brain, pons, medulla, cerebellum, and spinal cord), and are sometimes seen on the peripheral nerves. The nodules have a grey translucent appearance; on exposure to air they become pink in colour.

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[1] The Croonian Lectures on the Influence of the Circulation on the Nervous System.—*Lancet*, April 2, 1881, pages 529 and 530.

In the spinal cord the nodules present the greatest differences in distribution at different levels. For the most part, they are confined to the white columns, but they may involve the grey matter. They are usually 'indiscriminate;' but in some sections they are symmetrical; in others they very closely resemble secondary degenerations.

On microscopical examination the advanced patches are seen to be composed of wavy bundles of connective tissue, in which are embedded numerous compound granule corpuscles, and in some places crystals, which Frommann[1] thinks are composed of margarine. In the fully developed patches the nerve elements have entirely disappeared; the blood vessels are dilated, their walls may be thickened, numerous fatty globules and compound granule cells are seen adhering to their outer coats.

In the less advanced patches, and at the junction of the lesion with the healthy tissue, the connective tissue trabeculæ are increased, the connective tissue corpuscles (Deiters' cells) are large and prominent; the nerve tubes are being gradually surrounded by the connective tissue growth; their axis-cylinder processes are here and there hypertrophied.

As the lesion advances, the nerve tubes become destroyed; but even in the midst of the sclerotic tissue, axis-cylinders can often still be seen.

The following figures (57 to 70 inclusive) show the appearances of the cord lesion in a typical case, which I had under observation for several years.

The patient, a chemical worker, æt 28, was admitted to the Newcastle-on-Tyne Infirmary, under my care, on September 10th, 1874, complaining of, difficulty in walking, loss of power in the lower extremities, pain in the back, and giddiness.

His illness had commenced six years previously, and was apparently due to a severe blow on the back of the head. He had been a very heavy drinker.

He was a very large muscular man. There was no apparent loss of motor power. His movements were decidedly unsteady: the inco-ordination was, however, quite different from the inco-ordination of locomotor ataxia; it was, so to speak, of a 'coarser' kind, and involved the muscles of the trunk rather than the muscles of the lower extremities. In walking, the neck was held stiff, the head turned slightly to one side, and the eyes raised from the ground. On close observation, a fine jerking, rhythmical movement of the head could be observed. There was also occasional nystagmus.

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[1] Untersuchungen über die Gewebsveränderungen bei der multiplen Sklerose des Gehirns und Rückenmarks. Leipzig, 1878, Taf. ii., fig. 14.

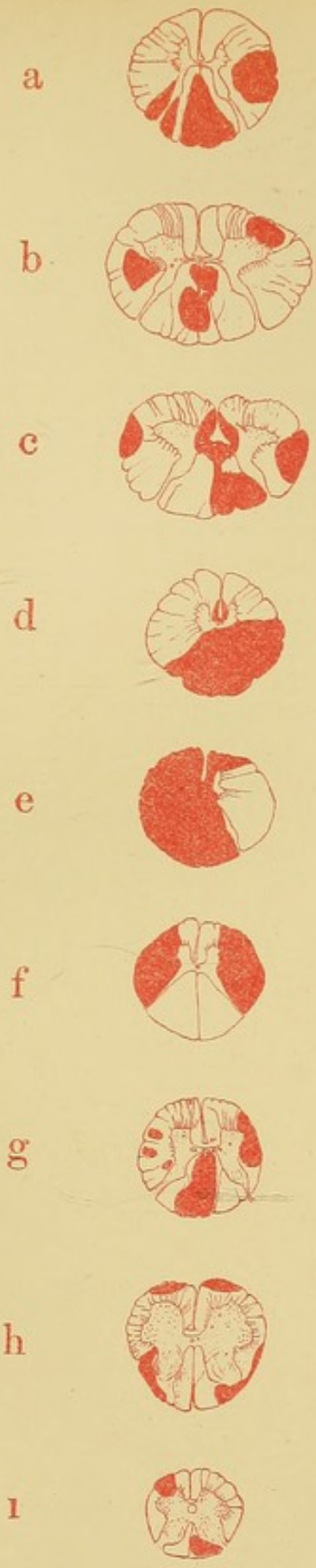


FIG. 57.

Transverse sections through the Spinal Cord, from a case of Cerebro-spinal Sclerosis, showing the position and extent of the sclerotic (deeply stained) patches at different heights. About one and a half times the natural size.

a, Upper cervical region; b, cervical enlargement; c. cervical enlargement; d, upper dorsal region; e, mid-dorsal region; f, mid-dorsal region; g, upper lumbar region; h, lower part of lumbar enlargement; i, filum terminale.

Sections a, b, c, d, e, g, h, and i are shown more highly magnified in succeeding figures.

B.B. del[t] ad. nat[m].

McLagan & Cumming, Lith. Edin[r].

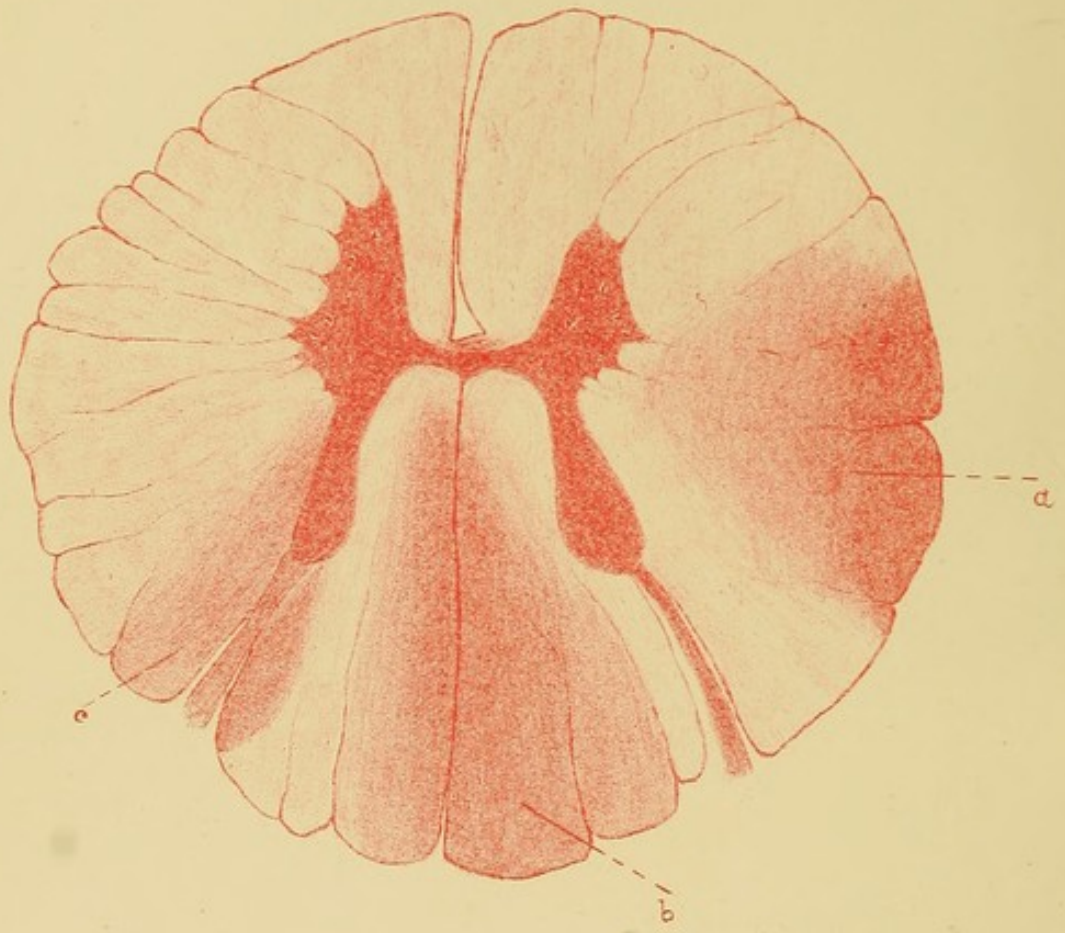


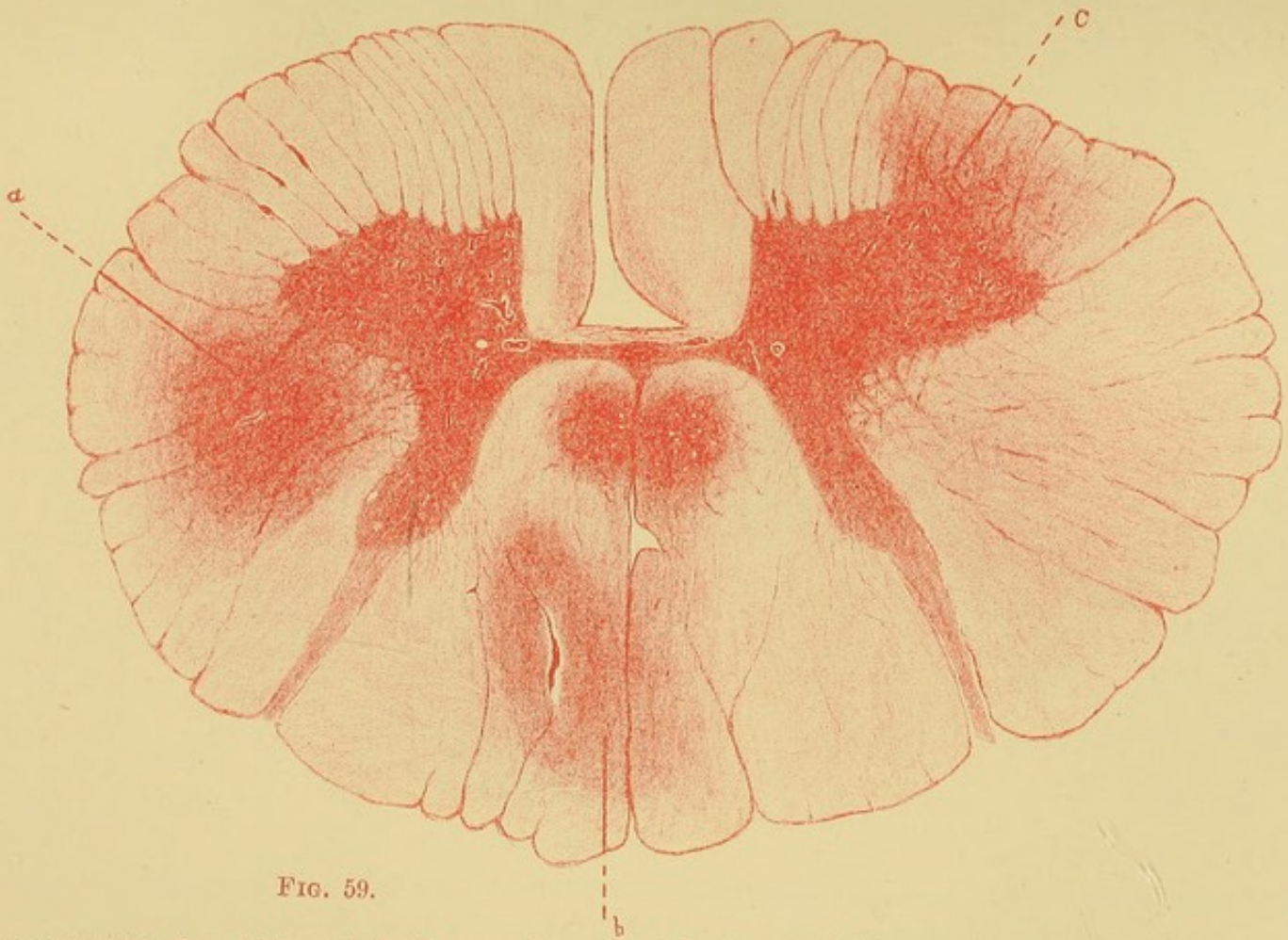
FIG. 58.

Transverse section through the upper cervical region in a case of Cerebro-spinal Sclerosis. Stained with osmic acid, mounted in dammar, and magnified about 10 *diameters.*

a, Patch of sclerosis in the right lateral column; b, patch of sclerosis in the posterior columns; c, patch of sclerosis in the left lateral column.

B.B. delt ad. natm.

McLagan & Cumming, Lith. Edinr.



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McLagan & Cumming, Lith. Edin.t

FIG. 59.

Transverse section through the cervical enlargement in a case of Cerebro-spinal Sclerosis. Stained with carmine, mounted in dammar, and magnified about 10 diameters.

a, Patch of sclerosis in the left lateral column; b, in the posterior column; and c, at the junction of the right lateral and anterior columns.



FIG. 60.

Transverse section through the cervical enlargement in a case of Cerebro-spinal Sclerosis, a little below the section shown in figure 59. *Stained with osmic acid, mounted in Farrant's solution, and magnified about* 10 *diameters.*

a, Symmetrical patch of sclerosis in each anterior column; b, symmetrical patch of sclerosis in each lateral column; c, patch of sclerosis in the posterior columns, the right being chiefly affected. The inner part of each anterior horn of grey matter is invaded.

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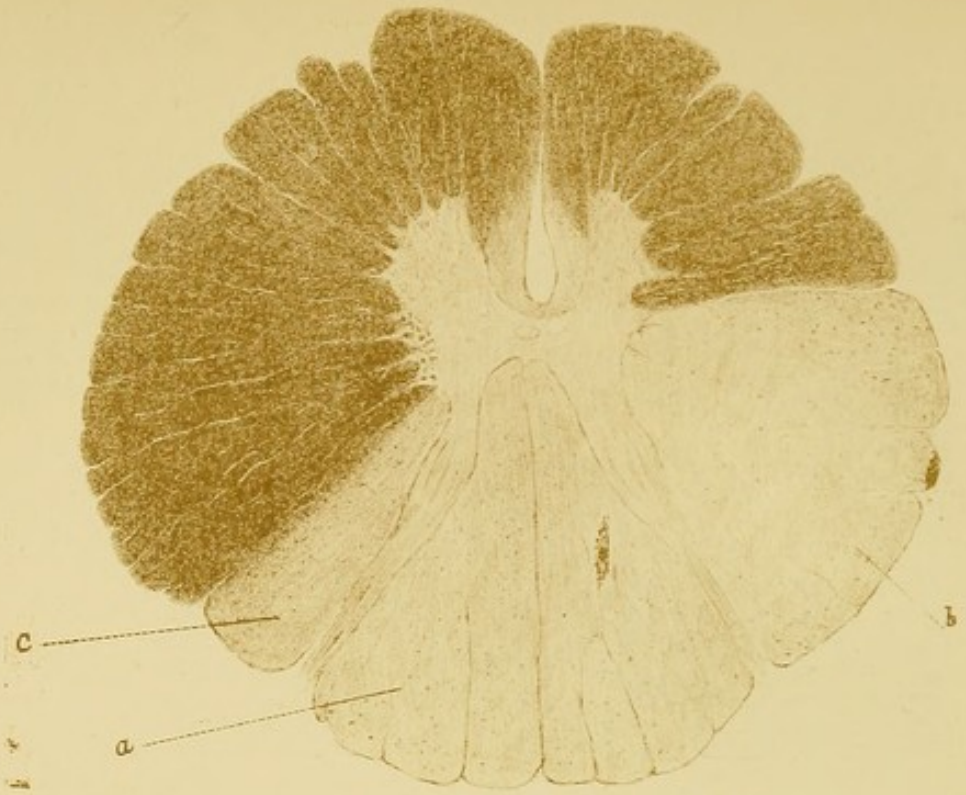


FIG. 61.

Transverse section through the upper dorsal region in a case of Cerebro-spinal Sclerosis, Stained with osmic acid, mounted in Farrant's solution, and magnified about 10 *diameters.*

The whole of the posterior columns (a); the greater part of the right lateral column (b); and a small part of the left lateral column (c), are invaded by the lesion.

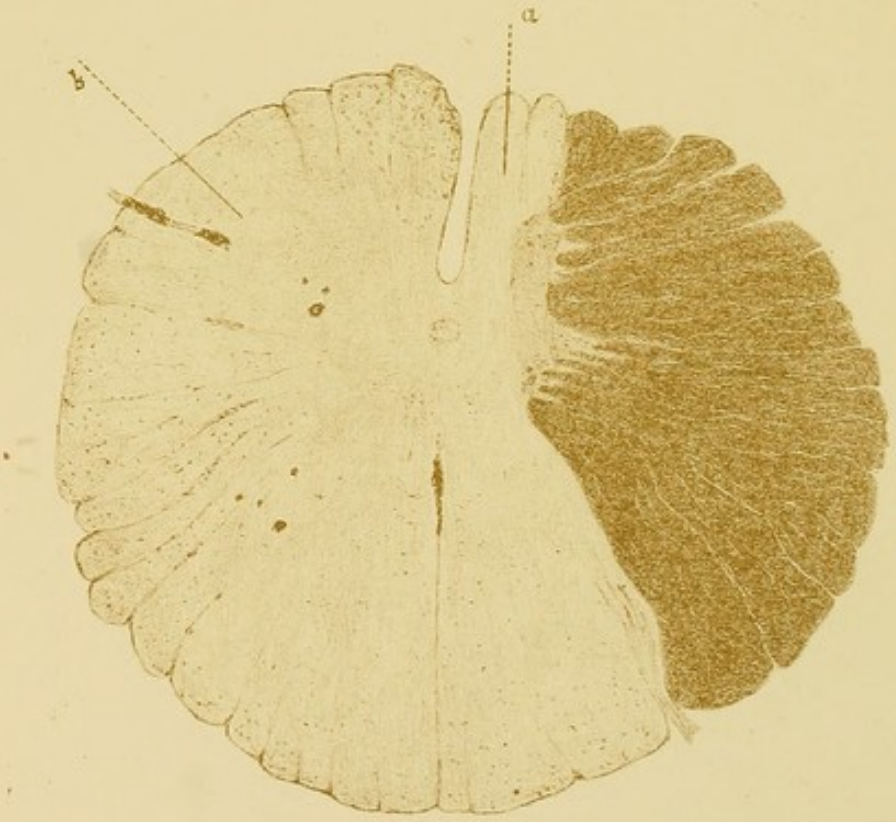


FIG. 62.

Transverse Section through the mid-dorsal region in a case of Cerebro-spinal Sclerosis. Stained with osmic acid, mounted in Farrant's solution, and magnified about 10 *diameters.*

The whole of the left half of the section (b), including the grey matter, is affected by the lesion. The posterior and anterior columns (a), on the right side, are also involved.

B.B. del.t ad. nat.m

M.cLagan & Cumming, Lith. Edin.r



FIG. 63.

Transverse section through the upper lumbar region in a case of Cerebro-spinal Sclerosis. Stained with carmine, mounted in dammar, and magnified about 10 *diameters.*

a, Patch of sclerosis in the right lateral column; b, in the left lateral column; and c, in the posterior columns.

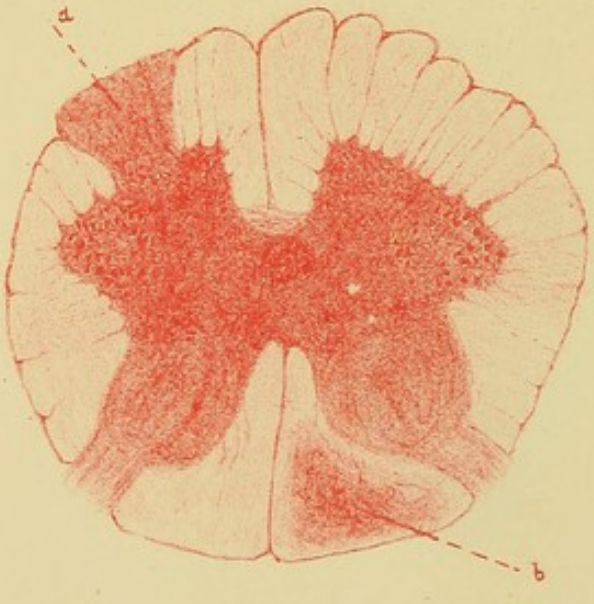


FIG. 65.

Transverse section through the lower part of the sacral region, in a case of Cerebro-spinal Sclerosis. Stained with carmine, mounted in dammar, and magnified about 10 *diameters.*

a, Patch of sclerosis at the junction of the left lateral and anterior columns; b, patch of sclerosis in the right posterior column.

The large size of the multipolar nerve cells at this part of the cord is well seen.

B.B. delt. ad. nat.m

McLagan & Cumming, Lith. Edin.r



FIG. 64.

Transverse section through the lower part of the lumbar enlargement in a case of Cerebro-spinal Sclerosis. Stained with osmic acid, mounted in Farrant's solution, and magnified about 10 *diameters.*

a, a, Symmetrical patches of sclerosis in the anterior columns; b, b, Symmetrical patches of sclerosis in the postero-external and posterior part of the lateral columns.

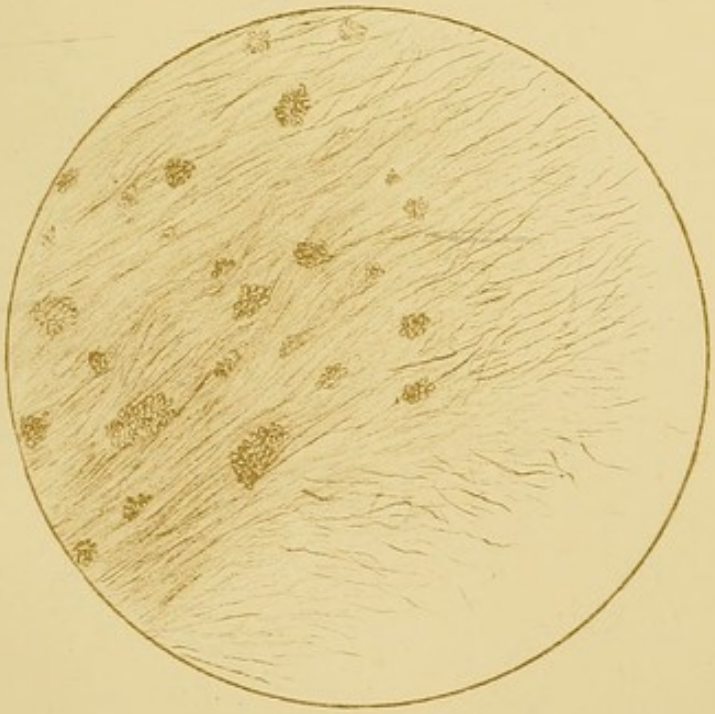


FIG. 66.

Longitudinal section through a patch of sclerotic tissue in a case of Cerebro-spinal Sclerosis. (Osmic acid and Farrant), × 250 *diameters.*

All the nerve tubes have disappeared, and are replaced by bundles of delicate connective tissue. Numerous fatty granules and (?) crystals are scattered throughout the section.

B.B. del.t ad. nat.m

McLagan & Cumming, Lith. Edin.r

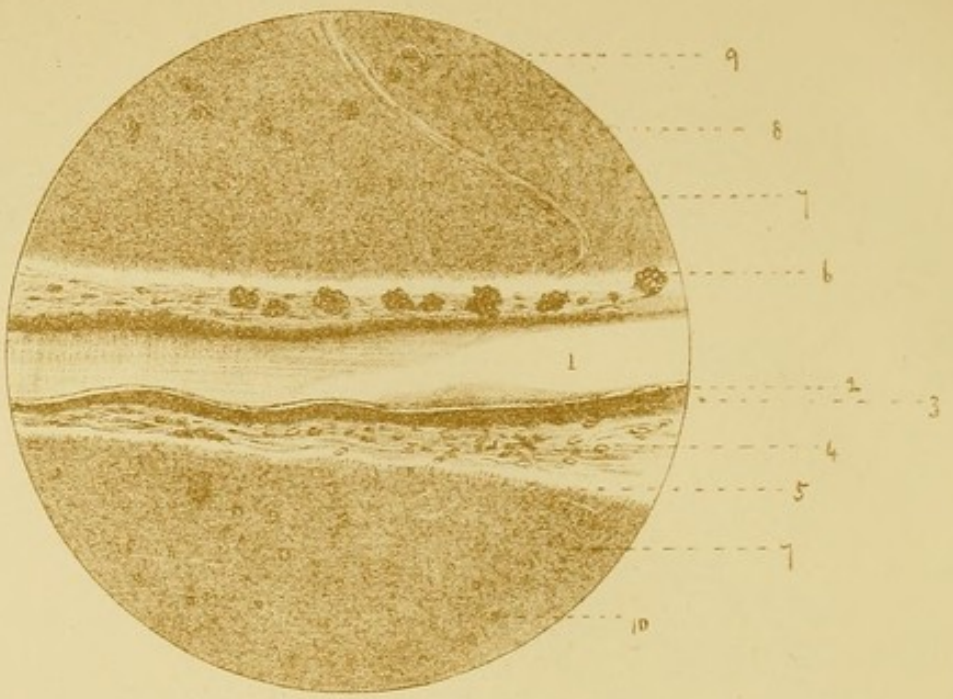


FIG. 67.

Transverse section through the medulla oblongata in a case of Cerebro-spinal Sclerosis showing compound granular corpuscles adhering to the outer coat of an artery. (Osmic acid and Farrant), × 200 *diameters.*

1, Longitudinally divided artery; 2, its inner, 3, its middle, and 4, its outer coats; 5, lymphatic space; 6, compound granular corpuscle; 7, 7, surrounding sclerosed tissue; 8, longitudinally divided nerve tube; 9, nerve cell; 10, leucocyte.



FIG. 68.

Transverse section through a blood vessel in the lateral column in a case of Cerebro-spinal Sclerosis, showing numerous fatty globules adhering to its outer coat. Stained with osmic acid, mounted in Farrant's solution, and magnified about 250 *diameters.*

a, Fatty globules in the outer coat of vessel; b, fatty globules in the surrounding nervous tissue. A few transversely divided nerve tubes, c, c, still remain.

B.B.del[t] ad. nat[m]

M[c]Lagan & Cumming, Lith. Edin[r]

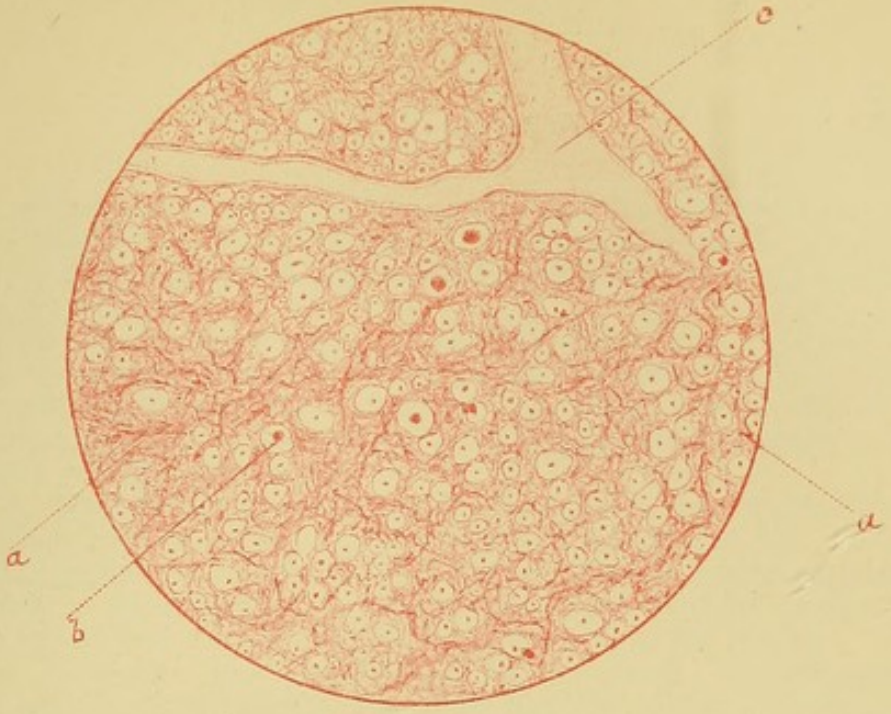


FIG. 69.

Transverse section through the lateral column of the cord, in a case of Cerebro-spinal Sclerosis, showing the early stage of the lesion. Stained with carmine, mounted in dammar, and magnified about 250 diameters.

a, a, Connective tissue between the transversely divided nerve tubes, which are, in places, widely separated: b, hypertrophied axis cylinder; c, longitudinally divided blood vessel.

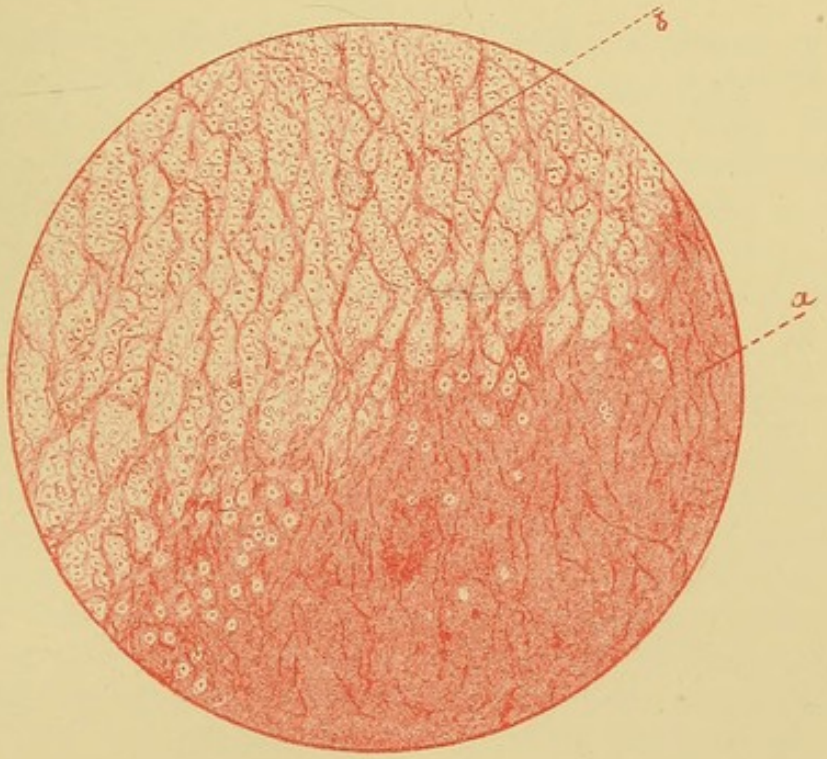


FIG. 70.

Transverse section through the lateral column, in a case of cerebro-spinal sclerosis, showing the junction of the sclerosed and healthy tissue. (Carmine and dammar), magnified about 50 diameters.

a, Sclerosed tissue; b, healthy tissue. Some transversely divided nerve tubes still remain in the middle of the sclerosed tissue.

B.B.delt. ad. nat.

McLagan & Cumming, Lith. Edin.

His memory was defective; he was very irritable and easily excited. He complained every now and again of double vision, but nothing abnormal could be detected in connection with the muscles of the eyeball, or with the fundus.

The speech had undergone a marked alteration during the past six months; and at the time of his admission, was thick and drawling. Pain in the back was at this time a prominent symptom; it was referred to the lower dorsal region, and the patient complained of tenderness on percussion over the same part.

There were (at that time) no other positive symptoms worthy of note.

On December 3d, 1874, he was discharged *in statu quo*.

From the time of his discharge until the date of his death, which took place in January 1881, I had frequent opportunities of visiting him in conjunction with my friend Dr Crease of South Shields. The progress of the case was steadily from bad to worse. The symptoms became highly characteristic, and confirmed in every particular the classical description which Professor Charcot has given us of the disease. My space will not, however, permit me to detail them now.

§ 34. *Intra-medullary hæmorrhage.* — This condition is rare. Its great causes seem to be disease of the vascular walls and increased blood pressure. It occasionally occurs in myelitis. Blood is sometimes extravasated from newly-formed vessels in the substance of a soft (gliomatous) tumour.

§ 35. *Intra-medullary tumours.*—Tumours arising within the cord itself are extremely rare. The forms met with are gliomata, sarcomata, tubercular, and syphilitic growths. Intra-medullary tumours are usually of small size. As the tumour grows, it compresses the cord, and generally produces more or less myelitis in its neighbourhood. Hæmorrhage may, as I have just remarked, result from rupture of the blood vessels in the tumour.

§ 36. *The symptoms produced by indiscriminate lesions.*

The effects (symptoms) of an indiscriminate lesion vary with the extent and position of the lesion in the transverse section, and to some extent with the nature of the morbid process. Myelitis produces destruction rather than irritation of the affected parts. The inflammatory process may involve; *(a)* the whole transverse section of the segment; or *(b)* it may destroy one lateral half; *(c)* in other cases it affects portions, but not necessarily symmetrical portions of both halves; *(d)* in other cases again it involves a portion only of one lateral half. I will now describe the symptoms which result from some of these types.

§ 37. TOTAL TRANSVERSE LESIONS.

The symptoms which result from *acute destruction of the whole transverse section* of our segment are diagrammatically represented in fig. 71. They are as follows:—

1. Paralysis of its muscular area and all the muscular areas which are situated below it.

2. Rapid atrophy, the 'reaction of degeneration,' and complete absence of all reflex movements in the muscular area of the segment.

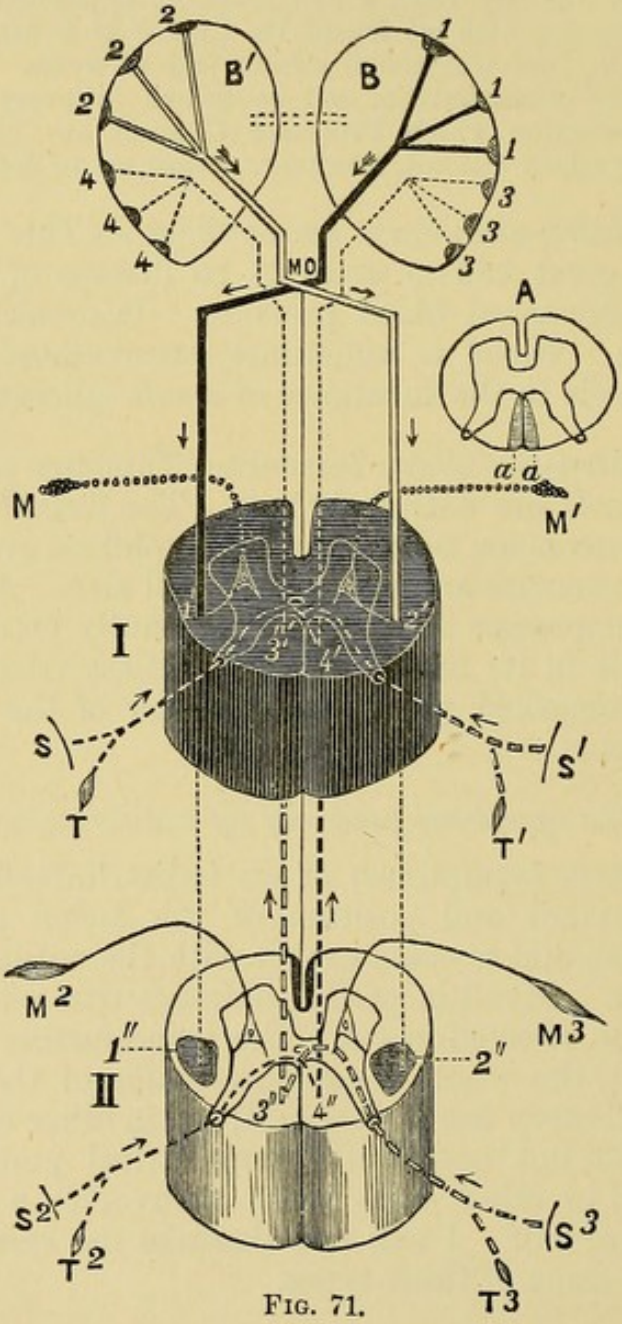


FIG. 71.

3. The trophic condition and electrical reactions of the muscles, supplied by segments below the lesion, are normal. The reflexes passing through the inferior segments are exaggerated. With the occurrence of secondary descending degeneration, the paralysed muscles, supplied by inferior segments, become tense and rigid.

4. The common and special sensibility (muscular sense) of the paralysed muscles is diminished or abolished.

5. Complete anæsthesia of the sensitive area of our segment, and of the sensitive areas of all the segments which are situated below it.

6. A narrow band of hyperæsthesia at the upper level of the lesion. This narrow band of hyperæsthesia is supposed to be due to irritation of the sensory nerve fibres at the upper level of the lesion. When the lesion is situated in the dorsal region of the cord, the band of hyperæsthesia extends round the trunk in the form of a belt. The patient feels as if a tight band were drawn round his body. The term, 'girdle sensation,' has been given to this symptom.

DESCRIPTION OF FIG. 71.

Diagrammatic representation of the effects of an acute total transverse lesion of the Spinal Cord.

I. The segment which is destroyed by the lesion; II. A segment of the cord below the lesion; B, the right, and B′ the left cerebral hemispheres; 1, 1, 1, motor centres in the right cerebral hemisphere from which the motor tract proceeds to the muscles M and M^2 on the left side of the body.—For the sake of clearness, the direct pyramidal tracts have been omitted—2, 2, 2, motor centres in the left cerebral hemisphere from which the motor tract proceeds to the muscles M^1 and M^3 on the right side of the body; 3, 3, 3, sensory centres in the right cerebral hemisphere to which the sensory tract proceeds from S, T, and S^2, T^2 the sensory areas on the left side of the body; 4, 4, 4, sensory centres in the left cerebral hemisphere to which the sensory tract proceeds from S′, T′ and S^3, T^3, the sensory areas on the right side of the body.

Motor impulses passing downwards through 1 and 2 are arrested at the seat of the lesion. The muscles M and M′ supplied by the diseased segment are in a state of acute atrophy. The muscles M^2 and M^3 supplied by segment II. retain their normal bulk. Reflex impulses cannot pass through the diseased segment. Reflex impulses passing through segment II. are exaggerated, for the reflex arc is perfect, and the lateral columns above this segment are degenerated. Sensory impressions passing from all the parts below the lesion are arrested in the diseased segment. Secondary descending degeneration of the crossed and direct pyramidal tracts below the lesion, is indicated by the dotted lines and is shown in segment II. Secondary ascending degeneration above the lesion is indicated by the dotted continuations of the sensory tracts and is shown at *a*, *a* in fig. A.

Note to Figs. 71 and 73.

In these figures a total sensory decussation in the cord is shown. As regards this point see page 23.

Where the lesion involves the lumbar or cervical segments, the band of hyeræsthesia is not circular, but is distributed more or less longitudinally over the limbs in accordance with the particular segment which happens to be affected. The distribution of the hyperæsthesia, in such cases, will be readily understood, by referring to the sensory functions of individual segments (see page 137). In fig. 72 I have diagrammatically represented the skin symptoms which result from a total transverse lesion.

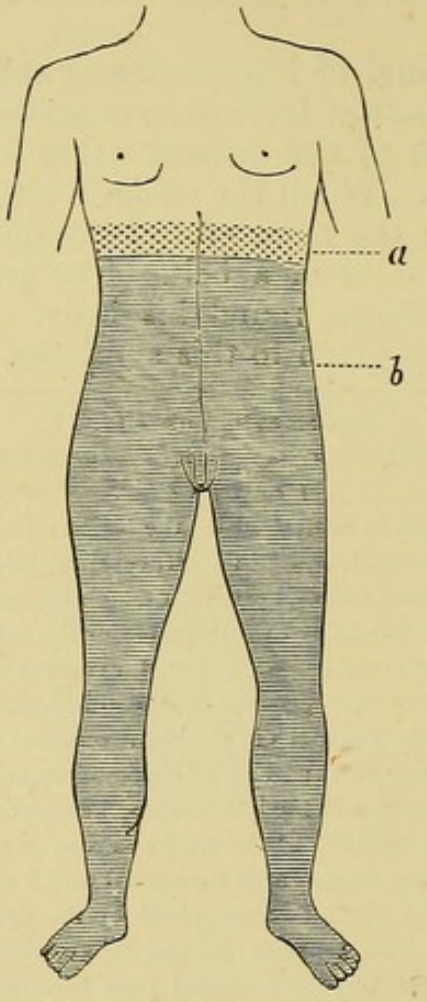


FIG. 72.

Diagrammatic representation of the skin symptoms in a total transverse lesion of the dorsal portion of the Spinal Cord.

The horizontal shading signifies anæsthesia of the skin; the dotted shading hyperæsthesia; a, hyperæsthetic zone at the upper level of the lesion; b, b′, anæsthetic below the lesion.

7. There is vaso-motor paralysis below the lesion. Secondary descending degeneration of the pyramidal tracts (crossed and direct) below the lesion, and secondary ascending

degeneration of the postero-internal columns above the lesion are gradually established.

8. Trophic disturbances in the skin of the sensitive area of the segment sometimes occur.

9. The functions of the bladder and rectum are often seriously deranged. The exact character of the vesical and rectal derangement depends upon the segment which happens to be affected. If the lower portion of the cord, in which the vesical and rectal reflex centres are situated, is destroyed, paralysis of the sphincters, ammoniacal urine, cystitis, etc., are developed. The urinary disturbances will afterwards be more minutely considered (see page 117).

Where the lesion is above the lumbar region, especially when the upper dorsal or cervical segments are involved, *priapism* may occur. This condition is probably due to irritation of 'excitor' fibres which pass from the cerebrum to the sexual reflex centre.

Lesions of the upper cervical region are sometimes attended with *hyper-pyrexia.*

The symptoms of a chronic transverse lesion will be referred to in speaking of the effect of slow compression of the cord (see page 67).

§ 38. UNILATERAL LESIONS.

The more important symptoms which follow *acute destruction of one half of our segment* are diagrammatically represented in fig. 73. They are as follows:—

1. Paralysis of the muscular area supplied by the anterior root arising from the affected half segment; and of all the muscular areas on the same side of the body which are situated below it.

2. The paralysed muscles supplied by the affected half segment are flaccid; they undergo rapid atrophy, and present the 'reaction of degeneration.'

3. The paralysed muscles supplied by half segments below the lesion do not undergo rapid atrophy. With the occurrence of secondary descending degeneration they become tense and rigid.

4. Theoretically we should expect to find the reflexes passing through the affected half segment, abolished; and the reflexes, passing through the inferior half segments on the same

side of the body, increased. But the exact condition of the reflexes in unilateral lesions has not yet been sufficiently investigated to allow any very positive statement to be made.

5. The common and special sensibility (muscular sense) of the paralysed muscles is diminished or abolished.

6. There is vaso-motor paralysis below the lesion on the same side.

7. Anæsthesia of the sensitive area of our segment. The anæsthesia is bilateral; for the sensory fibres, which enter by the posterior nerve root of the affected side, are 'cut' before their decussation, and the sensory fibres, which enter the segment by the posterior root of the opposite side, are 'cut' after they

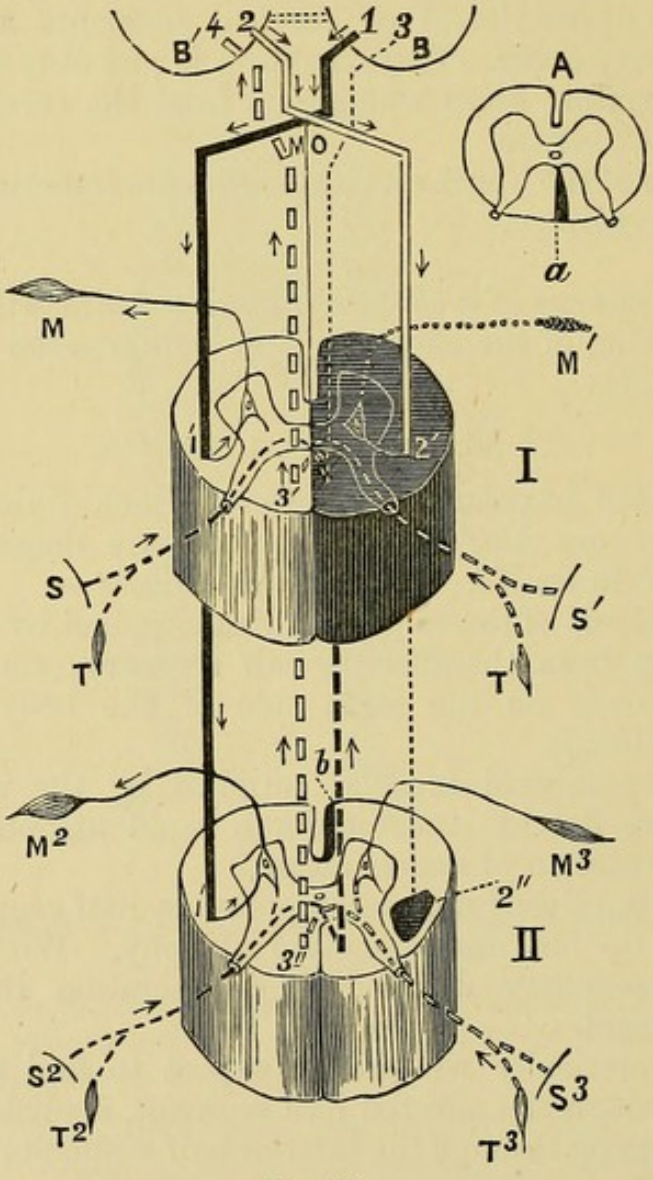


Fig. 73.

have decussated. When the lesion is situated in the dorsal region the band of anæsthesia is circular (see fig. 74); where the cervical or lumbar segments are involved, it is, for the reasons already given in speaking of total transverse lesions, more or less longitudinal.

8. Anæsthesia of the sensitive areas of all inferior half segments on the opposite side of the body.

9. Hyperæsthesia of the sensitive areas of all inferior segments on the same side of the body. The exact cause of the hyperæsthesia is not known.

10. A narrow band of hyperæsthesia corresponding to the upper level of the lesion on the same side of the body is usually described. It is supposed to be caused by irritation of the sensory fibres which enter the cord at the upper level of the lesion. The band of hyperæsthesia is circular when the lesion is situated in the dorsal region (see fig. 74); where the cervical or lumbar segments are involved, it is, for the reasons given in speaking of total transverse lesions, more or less longitudinal.

DESCRIPTION OF FIG. 73.

Diagrammatic representation of the effects of an acute unilateral transverse lesion of the Spinal Cord.

I. A segment of the spinal cord, the right half of which is destroyed by the lesion; II. a segment of the spinal cord below the lesion; 1 motor tract proceeding from the right hemisphere of the brain, B, to the muscles, M, and M^2, on the left side of the body (for the sake of clearness the direct pyramidal tracts have been omitted); 2, motor tract proceeding from the left hemisphere of the brain, B′ to the muscles M^1 and M^3, on the right side of the body; 3, the sensory tract proceeding to the right hemisphere of the brain from S, T, and from S^2, T^2, the sensitive areas on the left side of the body; 4, the sensory tract proceeding to the left hemisphere of the brain from S^1, T^1, and from S^3 T^3, the sensitive areas on the right side of the body.

Motor impulses passing from the left hemisphere of the brain along 2 are arrested at the seat of the lesion. There is, therefore, paralysis of the muscular areas, M^1 and M^3. The motor tract below 2′ is degenerated. The muscular area M^1 is acutely atrophied. Reflex, impulses passing from S^1, T^1 to M^1 are abolished. The paralysed muscles M^3 below the lesion are not atrophied. Reflexes passing from S^3 and T^3 to M^3 are exaggerated, for the reflex arc which passes through segment II. is intact, and the lateral column above segment II. is degenerated; the degeneration of the motor tract below 2′ is indicated by a dotted line, and is seen in segment II. Sensory impressions from the sensitive areas, below the lesion, and on the opposite side of the body, S^2 T^2, are arrested at the seat of the lesion. Sensory impressions from S, T, and S^1, T^1, the sensitive area of the affected segment on both sides of the body are also 'blocked' by the lesion. The main sensory tract proceeding to the right hemisphere of the brain is degenerated above the lesion as shown in fig. A. The arrows indicate the course of the nerve currents.

The character and distribution of the sensory disturbance in a unilateral lesion are well shown in fig. 74, which is copied from Erb.

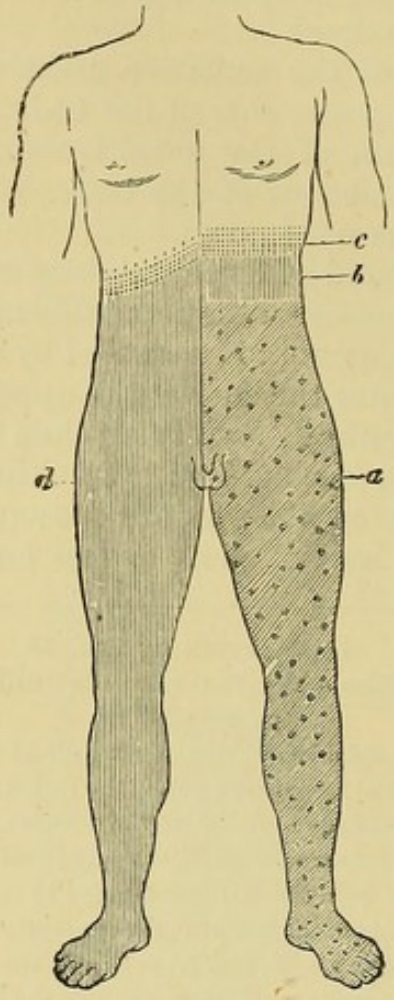


FIG. 74.

Diagrammatic representation of the skin symptoms in an unilateral lesion of the dorsal portion of the Spinal Cord on the left side. (After Erb).

The diagonal shading signifies motor and vaso-motor paralysis; the vertical shading signifies anæsthesia of the skin; the dotted shading indicates hyperæsthesia of the skin; c, band of hyperæsthesia on the left side of the body at the upper level of the lesion; b, band of anæsthesia on the left side of the body corresponding to the vertical extent of the lesion; a, hyperæsthesia below the level of the lesion; d, anæsthesia, in the parts below the level of the lesion on the opposite (right) side of the body.

11. Derangements of the vesical and rectal reflexes may occur. Trophic disturbances in the skin of the sensitive area of the affected half segment will occasionally be observed.

Secondary descending degeneration of the pyramidal tracts (crossed and direct) on the same side below the lesion, and secondary ascending degeneration of the postero-internal column on the same side above the lesion, will of course follow.

The fibres of the posterior root which enter the affected half segment and their upward prolongations in the opposite postero-internal column will also degenerate; but the fibres, entering by any one posterior root, form such a small proportion of the whole sensory tract that this ascending degeneration on the opposite side to the lesion is not perceptible.

§ 39. PARTIAL (NON-SYSTEM) LESIONS OF ONE OR BOTH HALVES OF THE SEGMENT.—In cases of partial destruction, the symptoms, of course, vary with the part of the transverse section which happens to be affected. After the full details already given as to the effects of lesions of the different tracts, it is unnecessary to indicate all the possible effects of indiscriminate lesions of this description. I should however say that there is a well marked type of myelitis, in which the inflammatory process affects the central grey matter, which must not be confounded with the system disease (acute inflammation of the anterior horn of grey matter). In *acute central myelitis* the anterior horns are, it is true, usually involved,—and this seems to be particularly the case where the lesion affects the lumbar enlargement,—but the central grey matter and the posterior cornua are affected too. There are, in consequence, well marked sensory disturbances. The bladder and rectum are usually profoundly affected, for the lesion generally involves the segments through which the vesical and rectal reflexes pass. There is often some trophic alteration in the skin to which the posterior nerve roots of the affected segments are distributed.

§ 40. EXTRA-MEDULLARY LESIONS.

I have now fully described the symptoms which result from lesions of the different parts of our spinal segment. So far, I have been speaking of the cases in which the morbid process originated in the cord itself. I shall next consider the second great group of cases, viz., those in which the lesion commences outside the spinal cord, and in which the derangement of the cord is a secondary, and, as it were, an accidental process.

The chief affections comprised in this group are: (*a*) spinal meningitis; (*b*) Pott's disease of the vertebræ; (*c*) tumours in the spinal canal springing from the bones, membranes, or nerve roots; (*d*) traumatic injuries, such as fractures or dislocations of the vertebræ, wounds of the spinal cord; and (*e*) hæmorrhage into the spinal canal.

The clinical picture which these cases present is generally a complicated one, for it is composed partly of symptoms due to the primary morbid condition (the disease of the vertebræ, membranes, etc.), and partly of symptoms which result from derangement of the spinal functions. It is only by keeping in view this double composition, so to speak, of the symptoms, and by clearly understanding the *manner* in which these lesions affect the nervous structures, that it is possible to comprehend these cases intelligently.

§ 41. *The spinal symptoms which are met with in extra-medullary lesions* may be due to: (1) pressure upon the spinal cord; (2) pressure upon the spinal nerve roots; (3) extension of the primary morbid process (inflammation, for example, in the case of meningitis) to the spinal cord or nerve roots respectively.[1]

The symptoms which result from inflammation of the different parts of the spinal segment have been already so fully detailed, that I need add nothing to that description further than to say, that the inflammatory process may be either acute or chronic; that in some cases it involves the whole thickness of the cord, while, in others, it is the peripheral layer which is chiefly affected. I must, however, particularly direct your attention to the effects of compression of the cord, roots, and membranes.

Speaking generally, the effects of pressure on nervous tissue vary with:—the function of the affected part; the severity of the pressure; and the rapidity with which it is exercised. Rapid pressure usually produces acute destruction, or acute inflammation, quickly passing on to destruction of tissue.

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[1] The pain which results from irritation of the sensory nerve filaments in the periosteum and membranes is, of course, a nervous phenomenon, but it is usually the *direct* result of the primary morbid condition (disease of the bones, membranes, etc.), and hence has not been included under the nervous symptoms which are due to extension of the morbid process, and to pressure.

Slow pressure is attended by gradual interruption of function, chronic inflammation, or atrophy of the compressed part.

§ 42. RAPID COMPRESSION OF THE SPINAL CORD.

This condition usually results from fractures or dislocations of the vertebræ. The effects of rapid compression of our segment by a displaced vertebra would be immediate interruption of its function, both as a conducting medium and a spinal centre. Acute inflammation, rapidly passing into destruction of its tissue, would speedily follow. The symptoms would, therefore, be those of an acute transverse myelitis. Death very generally follows. (See page 58.)

But, in addition, the compression would probably also involve the nerve roots arising from the segment, and the adjacent portions of the membranes. Other symptoms might result from these conditions, to which I will presently refer.

§ 43. SLOW COMPRESSION OF THE SPINAL CORD.

This condition is commonly met with in practice. It may result from the pressure of diseased bones, inflammatory products, or thickened membranes, as in Pott's disease of the vertebræ, and the various forms of meningitis; or, it may be due to the pressure of cancerous or other tumours.

Slow compression of our segment causes gradual interruption of its functions, both as a conducting medium and as a spinal centre. Motor conduction is first impaired; sensory conduction is affected later; chronic inflammation, and slow destruction of the segment, gradually supervene; secondary ascending and descending degenerations follow.

§ 44. *The symptoms, therefore, which result from slow compression of the cord itself* are:

1. Motor weakness in the muscular area of the compressed segment, and of all segments which are situated below it. As the compression increases, the muscular weakness becomes greater, and complete paralysis may be finally developed. As a matter of experience, we know that the motor weakness is more marked in the muscular areas of the compressed segment and of the segments which are situated immediately

below it, than in the muscular areas of segments far removed from the seat of lesion. Where, for example, the cervical enlargement is compressed, as in cases of hypertrophic cervical pachy-meningitis (see fig. 75), the loss of power is much more marked in the upper than in the lower extremities. The explanation is supposed to be that the motor fibres which are about to leave the cord are more superficially placed, and hence are more seriously affected by the pressure than those destined for distant segments.

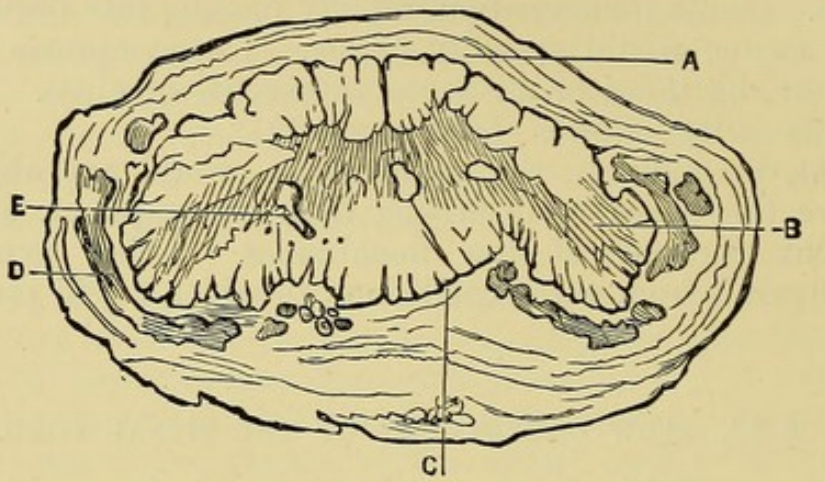


FIG. 75.

Transverse section of the cervical enlargement of the Spinal Cord in a case of hypertrophic cervical pachy-meninigitis. (After Joffroy).

A, Hypertrophied dura mater. B, Nerve roots traversing the thickened meninges. C, Pia mater confounded with the dura mater. E, Section of two canals newly excavated in the grey substance.

The nutrition of the weakened or paralysed muscles is not much affected;[1] there is no reaction of degeneration; increase of the reflexes below the seat of the compression occurs; the legs are often rigid; in short the condition of spastic paraplegia is present.

2. Anæsthesia of the sensitive areas of the compressed segment and of the segments which are inferior to it. As I have previously remarked, the anæsthesia is developed later than the paralysis.

3. As the segment becomes more and more compressed, its tissues become disorganised, and chronic inflammatory

[1] The muscles supplied by rapidly compressed anterior roots will undergo rapid atrophy, and will present the 'reaction of degeneration.' (See page 105).

changes are developed. Its muscular area becomes atrophied, and its reflexes are gradually abolished.

Secondary ascending and descending degenerations follow.

But, in addition to the symptoms which result from compression of the cord itself, others due to pressure on the nerve roots, and the associated affections of the spinal membranes are almost always present. These symptoms are of great diagnostic value, and it is important to remember that they are usually developed before the symptoms, which result from the compression of the cord itself, have become prominent.

The symptoms which result from compression of the nerve roots are as follows :—

§ 45. *Pressure on the posterior nerve roots* of our segment will interrupt the passage of peripheral impressions from the region of the body to which the fibres of those roots are distributed; and will therefore cause anæsthesia in the sensitive area of the segment, and abolition of its reflex movements.

Where the pressure is incomplete, and where irritation of the compressed fibres is produced—conditions which usually obtain in the early stages of the case—hyperæsthesia, or a combined condition of hyperæsthesia and partial anæsthesia may be observed.

Severe pressure may not only arrest conduction through the posterior roots, but may also produce irritation of their component fibres. Now, irritation of a sensory nerve causes stimulation of the sensory perceptive cerebral centre to which it proceeds, and the impression thus generated is referred by the mind, in accordance with the law of 'eccentric projection,' to that portion of the periphery to which the terminations of the irritated nerve are distributed; in other words, to that part of the body from which the sensory impressions which normally pass through the affected nerve proceed. Hence it is, that compression of the posterior roots of our segment may not only cause anæsthesia of its sensitive area, but may also produce painful sensations in the anæsthetic parts. The pains are usually of a sharp shooting character. This condition of combined anæsthesia and pain, to which the term '*anæsthesia dolorosa*' has been given, *is of considerable diagnostic value,* for it shows that the lesion is situated outside the *grey matter* of the spinal cord. Anæsthesia

dolorosa may result from irritation of the posterior root-fibres in the postero-external column, and it is in fact frequently met with in locomotor ataxia; or, it may be due to pressure on the posterior roots, or sensory nerve trunks. Hence its value as a localising symptom in the cases we are now considering.

Irritation of the posterior roots may also produce reflex muscular contraction (spasms, etc.), in the muscular area of the segment. This is probably, in part at least, the cause of the jerkings, spasms, and contractures, which occur in cases of spinal meningitis.

§ 46. *Compression of the anterior roots* of our segment produces diminution or total loss of motor power (paralysis) in its muscular area. Where the motor fibres are irritated, twitchings, spasms, and contractures may occur. This is another cause of the muscular spasms and contractures which are so characteristic of spinal meningitis. When the compression is sufficiently great to shut off the trophic influence of the multipolar nerve cells of the anterior cornua, marked atrophy of the paralysed muscles is present. When the compression is quickly established the atrophy may be *rapid*, and the 'reaction of degeneration' will then be observed. When the motor conduction is completely arrested, the reflexes in the muscular area of the segment are abolished; in minor degrees of compression the reflexes may be retained.

§ 47. *The symptoms which result from pressure on the spinal membranes.*—The spinal membranes, more particularly the dura and pia, are richly supplied with sensory nerves. Compression of the membranes is, therefore, in most cases attended with pain, which is referred to the region of the spinal column. Where the compressed membranes are inflamed the pain is proportionately greater. The pain of meningitis is increased on movement of the spinal column.

CHAPTER III.

METHOD OF CASE-TAKING — SUMMARY OF SYMPTOMS MET WITH IN DISEASES OF THE SPINAL CORD—THE CLINICAL EXAMINATION OF A CASE OF SPINAL CORD DISEASE—GENERAL PLAN OF THE DIAGNOSIS—GENERAL PLAN OF THE PROGNOSIS—GENERAL PLAN OF THE TREATMENT.

I HAVE now described the leading facts in the pathology of the spinal segment, and have pointed out the alterations in function which result from lesions of its different parts. I will next sketch out the 'method of case-taking' and the 'plan of examination' which I am in the habit of following in the clinical investigation of cases of spinal cord disease; and this will afford a convenient opportunity for summarising the symptoms which occur in this group of cases.

§ 48. But first, allow me to insist upon the great importance of system and method in clinical examination. A methodical plan of case-taking greatly facilitates the examination of any organ, but it is especially important in the case of the spinal cord, the functions of which are so numerous and so complicated. Indeed, the difficulties which students experience in investigating the affections of the spinal cord, at the bedside, are in great part due to the fact, that they enter upon the examination of the case without any well-considered and definite plan of study.

Various methods of 'case-taking' are in use, and all of them have some advantages. I do not pretend to claim that the following plan is better than the rest, but I can at least say, that it is simple and systematic, and that I have found it work well in practice.[1]

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[1] A methodical plan of case-taking is, of course, essential for scientific purposes, and it would be most advantageous if physicians could agree upon a uniform system.

§ 49. METHOD OF CASE-TAKING.

A. PRELIMINARY FACTS:—Name—Age—Sex—Married or Single—Occupation—Full Postal Address—Date of Admission to Hospital.

B. COMPLAINTS:—(The Symptoms which bring the patient to consult the physician).

C. THE HISTORY:—

(1.) Of the Present Illness:—The exact date of its commencement. The exact mode of commencement. The supposed cause of the attack. The exact character of the symptoms; the order of their appearance; and the treatment which has been adopted, up to the time when the patient comes under observation. (In acute cases take the temperature).

(2.) The Health History prior to the commencement of the present attack:—Especially a history of disease or injury likely to be followed by disease of the spinal cord. The habits, mode of life, and general surroundings of the patient.

(3.) The Family History:—Especially the occurrence of nervous affections amongst near relatives.

D. THE PRESENT CONDITION:—(The date at which the examination is made should be stated.)

(1.) The Physiognomy of the Case:—The description of any striking abnormal appearances. The facial expression. The attitude. The gait. The general state of nutrition, etc.

(2.) The Clinical Examination of the Spinal Cord:— *Motor Functions:* The condition of the muscles supplied by the different spinal nerves (muscular areas of the different segments) with respect to:—

1. Voluntary motor power.
2. State of nutrition.
3. Irritability. (Faradic, galvanic and mechanical.)
4. Tonicity.
5. Reflex movements. (Superficial, deep, and organic.)
6. Co-ordination and the muscular sense.

Sensory Functions: The condition of the sensory areas of different segments, with respect to:—

1. Subjective sensations:—(Pain, numbness, tingling, formication, heat, cold, etc.) Their exact character and distribution.
2. Objective sensations:—(Sensibility to touch, pain, and temperature is to be tested separately.)

The Vaso-motor and Trophic Condition of the Skin and Joints.

The Condition of the Spinal Column:—Its conformation, the presence of irregularities or curvatures. The presence or absence of pain on percussion or movement of the vertebræ. (The hot sponge test.)

(3.) The Condition of the other parts of the Nervous System.

(4.) The Condition of the Alimentary, Circulatory, Respiratory, Genito-urinary, and Integumentary Systems.

E. THE DIAGNOSIS.

F. THE PROGNOSIS.

G. THE TREATMENT:—Hygienic, dietetic, medicinal (general and local).

H. THE SUBSEQUENT COURSE OF EVENTS:— The progress of the case during the patient's stay in hospital: The mode of termination: The date of termination: In fatal cases the record of the *post-mortem* examination, and an account of the microscopical appearances of the spinal cord.

SUMMARY OF THE CHIEF SYMPTOMS.

PRELIMINARY FACTS.

§ 50. AGE.—Disease of the spinal cord may occur at any period of life, but some affections are much more common at one period than at another.

Childhood.—Acute inflammation of the anterior cornu *(Polio-myelitis anterior acuta)* is very common in children (the period of greatest frequency being between the ages of one and four); and is almost confined to early life. *Pseudo-hypertrophic paralysis* appears in the vast majority of cases during the first few years of life. *Inflammatory affections of the membranes*, especially the tubercular form, are frequent. Amongst the rarer diseases of the cord, *reflex paraplegia* is usually seen in young subjects. A peculiar form of *spastic paralysis*, of which I have seen several examples, occurs in young children, and appears to be congenital. A very rare, congenital form of *locomotor ataxia* has been described by Friedreich. *Disseminated sclerosis* is occasionally met with.

Youth and Early Adult Life.—Organic affections of the cord itself are not so frequent during youth as during the earlier and later periods of life. *Compression of the cord*, the result of Pott's disease of the vertebræ, is most common at this period. *Hysterical paraplegia*, and '*spinal irritation*,' occur in females. The functional derangements which have been classed under the term '*spinal nervous weakness*,' are met with amongst young males. *Meningitis* and *myelitis* are relatively common. *Cerebro-spinal sclerosis* usually commences between the ages of twenty and thirty.

Adult Life.—This is pre-eminently the age for the chronic organic affections of the cord. *Locomotor ataxia* occurs most frequently between the ages of twenty-five and forty-five. *Progressive muscular atrophy* is now relatively most common. *Myelitis* is also frequent. *Meningitis* not uncommon. Of the rarer affections, *primary lateral sclerosis* occurs between the ages of thirty and fifty; *amyotrophic lateral sclerosis* between thirty and forty-five; the very rare disease, *acute ascending paralysis* (Landry's paralysis), between twenty and forty; *polio-myelitis anterior chronica* between twenty and fifty.

§ 51. Sex.—Speaking generally, males suffer more frequently from diseases of the spinal cord than females. The greater liability of the male is partly due to the fact that he is more exposed to all sorts of injurious external influences, such as injuries, cold, syphilis, etc. *Pseudo-hypertrophic paralysis* is much more common in boys than in girls. *Locomotor ataxia,* and *Progressive muscular atrophy,* occur much more frequently in men than in women. *Hysterical paraplegia* and '*spinal irritation*' are practically confined to females. Functional derangements classed under the term '*spinal nervous weakness*' and *reflex paraplegia* are more common in males. The *congenital form of locomotor ataxia* seems to occur, at least, as frequently in girls as in boys.

§ 52. Occupation.—The occupation of the patient is not a factor of much importance in the production of cord diseases. There are, however, one or two notable exceptions to this general rule. Divers and others who work under very great atmospheric pressure are apt to suffer from *paraplegia.* Certain forms of *progressive muscular atrophy* seem to occur in persons who follow particular trades, and to be due to the over-use and consequent exhaustion of certain muscles. Lead impregnation seems also to produce a general form of muscular atrophy. The ordinary form of lead paralysis (*wrist drop*) which occurs in persons who work in lead is well-known. Those who are exposed to injurious external influences are more liable to suffer from *meningitis* and *myelitis* than the well-protected. *Pott's disease* is most common in the ill-fed and badly-clothed. *Hysterical paraplegia,* and all forms of *functional derangement* are much more frequent amongst women who lead indolent and luxurious lives than amongst the labouring classes.

§ 53. COMPLAINTS.

Most patients, who come to a physician, suffering from spinal cord disease, complain of some form of motor derangement (weakness, paralysis, unsteadiness of gait, stiffness, etc.). Patients with commencing *locomotor ataxia* complain of rheumatism and neuralgia; or, they consult an oculist on account of diplopia, squint, dimness of vision, etc. In cases

of *slow compression of the cord*, and of *chronic meningitis*, pain in the back, or in the course of certain nerves, is usually the prominent symptom. In cases of *myelitis*, difficulty in micturition may be the first symptom, but is usually soon associated with loss of voluntary motor power. In '*spinal irritation*' pain and tenderness over the spinal column is the chief complaint. Males suffering from '*spinal exhaustion*' often complain of ill-defined pains in the back and limbs, or of some derangement of the sexual functions (weakness, irritability, impotence, and the like.)

THE HISTORY OF THE CASE.

§ 54. **History of the Present Illness.**—By ascertaining the *exact date at which the illness commenced* we at once determine whether the case is *acute* or *chronic*, a fact which may be of considerable diagnostic value. Two patients, for example, present the symptoms of commencing progressive muscular atrophy, viz., atrophy of the interossei and thenar muscles. In the one case, the atrophy is of some duration, in the other, it only commenced a few weeks ago. The former is probably a typical case of progressive muscular atrophy; the rapid progress of the latter is suggestive of local disease of the ulnar nerve, and should lead to the examination of the parts about the elbow. If again the atrophy had appeared years ago and had not advanced, the idea of *progressive* muscular atrophy would be negatived.

Hæmorrhage into the cord and some traumatic injuries, such as fractures, dislocations of the spine, or wounds of the cord, are attended with *immediate* symptoms. In cases of hysterical paraplegia the onset may occur *suddenly* and immediately after an hysterical fit. Acute myelitis may develop with great rapidity, but the onset is not instantaneous, as it may be in hæmorrhage. In other inflammatory cases, the onset is *more gradual*. In slow compression of the cord, from whatever cause, the development is *still more tardy;* while, in the chronic affections, such as progressive muscular atrophy, locomotor ataxia, primary lateral sclerosis, and disseminated sclerosis, the departure from health is, usually, *very gradual*, and the course extremely slow and chronic. Indeed, so gradual is the onset in some of these cases, that the patient

is unable to fix the date at which the first symptoms commenced.

The mode of commencement of the attack, the character of the symptoms, and *the order in which they were developed,* are often of great diagnostic importance. Two patients, for example, present extreme atrophy and paralysis of the right arm. In the one case the paralysis appeared suddenly at the outset of the attack, and was quickly followed by marked atrophy. Such a history (in the absence of wound or injury) would suggest *polio-myelitis anterior acuta.* In the second case the atrophy preceded the paralysis, the motor weakness increasing *pari passu* with the muscular wasting. Such a mode of commencement would indicate *progressive muscular atrophy.* So too in a case of spastic paraplegia in a female, the *sudden* occurrence after a fit would point to the functional (hysterical) nature of the paralysis; while the *gradual* onset without hysterical phenomena would be in favour of organic disease (transverse myelitis, etc.)

If there is any *alleged cause* for the attack, its exact nature must be carefully inquired into. Where external violence is said to be the cause, the degree and kind of violence, the exact part of the body which was injured, the exact character of the symptoms and appearances which followed the injury, and the subsequent local changes and constitutional symptoms must all be carefully noted. It is only by attention to these details that we can possibly determine whether the alleged injury was adequate and likely to produce the symptoms from which the patient is suffering at the time of examination. You must, however, know that severe, and even fatal, cord disease sometimes results from concussion of the spine, such as is produced by a railway collision, without any evidence of bruising or local injury. Careful attention to the points I have just mentioned, sometimes gives valuable information as to the severity of the injury, as in the following case:

A lady was seated in a second-class railway carriage when a collision occurred. She was first thrown forwards, then backwards, and in falling backwards the calf of the left leg came in contact with the edge of the seat on which she was sitting when the accident occurred. Pain and tenderness on pressure were complained of, and there was some swelling of the calf; but for several days after the accident no other local indication of a bruise was observed. At the end of ten days discoloration commenced, the leg from the knee to the ankle became

deeply ecchymosed. The discoloration continued for several weeks. Now, the tardy appearance of the discoloration and its long continuance proved the severity of the injury, for they showed that the extravasation was extensive and deep-seated, *i.e.*, below the calf muscles; and such an extravasation could only be produced in a healthy individual by very considerable violence.

The nature of the treatment up to the time when the patient comes under observation is in some cases of importance, for symptoms are often modified, and sometimes produced by treatment. The fact that the patient has gone through a prolonged course of anti-syphilitic remedies without any beneficial result would rather negative the idea of syphilis being the cause, and so would make the prognosis more serious. Again, in a case of motor weakness, in which stiffness and rigidity were prominent features, the fact that the patient had been taking strychnine, and that the stiffness and rigidity had appeared *after* its use, would be very suggestive of cause and effect, and would make the prognosis, as regards the stiffness and rigidity, at all events, more hopeful.

§ 55. **The History as to the State of Health before the commencement of the Present Illness, the Habits, Mode of Life, and general surroundings of the Patient** are also very important. *Syphilis* undoubtedly is a cause of some cases of myelitis; and the statistics of Erb, Gowers, and others, show that it plays an important part in the production of locomotor ataxia.

The question as to syphilis being *the* cause of locomotor ataxia is still under discussion. There is one form of proof which would, I think, be conclusive should it be forthcoming. Women, in this country at least, seldom suffer from locomotor ataxia. Now, if syphilis is the cause of the affection, prostitutes who are so often syphilitic, ought to be affected with locomotor ataxia much more frequently than the general mass of the female population. Unless this fact is forthcoming, we cannot allow that syphilis is *the sole* cause, but must admit that there is some other condition in addition to the syphilis which renders males more liable to the affection (locomotor ataxia) than females.

Diphtheria is the most common cause of paralysis of the palate, and it is sometimes followed by paralysis of muscles supplied by spinal nerves. *Cerebro-spinal meningitis* occasionally results from an epidemic poison; in cases of cerebro-

spinal meningitis, therefore, where there is no ear disease or other obvious cause, a history of previous cases in the neighbourhood, or the prevalence of an epidemic, would be important. *Plumbism.*—It is well known that lead gives rise to various forms of nerve disease. A history, therefore, of exposure to lead impregnation, or of previous symptoms of lead poisoning, such as dry colic, wrist drop, etc., should carefully be inquired into in all cases of atrophic paralysis. *Sexual excess* used to be considered an important cause of spinal cord disease. Inasmuch as it lowers the general tone of the system, and debilitates the spinal cord in particular, sexual excess may be considered as a predisposing cause of cord disease; but the evidence is, I think, as yet inconclusive, that any organic spinal affection is produced by it. *Typhoid fever, small-pox,* and the other acute infectious diseases are sometimes followed by paraplegia; in some cases, probably the majority, the spinal symptoms are functional, in others the paraplegia is due to myelitis, etc. *Excess in alcohol* would also appear to be an occasional cause of paraplegia.

§ 56. **The Family History.** Many nervous affections are hereditary. It is very important therefore to ascertain whether any of the patient's near relatives have suffered from symptoms similar to those for which he comes under observation.

Some spinal affections are eminently hereditary. *Pseudo-hypertrophic* paralysis is so in a large proportion of cases, and is chiefly transmitted through the females. *Progressive muscular atrophy,* too, is often hereditary. Occasionally, though very rarely, *locomotor ataxia* runs in families; Carré, quoted by Erb,[1] saw eighteen cases of the disease in one family in three generations.

But in addition it is necessary to inquire whether there is *any* tendency to nervous disease in the family, for we frequently find the type of disease changing in different generations; the mother, for example, may be hysterical, the children epileptic, the grand-children idiotic, and so forth.

The general hereditary tendencies of the patient are also important. In a case of slow compression of the cord, a strong family tendency to cancer, in the absence of evidence

[1] *Ziemssen's Cyclopædia,* vol. xiii., page 523.

as to the exact nature of the compressing cause, would be suggestive of a malignant growth within the spinal canal. So, too, in a case of spinal meningitis, arising without any obvious cause, an hereditary tendency to scrofula would point to the tubercular character of the lesion.

PRESENT CONDITION.

[The date at which the examination is made should be stated, for in hospital practice the case may not be taken for some days after the patient's admission.]

§ 57. **The Physiognomy of the Case.**[1] While the preliminary facts and previous history are being investigated, the physician is both consciously and unconsciously learning many important particulars as to the nature of the case.

In some cases the physiognomy at once suggests the nature of the disease. I must, however, warn you against physiognomic diagnoses, for the physiognomy is seldom pathognomonic. A patient, for example, presents the characteristic gait of locomotor ataxia, and you diagnose that condition. This is a method of reasoning which we are all, no doubt, going through daily, but it is not strictly accurate. The peculiar gait only shows that the patient is suffering from that form of inco-ordination which results from a lesion of the posterior columns. The further conclusion that the disease is locomotor ataxia would, doubtless, in the vast majority of cases be correct, for, the sclerotic lesion of locomotor ataxia is by far the most common lesion of the posterior columns. But the conclusion is not logical, and in a small proportion of cases would be quite erroneous, for, other lesions may chance to be limited to the posterior columns, and to be attended with the same form of inco-ordination. The conclusion which we could correctly draw in such a case would be, 'the posterior columns are affected, and the strong probability is, that the case is one of locomotor ataxia.'

The physiognomy of the case is, however, always important, and in some cases of spinal cord disease, the picture which the patient presents is a very striking one.

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[1] By the term physiognomy of the case, I mean not only the facial physiognomy, but the appearance of the case *as a whole*.

In *acute cases* he is generally in bed. In *meningitis* the countenance usually exhibits evidence of suffering; the head may be retracted by spasm of the cervical muscles; the legs drawn up; the patient avoids and dreads movement, for it increases the pain and spasm.

In the earlier stages of *polio-myelitis anterior acuta*, there is usually considerable fever: the little patient may be flushed or slightly delirious; twitchings and tremors in the tendons of the wrist, and in the facial or other muscles are often observed;[1] the paralysed limbs lie flaccid and helpless.

In *hysterical paraplegia* the patient is a female, and in most cases young. She is probably confined to bed; the facial appearance is characterised by 'a remarkable depth and prominent fulness with more or less thickness of the upper lip, and by a peculiar drooping of the upper eyelid' (Todd). The paralysed muscles are in some cases flaccid; in others rigid, the legs extended, and the feet inverted; they are not markedly atrophied, and the general state of nutrition is unaffected.

In *locomotor ataxia* the patient is in the great majority of cases a male of from twenty-five to fifty years of age. A haggard expression of face is often noticeable, and is very suggestive of frequent and severe attacks of pain. Paralysis of one or other of the ocular muscles, or of the upper eyelid, is not uncommon, more particularly in the earlier stages. Later on, the pupils may be 'pin point.' The general state of nutrition is usually good; the gait is very characteristic, though, as has been already mentioned, not absolutely pathognomonic; the patient walks with two sticks; he keeps his eyes fixed on his feet or on the ground in front of him; the steps are taken at regular intervals, slowly, and with deliberation, but the movements of the legs are irregular; the feet are jerked outwards, the heels brought suddenly to the ground with a stamp. In uncomplicated cases he walks in a straight line. There is no giddiness, the unsteadiness is, as Duchenne long ago pointed out, not due to the head but to the legs.

In *spastic paraplegia*, a condition which is common as the

[1] These muscular twitchings are not characteristic of infantile paralysis; they are commonly observed in cases of rapid elevation of temperature in children.

result of a transverse myelitis, but very rare as a primary system disease (*primary lateral sclerosis*), the facial appearance is in no way altered: the general state of nutrition is good. In advanced cases the patient lies in bed, with the legs stiffly extended and adducted, the feet are usually inverted. In the earlier stages the patient is able to walk about, and the gait is peculiar: he walks with two sticks: each step is attended with evident effort; the feet appear to be stuck to the ground, and can only be moved forward by raising the pelvis, and with it the limb as a whole. In this process the back is strongly arched, the chest thrown forward, the patient leans forcibly, first on one stick and then on the other, and appears to aid the elevation of the trunk by movements of his arms. The toes are dragged along the ground with an unpleasant scraping noise, the knees are apt to interlock, and the foot which is being brought forward tends to cross in front of its fellow. In some cases, after the foot leaves the ground, a peculiar hopping movement of the whole body is observed. It is due, according to Erb, to spasmodic contraction of the calf muscles. Another form of gait, which bears some resemblance to the 'hopping' gait of spastic paraplegia, is the so-called 'high action,' or 'equine' gait. At each step the leg is raised high off the ground by flexion of the thigh or the abdomen. This form of gait is usually associated with paralysis of the muscle on the front of the leg, and is a voluntary effort to 'dodge the paralysis,' *i.e.*, to prevent the toes which hang down catching the ground. In other cases the high action is involuntary, and seems to be due to spasmodic action of the muscles which flex the thigh on the abdomen.

In *paraplegia with flaccidity*, the feet are trailed with difficulty one after the other. The knees do not interlock. There is not the marked arching of the back which is seen in the spastic form, and the foot which is being brought forward does not tend to cross the middle line.

In *progressive muscular atrophy* the facial appearance is seldom altered. In the earlier stages the wasting of the muscles of the hand, and in some cases the 'bird claw' appearance of the fingers, at once attract attention. In advanced cases the wasted limbs hang helplessly by the sides, as if they were hung on to the trunk by strings. The lower limbs are seldom so profoundly atrophied as to prevent the patient getting about; but in advanced cases, they, like the arms,

seem to hang loosely. When the lumbar muscles are affected, the back is arched, the belly protruded, but the gait is not 'waddling,' as in pseudo-hypertrophic paralysis.

In *pseudo-hypertrophic paralysis* the gait is pathognomonic. The patients are, in the great majority of cases, boys. The back is strongly arched; the chest protruded; the feet widely separated; the heels are usually drawn up and raised from the ground, in consequence of the cirrhotic contraction of the calf muscles; the arms are held down, and slightly out from the sides, with the object of balancing the body. The gait is 'waddling,' the body oscillates from side to side; there is an evident difficulty of flexing the thigh on the abdomen, and of projecting the foot forwards, for after the foot is raised, the movement in advance is small. The calves, and possibly some of the other muscles, are large, and apparently hypertrophied. Some muscles are usually atrophied.

In *cerebro-spinal sclerosis* the patient is generally between twenty and forty years of age. The countenance usually presents a stupid, vacant, stolid, expression; the mouth may be half open; the speech is altered, the tone monotonous, the words drawled out in a scanning manner; nystagmus (oscillation of the eyeballs) occurs in a certain proportion of cases; the pupils are sometimes markedly contracted. In the earlier stages the gait is not much altered, but the experienced observer can often, even then, detect characteristic alterations. In many cases the patient walks with the head erect or turned slightly to one side, and perhaps drawn a little back; there is an evident effort to keep the neck stiff and the head steady. On close observation, slight jerking movements of the head and neck can be perceived. In more advanced cases the gait is extremely unsteady and irregular; but in characteristic cases the inco-ordination is quite different from the gait of locomotor ataxia; it is, so to speak, of a coarser kind, and seems to involve the muscles of the trunk rather than the muscles of the legs. The patient does not walk deliberately and in a straight line (as the subject of locomotor ataxia does), but is apt to shoot suddenly forwards or to one side; the loss of control is sometimes so great, that he knocks up against surrounding objects. I have seen a patient in a small room fall violently against the opposite wall.

§ 58. THE CLINICAL EXAMINATION OF THE SPINAL CORD.

As I have previously remarked, the essence of the clinical examination of the spinal cord consists in the separate and systematic examination of its individual segments. The motor, sensory, reflex, vaso-motor, and trophic functions of each segment should be investigated as accurately as our present knowledge will allow.

I will now briefly describe the symptoms which are met with in diseases of the spinal cord, and will indicate the points to which attention is to be directed in the clinical examination of the case.

§ 59. MOTOR DERANGEMENTS.

The symptoms which result from derangement of the motor functions of the cord may be either in the direction of diminished or increased function. Paralysis, paresis, and inco-ordination are examples of the former; spasms, cramps, and rigidity, of the latter.

§ 60. PARALYSIS.—Spinal paralysis is, in the great majority of cases, bilateral and limited to the muscles of the lower extremities; it is, then, termed *paraplegia*. The loss of motor power is not unfrequently greater on one side than on the other. The bilateral character of the paralysis is due to the facts:—that the motor tracts of the two sides of the body are in such close relation in the spinal cord, that acute indiscriminate lesions, such as myelitis, hæmorrhage and traumatic injuries, can with difficulty affect one side without implicating the other; and that the system lesions of the motor tract have a strong tendency to be bilateral.[1]

In rare cases the lesion is situated in or above the cervical enlargement; the upper limbs are then involved; and the condition is termed *cervical paraplegia*. In exceptional cases the lower limbs escape while the upper limbs are paralysed.

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[1] In polio-myelitis anterior acuta, the lesion is sometimes limited to one anterior horn, but even in that affection paraplegia is the rule.

Paraplegia may result from a total transverse lesion (myelitis, compression), from lesions which involve the pyramidal tracts on both sides of the cord (primary lateral sclerosis, etc.), the anterior cornua in corresponding half segments, the anterior root-fibres or the anterior roots on both sides of the body.

Occasionally a lesion of the cord is limited to one side, and produces one-sided paralysis (*spinal hemiplegia* or *spinal monoplegia*). The loss of motor power is on the same side as the lesion. (See page 60).

§ 61. *The points to which attention is to be directed in the examination of a case of spinal paralysis are : —*

1. The exact distribution of the paralysis.
2. The amount (degree) of the paralysis.
3. The trophic condition of the paralysed muscles.
4. The irritability (electrical and mechanical) of the paralysed muscles.
5. The tonicity of the paralysed muscles.
6. The condition of the reflexes.
7. The condition of co-ordination and of the muscular sense.

§ 62. The Exact Distribution of the Paralysis.—The distribution of the paralysis can often be roughly determined by observing the attitude and gait of the patient. It is accurately ascertained by testing his capability of executing muscular movements; by noting the movements which are wanting, or which are imperfectly performed, we can, provided of course we know the muscle concerned in the production of the movement, determine the muscles which are at fault. The exact distribution of the paralysis gives important information as to the position of the lesion in the cord. The paralysis is, then, to some extent a localising symptom.

§ 63. *The Motor Functions of Individual Segments.*—In examining the condition of the motor functions of the spinal cord, it is important for scientific purposes, or where great accuracy in localisation and diagnosis is required, to examine the motor condition of individual segments by observing the condition of the muscles which they supply.

The motor functions of the different nerve roots (*i.e.*, of

the different segments) of the cervical and lumbar enlargements are, according to recent observations, as follows:—

Functions of the nerve roots of the cervical enlargement (according to Professors Ferrier and Yeo, from observations made on monkeys).

Fourth cervical.—Flexion of the forearm, with supination and extension of the wrist and fingers, the upper arm raised upwards and backwards.

Fifth cervical.—Movement of the hand towards the mouth, viz., raising the upper arm inwards, flexion of the forearm with supination, and extension of the wrist and fingers.

Sixth cervical.—The movement of 'attention' viz., adduction and retraction of the upper arm, extension of forearm, pronation and flexion of wrist, the palm of the hand being brought towards the pubes.

Seventh cervical.—The *scalptor ani* action, viz., adduction and rotation inwards and retraction of the upper arm, extension of the forearm and flexion of wrist and fingers so as to bring the tips against the flank.

Eighth cervical.—Closure of the fist with pronation by ulnar flexion of wrist, retraction of the arm with extension of the forearm.

First dorsal.—Action of the intrinsic muscles of the hand, muscles of ball of thumb, interossei, etc.

Functions of the nerve roots of the lumbar enlargement (according to Professors Ferrier and Yeo, from observations made on monkeys; and M. M. Paul Bert and Marcacci, from observations made on cats and dogs).

First lumbar.—Determines contraction of the sartorius, rectus, and psoas—which flex the hip upon the trunk. (P. B. and M.).

Second lumbar.—Excites contraction of the anterior portion of the vastus externus, a part of the tensor of the fascia lata, and the vastus internus—viz., the muscles which extend the leg or the thigh. (P. B. and M.).

Third lumbar.—Similar to that of the second, with some differences in detail. It excites part of the vastus externus and the anterior part of the biceps, which is an extensor, while the posterior portion is a flexor. (P. B. and M.).

According to Ferrier and Yeo, stimulation of the third lumbar in the monkey causes flexion of the thigh and extension of the leg.

Fourth lumbar, according to M.M. Paul Bert and Marcacci, causes in the cat and dog, movements in the posterior part of the biceps, the semi-tendinosus, and the semi-membranosus (flexors of the leg or the thigh), the second and third adductors of the thigh and the extensors of the thigh. It thus innervates three kinds of movements which are in no respect opposed or contradictory.

According to Professors Ferrier and Yeo, irritation of the fourth root in the monkey causes extension of the thigh, extension of the leg, and pointing of the great toe.

Fifth root.—M.M. Paul Bert and Marcacci find that in the dog and cat the fifth root presides over the movements of the tail. According

to Ferrier and Yeo, irritation of the fifth root in the monkey produces :—Outward rotation of the thigh, flexion and inward rotation of the leg, plantar flexion of the foot, and flexion of the distal phalanges.

First sacral.—Flexion of the leg, plantar flexion of the foot, flexion of all the toes at the proximal phalanges, and also of the distal phalanx of the hallux. (F. and Y.).

Second sacral.—Action of the intrinsic muscles of the foot, viz., adduction and flexion of the hallux, with flexion of the proximal phalanges and extension of the distal. (F. and Y.).

§ 64. The Amount (Degree) of the Paralysis.—Where the paralysis is complete all muscular movement is of course absent. In incomplete paralysis (paresis) the amount of motor impairment is measured by observing the amount of force which the paralysed muscles are capable of exercising. In estimating the force of a muscle in this manner, the movement which it is made to execute should resemble, so far as possible, the movement which it is normally in the habit of executing. In measuring the force of the calf muscles, for example, jumping on tip-toe is, as Dr Gowers points out, a better test than the degree of force which can be exercised in opposition to passive

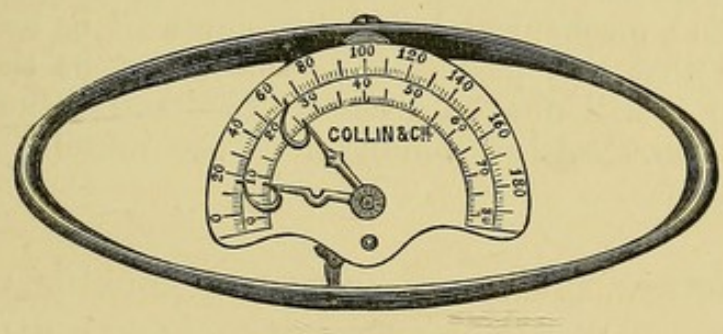


Fig. 76.

dorsi-flexion of the foot. The dynamometer (see fig. 76) is a ready means of estimating the exact force of the flexor muscles of the fingers (grasping power). The strength of the other muscles is ascertained by observing :—(1) the force with which voluntary movements are executed, and (2) the amount of resistance which can be offered by the patient to passive movements made by the physician. In cases of one-sided paralysis, the standard of comparison is the motor force of the corresponding muscles on the healthy side. Where the paralysis is bilateral, the amount of motor power must be compared with that of a healthy individual. Where another individual is taken as the standard of comparison, his

muscular development should resemble, as much as possible, the muscular development of the patient prior to the onset of the paralysis.

§ 65. The Trophic Condition of the Muscles.—In some cases of spinal paralysis the muscles undergo 'rapid atrophy;' in other cases their state of nutrition is well preserved; in others again, they are moderately wasted. The trophic condition of the paralysed muscles depends upon the position of the lesion. Where the lesion is situated *above the trophic nucleus* (the multipolar nerve cells of the anterior cornu), the paralysed muscles may be well nourished, or moderately wasted (atrophy of disuse). Where the multipolar nerve cells are acutely destroyed, or where their trophic influence is suddenly shut off by lesions of the anterior roots or peripheral nerves, 'rapid atrophy' results. Gradual destruction of the multipolar nerve cells is attended with gradual but progressive atrophy, which may ultimately become extreme.

§ 66. The Irritabilty of the Muscles.—The Faradic, galvanic, and mechanical irritability, must all be tested. In order that that this part of the subject may be thoroughly understood, it will now be necessary to describe the manner in which the electrical condition of the muscles is to be ascertained.

§ 67. Practical Directions as to the Mode of Applying Electricity in the Diagnosis of Cases of Paralysis.

1. *See that the battery is in working order.* Firmly attach the rheophores to the binding screws (I, I, fig. 77) of the battery and to the electrodes. Thoroughly moisten the electrodes. See that the interruptor (G) is in the proper position. Place the required number of cells into action by means of the handle shown on the dial (E). Try the effect of the current on your own person before applying it to the patient; the ball of the left thumb is the most convenient site, the electrodes being held in the right hand, as shown in fig. 78. If there is any doubt as to the battery acting, *i.e.*, as to a current passing, the electrodes may be applied to the tip of the tongue; a very weak current should be used for this purpose.

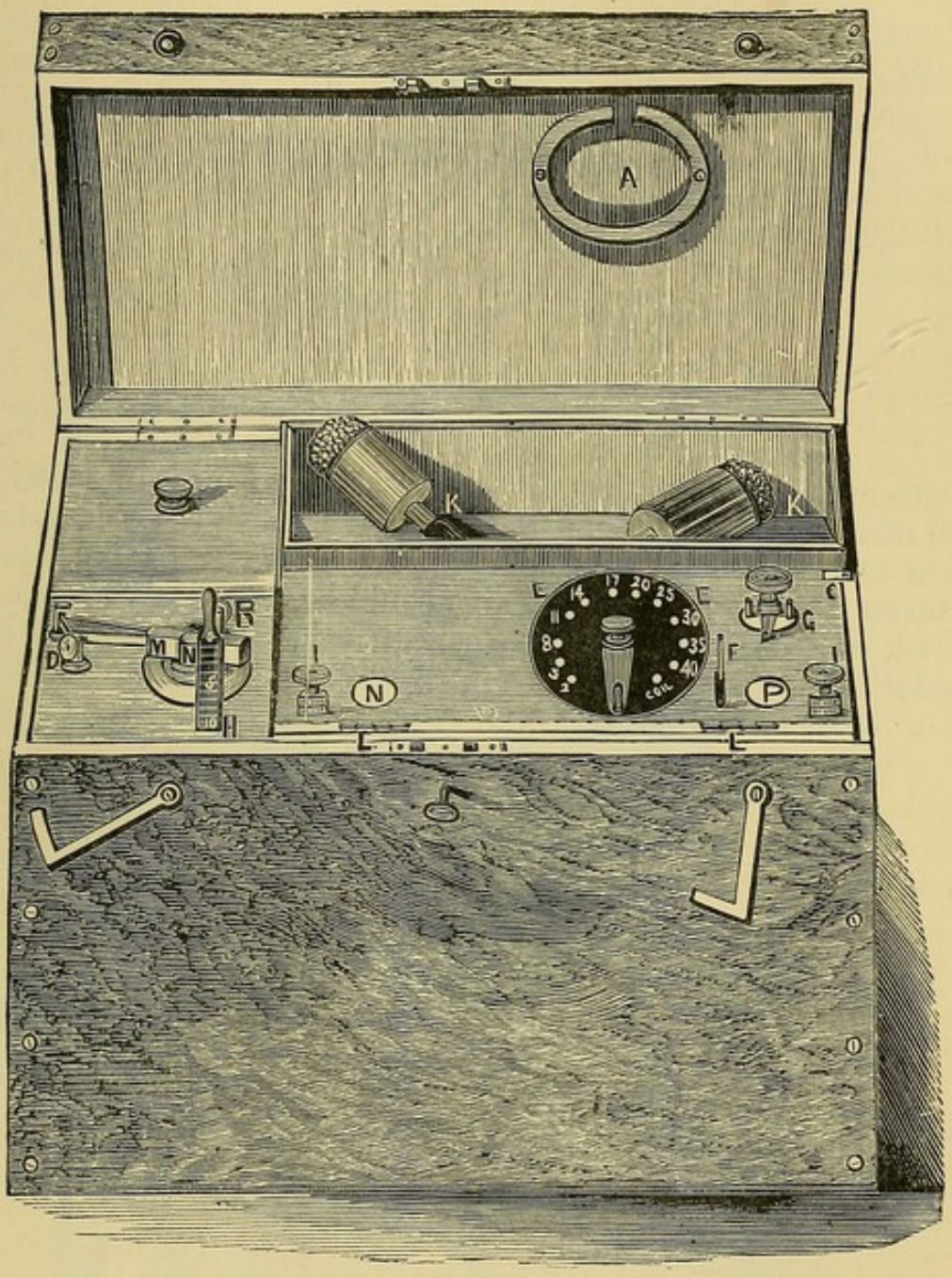


FIG. 77.

The Tibbits Combined Hospital Battery—the form which I am in the habit of using.

A. Guard Block.
C. Bolt.
D. Screw regulating spring of hammer.
E. Dial.
F. Commutator of poles.
G. Key.
H. Graduator of induction coil.
I. I. Binding screws.
K. Tray for holding accessories.
L, L. Hinges of element board.
M. Hammer.
N. Electro-magnet.
R. Screw regulating needle.

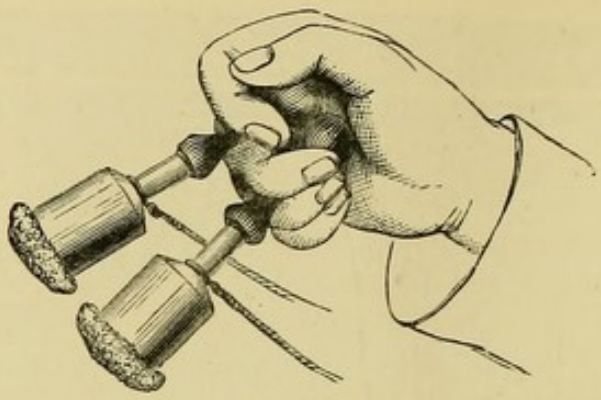


FIG. 78.

2. *Make the skin a good conductor.*—In order that the current may reach the muscles and nerves, it is necessary to moisten those portions of the skin to which the electrodes are to be applied, for the dry skin is an extremely bad conductor of electricity. The skin should be thoroughly sponged with warm water, holding a small quantity of common salt in solution.

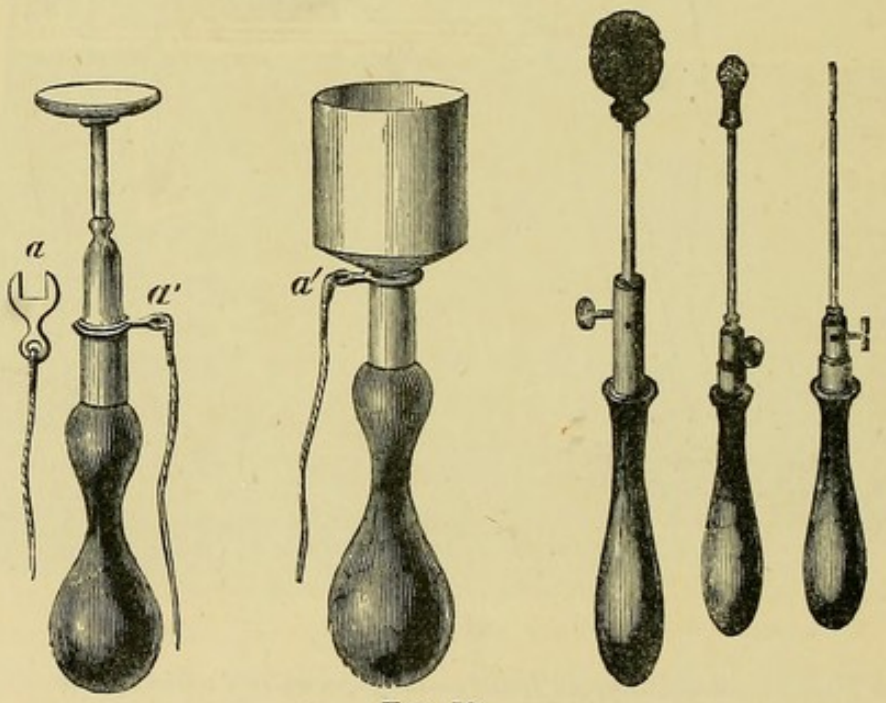


FIG. 79.

Various forms of Electrodes.—*a*, rheophore detached from the electrode; *a'*, *a'*, rheophore attached to the electrodes.

3. *Apply the electrodes.*—In using the continuous current, the 'polar method' of applying the electrodes should be adopted. The *polar method* is as follows: One pole is applied over a distant and neutral part, such as the manubrium

sterni, or the cervical spines; the other over the muscle or nerve trunk which is to be tested. By this means the action of the two poles (a most important point in the diagnosis of some cases of paralysis) can be readily distinguished.

If it is desired to throw the whole muscle into action or to act on the muscle through its nerve, the peripheral electrode should be placed over that part of the muscle where the motor nerve enters. This point is called the motor-point.

In the following plates, copied from Von Ziemssen, the position of the chief motor points is shown.

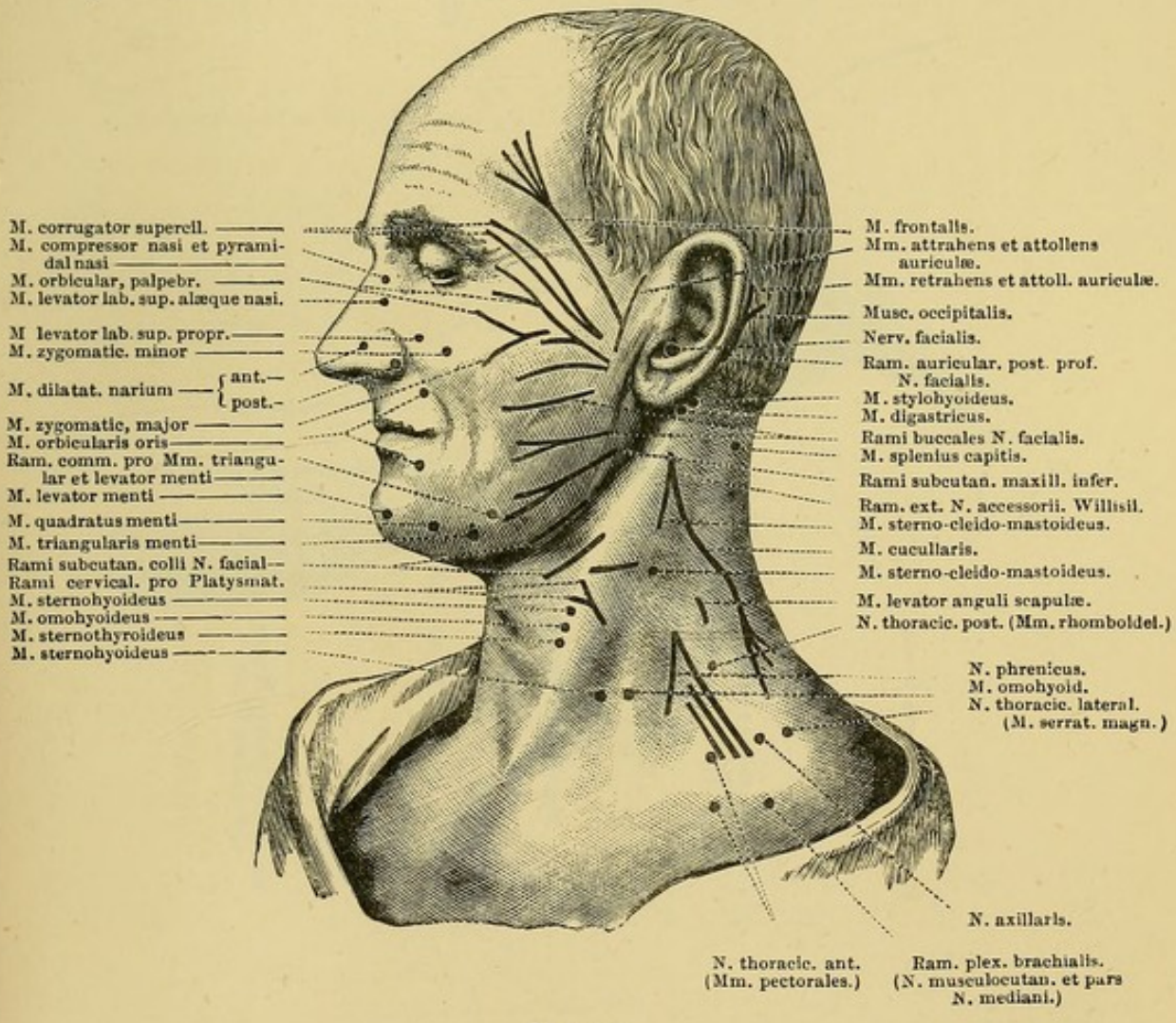


FIG. 80.

Motor Points of the Face and Neck.

Showing the position of the electrodes in electrisation of the facial nerves and muscles. The anode is placed in the mastoid fossa, and the cathode upon the point indicated in the figure.

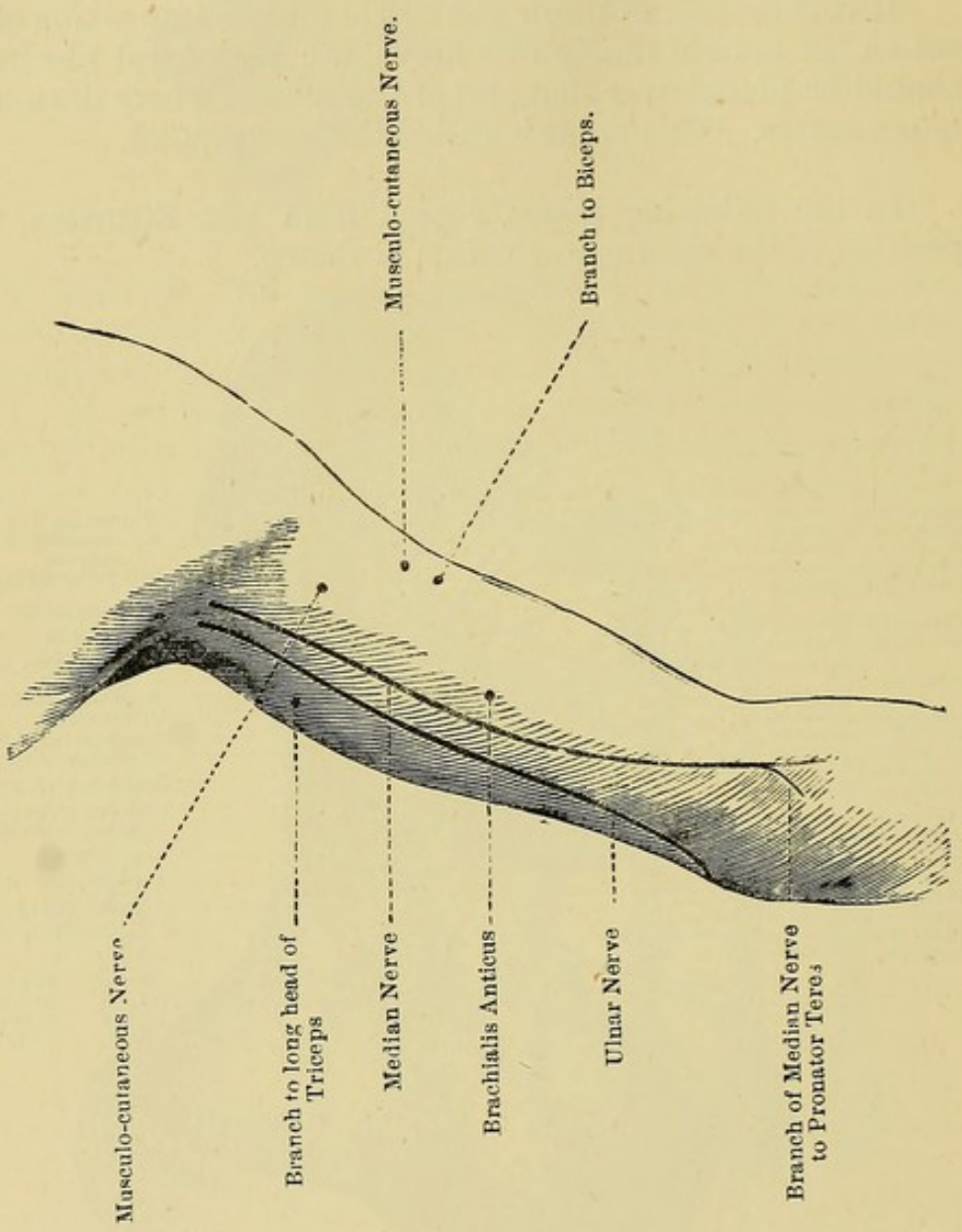


FIG. 81.

Motor Points on the Anterior (flexor) Surface of the Left Arm.

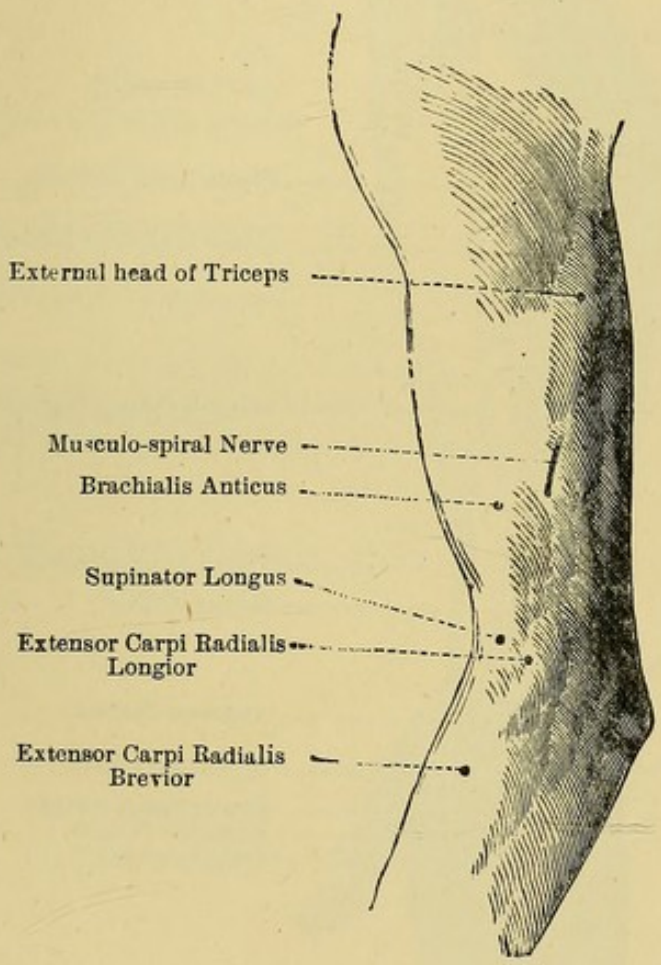


FIG. 82.

Motor Points on the Posterior (extensor) Surface of the Left Arm.

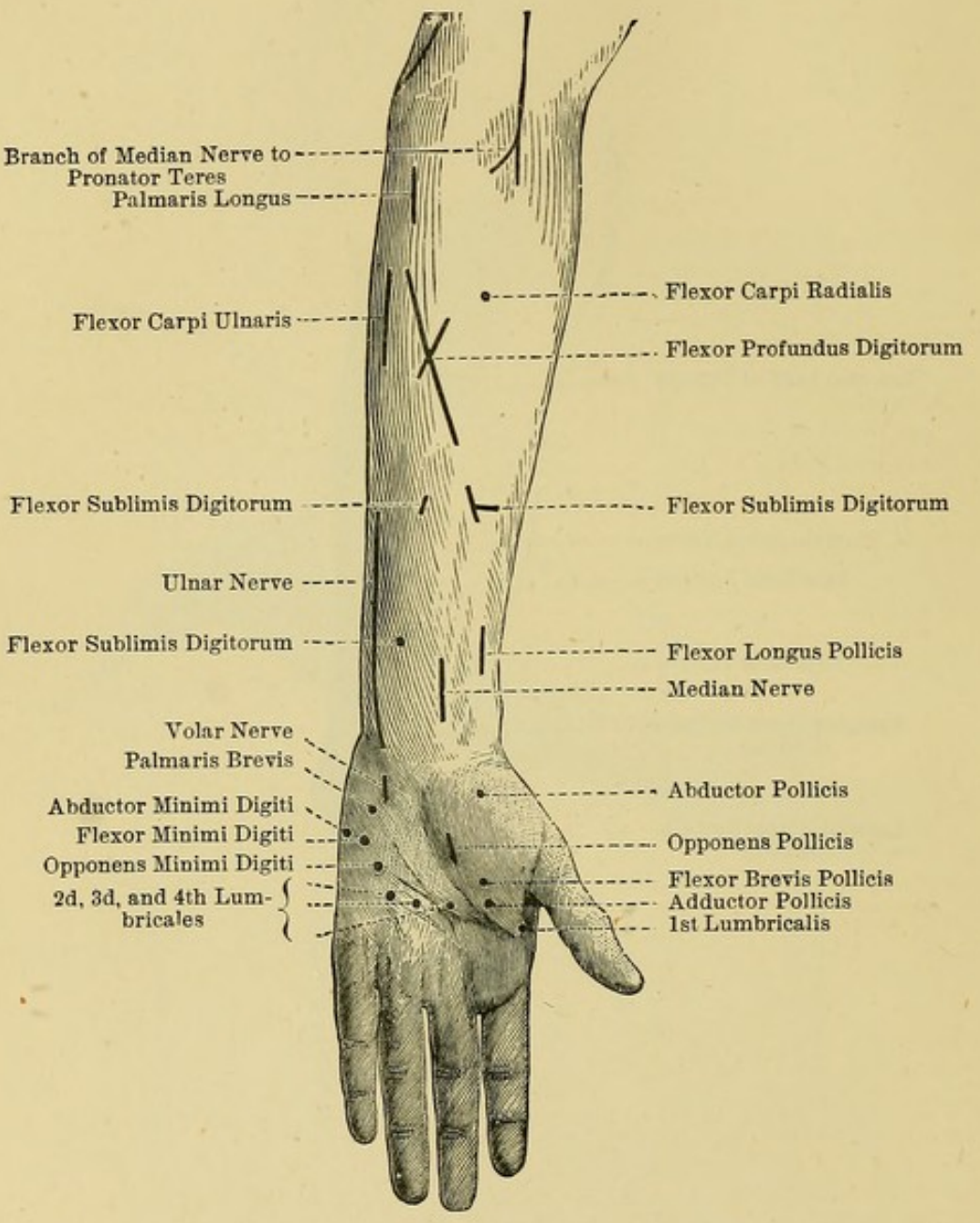


FIG. 83.

Motor Points on the Anterior (flexor) Surface of the Left Forearm.

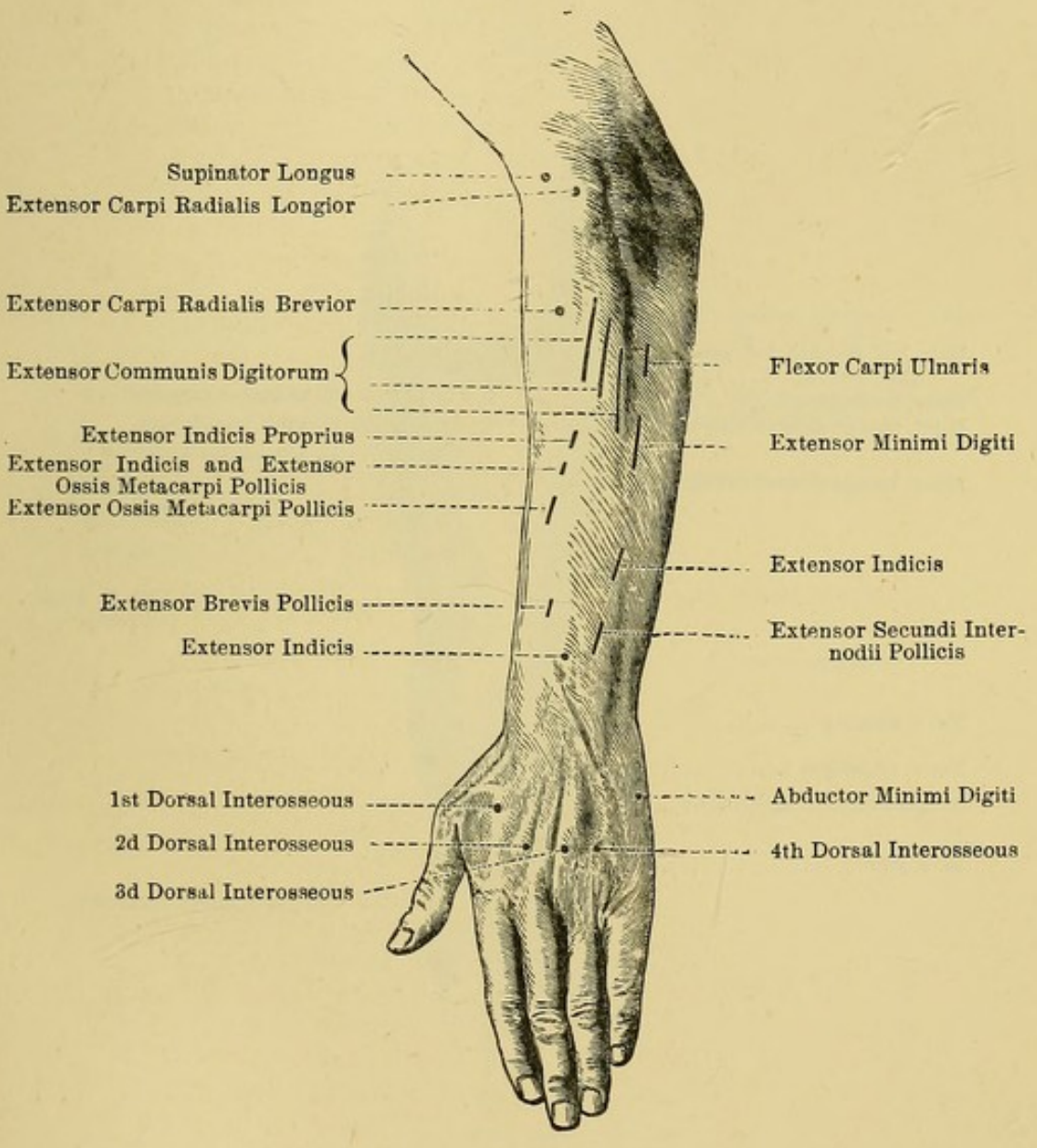


FIG. 84.

Motor Points on the Posterior (extensor) Surface of the Left Forearm.

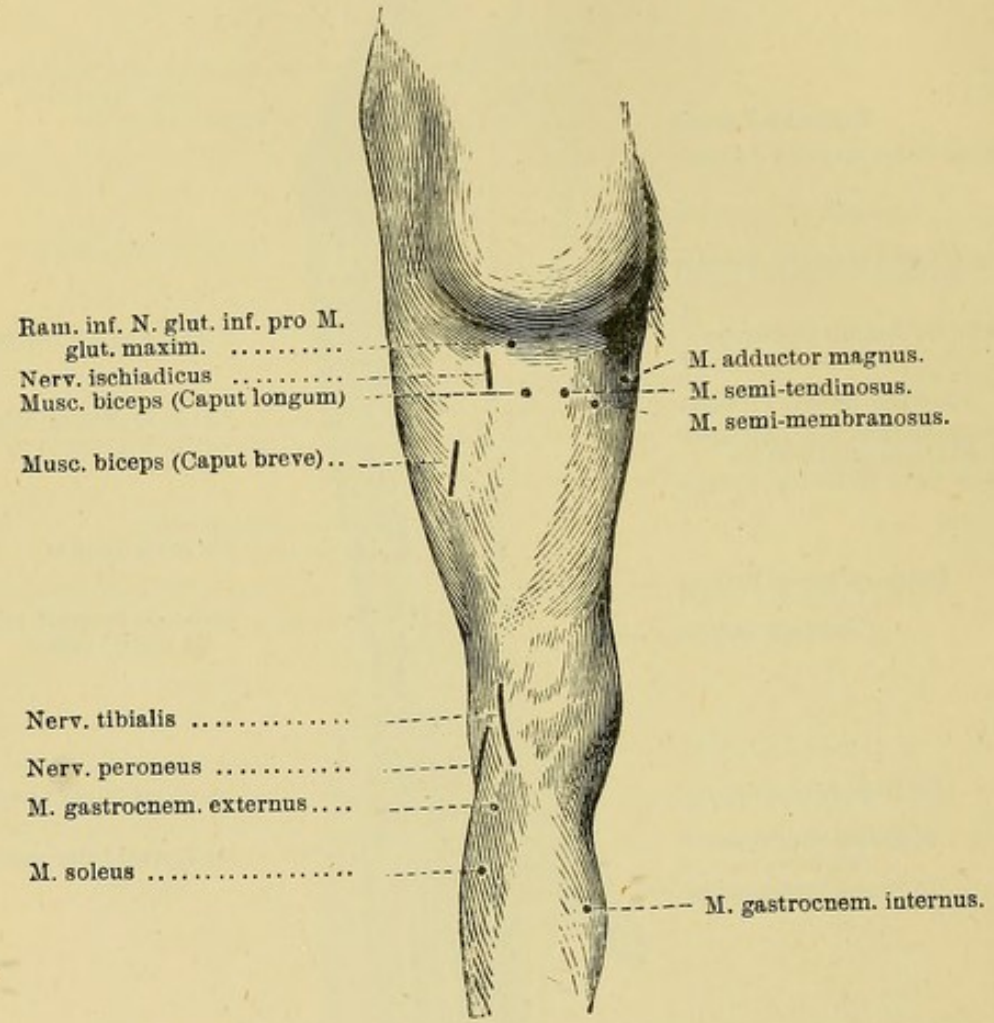


FIG. 85.

Motor Points on the Posterior Surface of the Thigh.

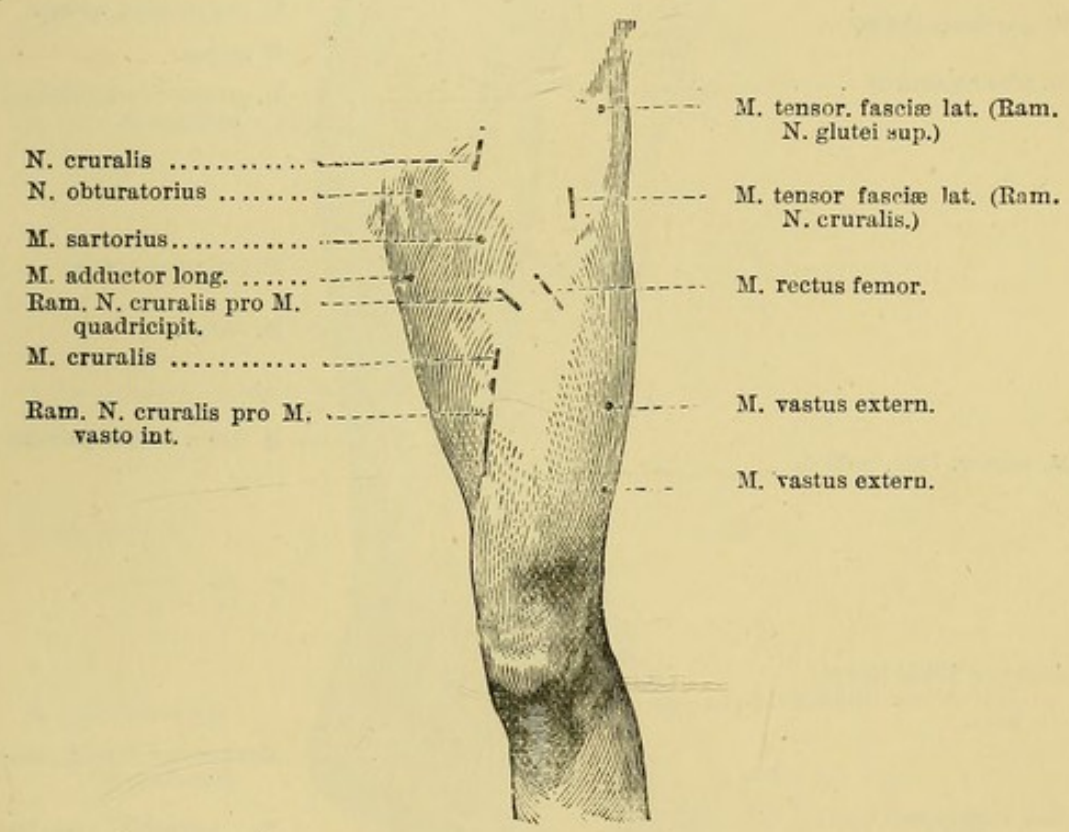


FIG. 86.

Motor Points on the Anterior Surface of the Thigh.

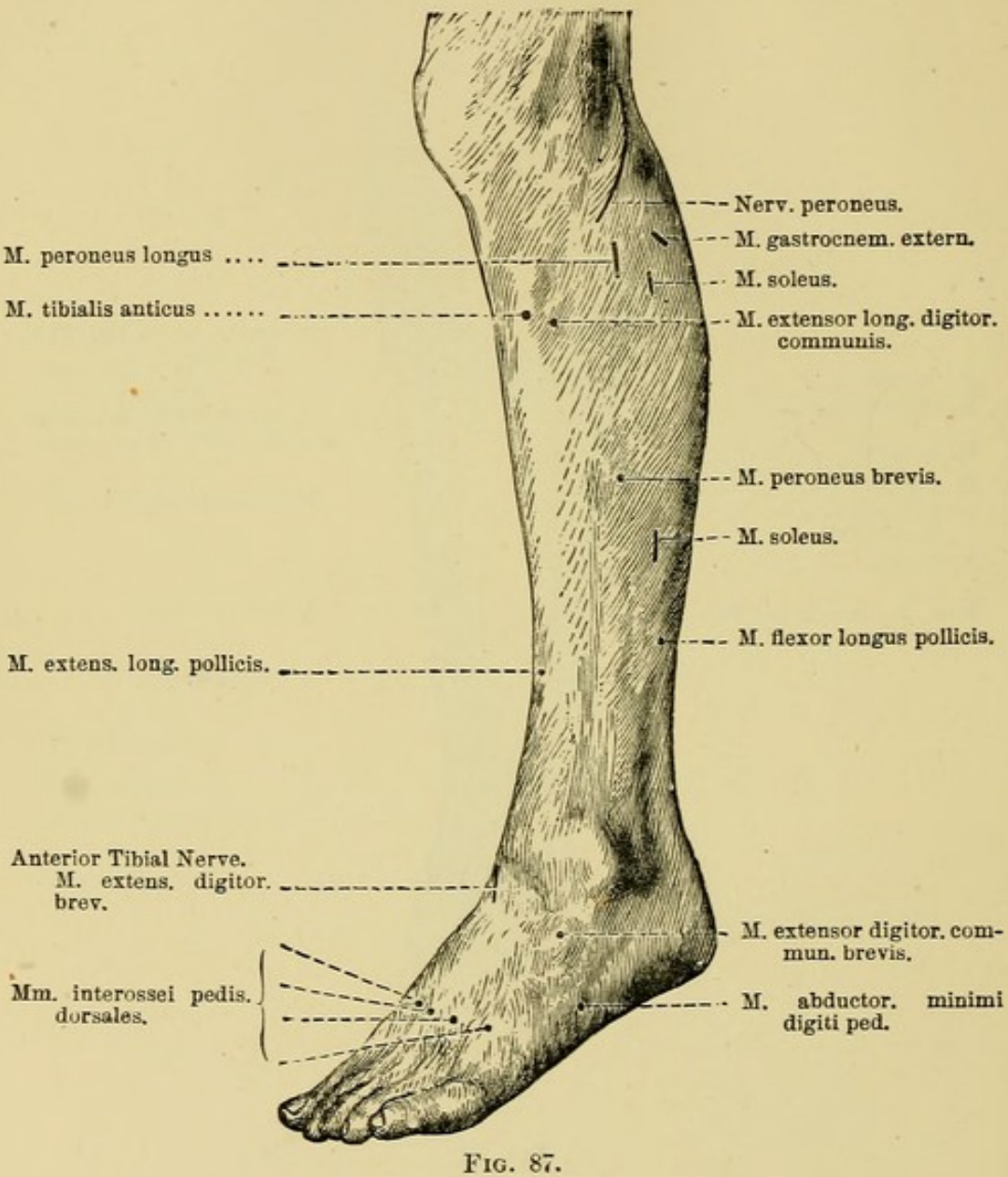


FIG. 87.

Motor Points on the Outer Surface of the Leg.

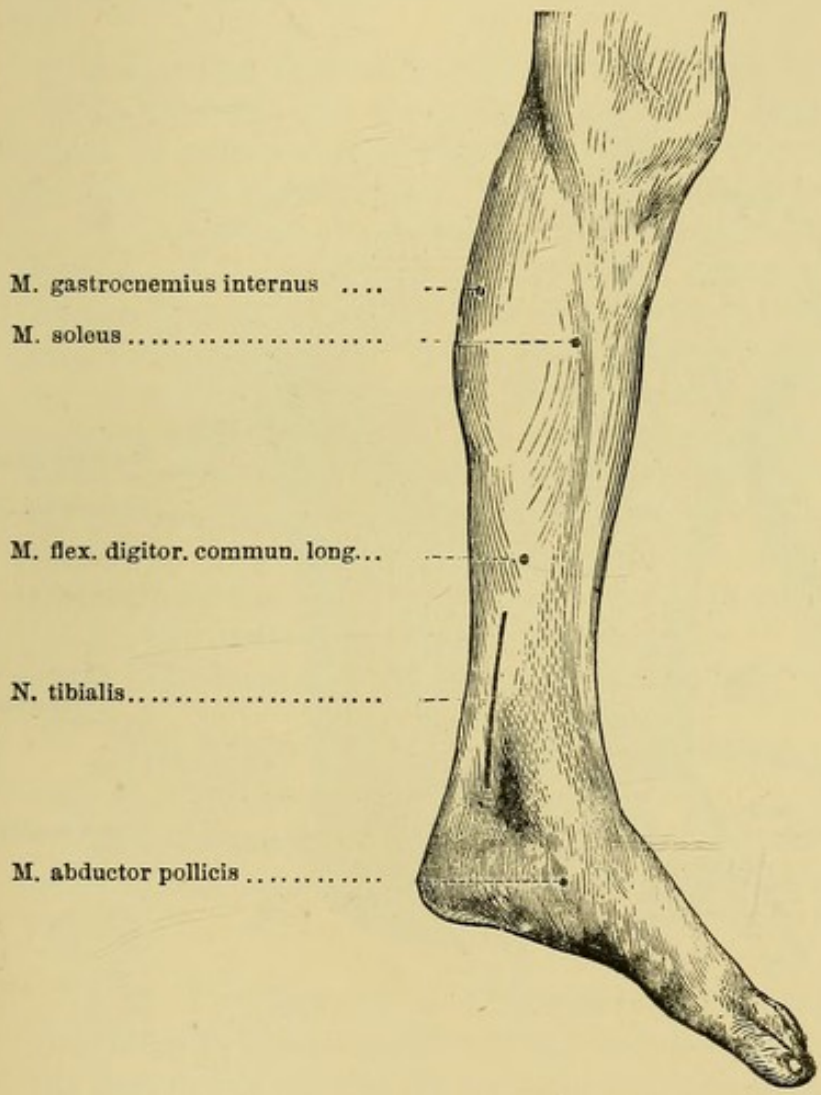


FIG. 88.

Motor Points on Inner Surface of the Leg.

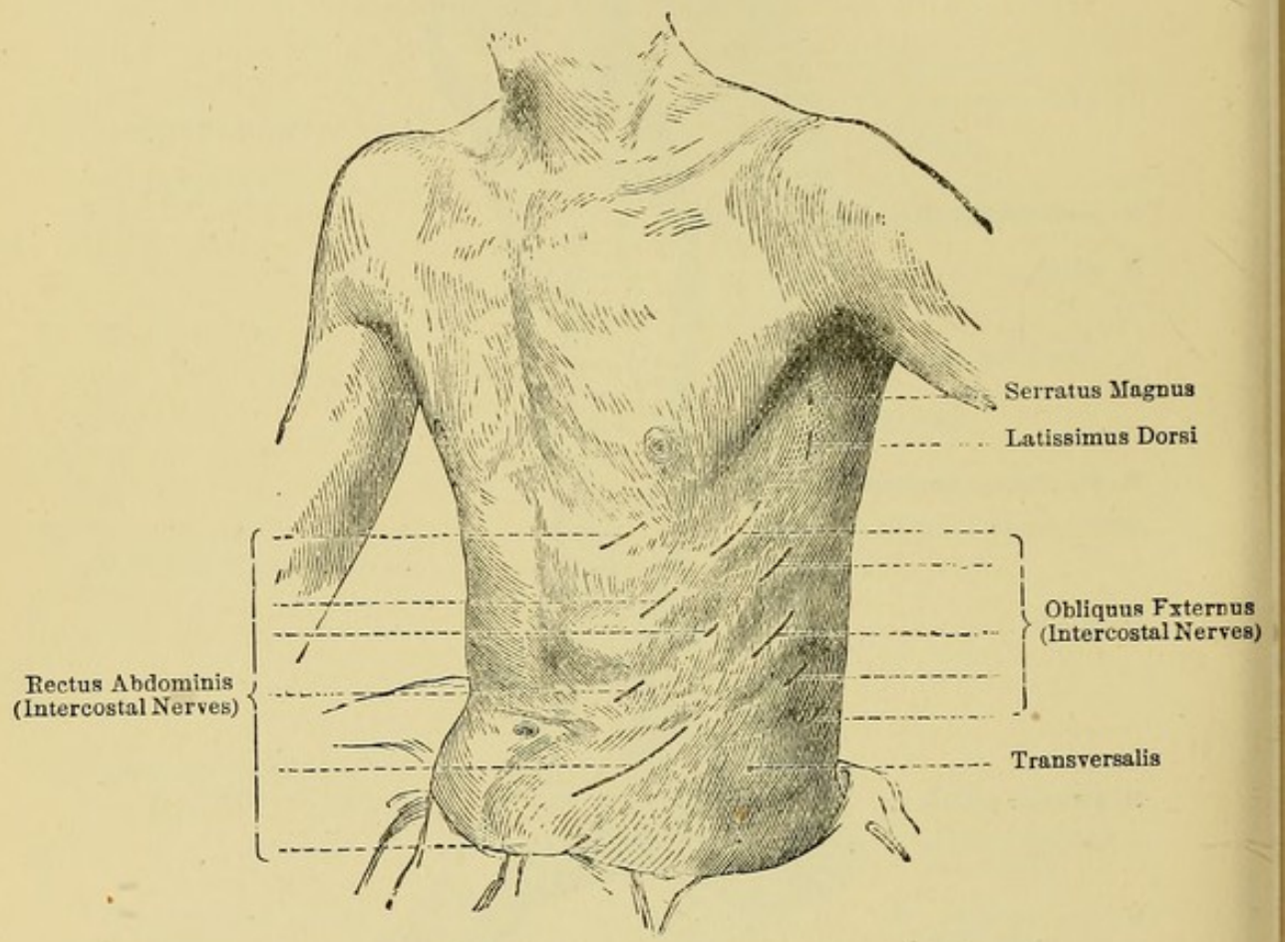


FIG. 89.

Motor Points on the Anterior Surface of the Trunk.

In using the Faradic (interrupted) current the polar method is not so important. It should, however, always be employed where great accuracy in diagnosis is required. In using the Faradic current for the purposes of treatment, both electrodes may be placed *at a short distance from each other over the muscle* to be acted upon. This method is less painful than when they are wide apart.

§ 68. The Use of Electricity as a Diagnostic in Cases of Paralysis.—In using electricity as a diagnostic agent in cases of paralysis, the reactions from nerve and muscle must be carefully distinguished, and the following points noted:—

1. *The minimum strength of current required to produce a contraction in the paralysed muscles.*—The corresponding muscles on the opposite (healthy) side of the body are then tested and any variations noted. When the paralysis is bilateral, the non-paralysed muscles are taken as a standard of comparison. In making this comparison, and in making comparative observations on different individuals, great care must be taken to have all the conditions (strength of current, position of electrodes, condition of muscles as to tension, position of the limbs, moisture of the skin, etc., etc.), as far as possible the same.

2. *The order of the polar reactions.*

3. *The character of the contractions, their rapidity, duration, etc.*

4. The *maximum amount* of muscular force which can be produced by strong currents.

Before describing the results which occur in disease, it will be necessary to consider the effects produced on the healthy tissues.

§ 69. The Effect of Electricity on Healthy Muscles and Motor Nerves.—Electricity has the property of exciting the functional activity of healthy muscles and motor nerves, the result of which is to produce muscular contractions.

Muscular contractions are only produced *when alterations occur in the strength of the current* (*i.e.*, when the current is made or broken, or when its strength is diminished or increased).

In health the contractions are short, sharp, and sudden; and the effects produced by stimulating the motor nerve and the muscular fibres directly (*i.e.*, the electrodes being applied

over the body of the muscle, and not over its motor nerve) are identically the same. This is due to the fact, that it is impossible to stimulate a healthy muscle without at the same time stimulating the motor nerve-endings which it contains; and it is probable that the contractions which result from *direct* stimulation of a healthy muscle, are due to stimulation of these motor nerves.

Where the nerve-endings are destroyed by disease, or paralysed artificially by curare, important alterations occur. Muscular contractions are no longer produced when the muscle is directly stimulated by rapidly interrupted currents, such as the Faradic. The character of the contractions produced by slowly interrupted continuous currents is modified; instead of being short, sharp, and sudden, they are slow, prolonged, and apt to become tetanic. Important alterations in the sequence of the polar reactions also occur. (See § 74.)

Since the functional activity of muscles and motor nerves is only excited when changes occur in the strength of the electric current, it follows:

1. That in the case of the interrupted or Faradic current (in which a variation of strength occurs with each interruption), a muscular contraction will accompany each break in the current, and since the interruptions produced by the automatic interruptor follow one another with extreme rapidity, an apparently continuous contraction or tetanic condition will result whenever the electrodes are applied.

The strength of the contractions produced by the interrupted current varies directly with the strength of the current. Very weak currents do not produce contractions.

2. That in the case of the continuous current, muscular contractions will not occur so long as the current strength is constant, *i.e.*, so long as the current is passing, but only when it is made or broken, or when sudden variations in its strength occur.

The effects of the continuous current are not, however, so simple as this statement would imply, for they vary with the relative position of the poles and with the strength of the current.

Since there are two poles (the *anode* or positive, and the *cathode* or negative), and since muscular contractions occur on making or closing, and on breaking or opening the current, it follows that there are four possible forms of contraction, viz.:

(*A*. When the negative pole is on the muscle or motor nerve, the positive pole on the distant or neutral point, *i.e.*, with descending currents.)

1. The contraction which occurs on closing=the cathodal closing contraction C.C.C.

2. The contraction which occurs on breaking or opening=cathodal opening contraction C.O.C.

(*B*. When the positive pole is on the muscle or motor nerve, the negative pole on the distant point, *i.e.*, with ascending currents.)

3. The contraction which occurs on closing=anodal closing contraction A.C.C.

4. The contraction which occurs on opening=anodal opening contraction=A.O.C.

These four forms of contraction occur in a definite order with currents of increasing intensity, viz.:

1. C.C.C.
2.[1] A.C.C.
3. A.O.C.
4. C.O.C.

As the current strength increases so does the intensity of each contraction. We may, therefore, represent the character of the contractions with a gradually increasing strength of current as follows:

1. Very feeble current. No contraction either on opening or closing with either pole.
2. Weak current, C.C.C.
3. Somewhat stronger current, C′.C′.C′. + A.C.C.
4. Still stronger current, C″C″C″ + A′.C′.C′. + A.O.C.
5. Strongest current, C‴C‴C‴ (cathodal tetanus) + A″C″C″ + A′O′C′. + COC.

§ 70. ALTERATIONS IN THE ELECTRICAL REACTIONS WHICH OCCUR IN DISEASES. THE ELECTRICAL CONDITION OF PARALYSED MUSCLES AND NERVES.

The alterations which occur in paralysis are either quantitative or qualitative.

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[1] The A.C.C. and A.O.C. often appear simultaneously. Sometimes the A.O.C. appears before the A.C.C.

When the lesion is situated *above the trophic nucleus* the electrical reactions are either *normal;* or *quantitative* alterations (simple diminution or simple increase, the polar reactions appearing in normal sequence, the character of the contractions natural, the results obtained by stimulating nerve and muscle the same) are observed. In such cases electricity is of little value as a *positive* diagnostic of paralysis; the evidence it affords is chiefly *negative.*

Where the lesion *suddenly destroys the trophic nucleus,* or so involves the nerve trunk as to *rapidly shut off the trophic influence of the nucleus,* as in severe forms of peripheral paralysis, very important qualitative as well as quantitative alterations in the electrical condition of the paralysed muscles occur. To these alterations Professor Erb has given the name of the '*reaction of degeneration.*'

We shall now consider a little more in detail the alterations in the electrical reactions which occur in cases of paralysis.

§ 71. *Normal electrical conditions.*—In the earlier stages of those cerebral and spinal paralyses in which the lesion is situated *above the trophic nucleus,* the electrical condition of the paralysed muscles may be normal. After a time when atrophic changes begin to occur, simple diminution appears. Normal electrical conditions are also seen in cases of so-called functional paralysis, and in those cases of peripheral paralysis, in which the lesion is so slight that it does not shut off the trophic influence, *i.e.,* in those forms of peripheral paralysis in which the rapid form of atrophy does not occur.

§ 72. *Simple diminution.*—The only alteration from the normal is that a stronger current (galvanic and Faradic) is required to produce a contraction. This form of alteration occurs in those cases of cerebral and spinal paralysis in which the lesion is situated *above the trophic nucleus.* Simple diminution accompanies the slow and moderate form of atrophy which occurs in such cases. The amount of the decrease corresponds to the amount and stage of the atrophy. In the later stages of very chronic cases, the electrical contractility of the paralysed muscles may have almost entirely disappeared.

§ 73. *Simple increase.*—In this condition, which is rare, a weaker current (both galvanic and Faradic) produces

muscular contractions, than in health; but the character of the contractions, and the sequence of the polar reactions, etc., are normal.

Simple increase must be carefully distinguished from the increase of galvanic irritability, which is one of the most characteristic features of the 'reaction of degeneration.'

Simple increase is said to occur in the earlier stages of some cases of hemiplegia; and in some cases of spinal paralysis, in which irritative (spastic) phenomena are present, but in which there is no muscular atrophy.[1]

§ 74. *The 'reaction of degeneration.'*—Where the lesion destroys the nerve nucleus (trophic centre), or destroys or seriously injures the nerve trunk, marked alterations, both qualitative and quantitative, occur.

The alterations, which constitute the 'reaction of degeneration,' are as follows:

1. With the development of the degeneration of the nerve, the electrical excitability of the nerve (both to the galvanic and Faradic currents) diminishes, and at the end of about a fortnight ceases altogether.

2. The rapidly interrupted (Faradic) current ceases to cause muscular contractions when applied directly to the muscle; for the motor nerve endings in the muscle are degenerated, and the muscular fibre itself does not respond to currents of momentary duration.

3. During the first ten days or so there is *simple diminution* of the contractility produced by the galvanic stimulation of the muscle; the excitability of the muscles to slowly interrupted galvanic currents then *increases*, and,

4. The following *qualitative* changes are observed:—The sequence of the polar reactions is altered, the anode takes the place of the cathode. The order of appearance of the polar reactions is therefore:—

1. A. C. C.		1. C. C. C.
2. C. C. C.	instead of	2. A. C. C.
3. C. O. C.		3. A. O. C.
4. A. O. C.		4. C. O. C.

[1] In all the cases of spastic paralysis which I have had an opportunity of examining, there was *simple* diminution instead of *simple* increase.

5. The character of the contractions is modified; instead of being short, sharp, and sudden, they are slow in appearing, prolonged in duration, and apt to become tetanic, even when produced by weak currents.

To sum up then, where the 'reaction of degeneration' is present, the galvanic stimulation of the muscle produces contraction more easily than in health, but the character of the contractions, and the sequence of the polar reactions, are altered. This increase to the slowly interrupted continuous current occurs at a time when stimulation of the nerve (both by the galvanic and Faradic currents) and of the muscle by the Faradic current produces no contraction.

The increased excitability of the muscle to the galvanic current continues for a longer or shorter period in different cases. In incurable cases, after twenty or thirty weeks it gradually declines, and finally ceases (see fig. 92); then the muscle, like the nerve, ceases to respond to all forms of current. In cases which recover, it lasts for a few weeks, and then gradually diminishes, the normal reactions reappearing. (See figs. 90, 91.)

The following diagrams, taken from Erb, illustrate the course and duration of the 'reaction of degeneration' in different cases:—

Rapid Recovery.

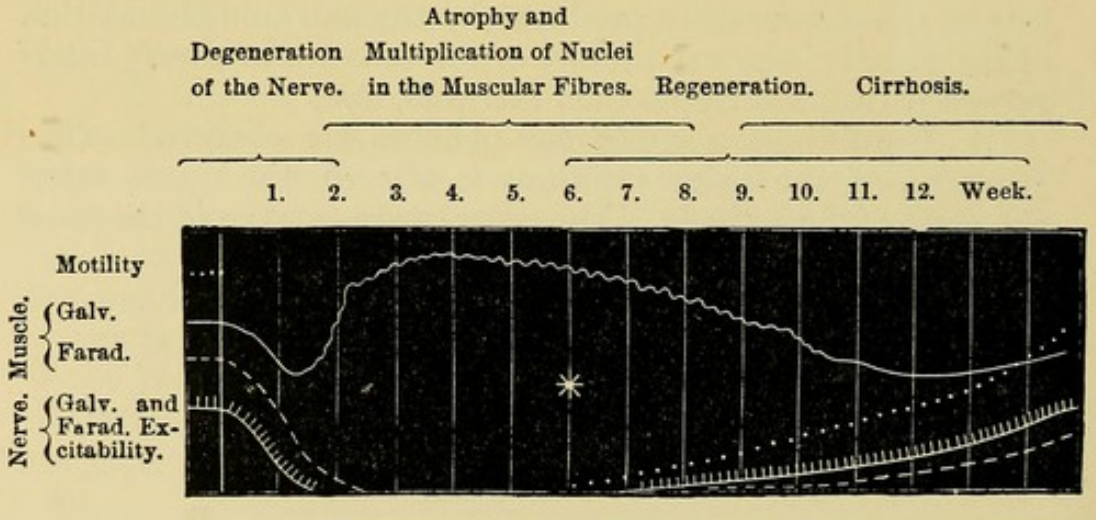


FIG. 90.

Slow Recovery.

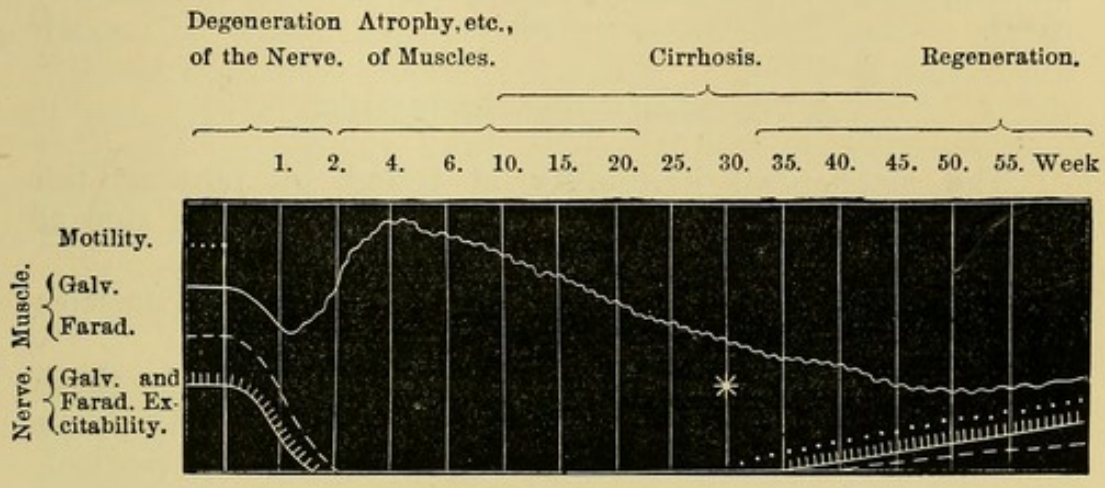


FIG. 91.

No Recovery.

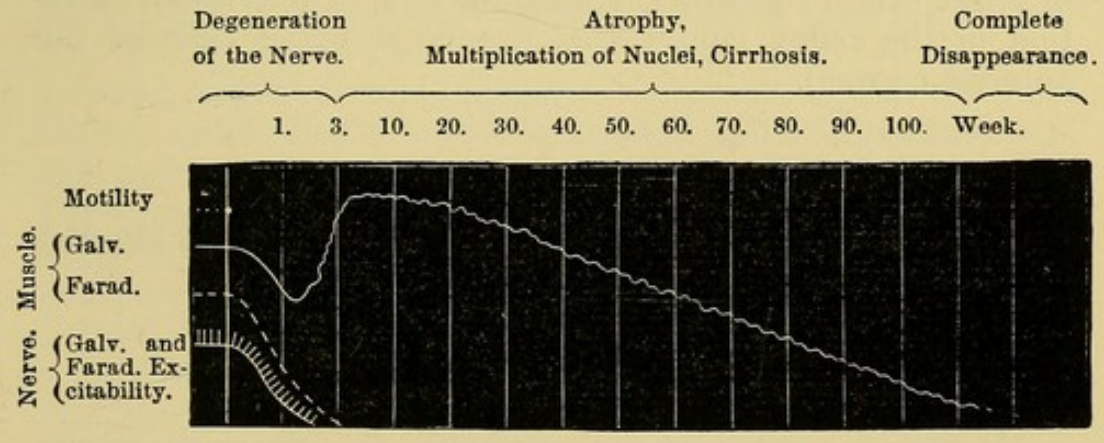


FIG. 92.

§ 75. *Clinical significance of the reaction of degeneration.*—The 'reaction of degeneration' occurs in all severe peripheral paralyses, and in all cases in which the nerve nuclei are rapidly destroyed; it, therefore, occurs in polio-myelitis anterior acuta, and acute myelitis involving the anterior cornu. In transverse myelitis it is, of course, only observed in those muscles supplied by nerves arising from the affected segment. (See fig. 71, page 58.)

According to some writers the 'reaction of degeneration' is also seen in progressive muscular atrophy and bulbar paralysis; personally, I have never observed it in such cases.

§ 76. MECHANICAL IRRITABILITY.

The mechanical irritability is tested by tapping the paralysed muscles with the tip of the finger, the side of the hand, or a percussion hammer.

As a rule the mechanical excitability of paralysed muscles is lowered.

Increased mechanical excitability occurs:—(1) In those cases in which the electrical (galvanic) excitability is in excess, *i.e.*, where the 'reaction of degeneration' is well marked. According to Erb the increased excitability to mechanical stimuli appears in these cases later than the increased excitability to galvanic stimuli.

(2) Where the deep reflexes are excessive, as in cases of lateral sclerosis. Here the increased excitability is probably of reflex origin; possibly it may be due to perverted nutrition of the reflex centre (*i.e.*, the large nerve cells of the anterior cornua).

§ 77. THE TONICITY OF THE MUSCLES.

In cases of spinal cord disease the tonicity of the muscles may be normal, diminished or increased.

§ 78. *Flaccidity or Diminished Tonicity.*

It may be stated as a general rule, that flaccidity occurs in those cases in which the muscle is atrophied. Exceptions occasionally occur—where, for example, rigid muscles become atrophied, a combined condition of tension and atrophy may for a time, at least, be present, as in amyo-trophic lateral sclerosis.

Flaccidity, therefore, occurs in those spinal diseases in which the lesion involves the anterior cornua. It is also the usual condition in functional derangements, in mild cases of myelitis, and in severe cases of myelitis prior to the onset of secondary descending degeneration.

§ 79. *Rigidity and Tension (increased Tonicity.)*

Rigidity and tension occur in those cases of spinal cord disease in which there is irritation of motor nerve tissue. The irritation may be direct or reflex. Possibly in some cases it (the rigidity) is due to arrested cerebral control. Other irritative phenomena, such as twitchings, tremors, cramps, spasms, contractures, are often associated with the increased muscular tension.

Clinically, rigidity and tension are met with in the following affections :—

(1) *Meningitis.* — Here the rigidity may result from (*a*) direct irritation of the anterior (motor) roots ; (*b*) irritation of the posterior (sensory) roots, and resulting reflex spasm ; (*c*) increased excitability of the grey matter of the cord.

(2) *Sclerosis of the pyramidal tracts* (primary lateral sclerosis, and secondary degenerations).—The exact manner in which lesions of the crossed pyramidal tracts cause tension in the muscles is still undetermined. Some authorities say the rigidity is due to diminished or arrested cerebral control, and to the increase of the reflexes (especially of the deep reflexes) which results therefrom. Others think that irritation of the motor fibres in the pyramidal tracts is produced by the process of secondary descending degeneration, that the irritation is propagated to the multipolar nerve cells of the anterior cornua, and thence to the muscles. Both explanations are probably correct; but the latter is certainly the most important.

(3) In some cases of *hysterical paraplegia* the muscles are rigid, the deep reflexes are increased, in short, the condition exactly resembles the spastic paraplegia of organic disease.

The distribution of the rigidity.—In cases of meningitis the rigidity chiefly involves the flexors ; the thigh, for example, is flexed on the abdomen, and the leg on the thigh. In lateral sclerosis the extensors are most affected : in advanced cases the patient lies in bed with the legs rigidly extended, the thighs adducted, and the feet inverted : in the earlier stages he is able to walk with the help of sticks. The term spastic or spasmodic paraplegia is usually given to these cases.

§ 80. *Fibrillary twitchings or tremors.*

Fibrillary twitchings are produced by the contraction of individual muscular fasciculi. The skin covering the muscular fibre appears suddenly raised and stretched as if a thread were made tense beneath it. The patient is generally conscious of a 'quivering' sensation in the affected part.

Fibrillary twitchings were formerly thought to be pathognomonic of progressive muscular atrophy. We now know that they occur in many other conditions. They are seen in some cases of hypochondriasis, in which the patient gives great attention to his limbs, believing that he is the subject of spinal cord disease. As Professor Charcot points out, these patients are usually medical men or medical students. Fibrillary twitchings are also observed in cases of functional disorder associated with 'irritable' weakness of the spinal cord. They are seen in all cases in which motor nerve tissue is undergoing destruction, but especially where motor nerve cells are being slowly destroyed. Fibrillary twitchings occur therefore in many different forms of spinal disease. They are constantly met with in progressive muscular atrophy.[1]

§ 81. *Contracture.*

In many cases of atrophic paralysis a permanent condition of shortening and consequent deformity results, to which the term *contracture,* is applied. In some of these cases the rigidity depends upon cirrhosis of the affected muscles (atrophy of the muscular fibres, the production and subsequent contraction of fibrous tissue). In other cases the contracture is due to unrestrained action of one set of muscles, their opponents being paralysed. Mechanical pressure on paralysed parts may also aid in its production. Contractures are common as the result of infantile paralysis. In pseudo-hypertrophic paralysis, talipes equinus is usually present.

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[1] I do not mean to imply that fibrillary twitchings can always be seen on casual observation of a case of progressive muscular atrophy. They will, I think, always be noticed during the active period of the disease, provided that the affected muscle is observed for a sufficiently long period. Exposure of the affected part, or gentle irritation of the muscle, such as stroking the skin, sometimes excites the contraction.

§ 82. THE CONDITION OF THE REFLEX MOVEMENTS.

The examination of the reflexes is a most important means of ascertaining the condition of the segment through which the reflex passes.

§ 83. *Persistence of the reflexes.*—The *presence* of a reflex in any segment shows that the reflex path is still open, *i.e.*, that there is no serious disease in that portion of the segment through which the reflex impulse travels.

The *presence* of a reflex in any segment does not of necessity show that there is *no* serious disease in the segment. The postero-internal columns, the lateral columns, and the columns of Türck may, of course, be absolutely destroyed without any injury to the reflex track.

The *absence* of a reflex in any segment does not *necessarily* prove that the segment is diseased; for the reflexes are sometimes absent as a physiological idiosyncrasy, and in most healthy individuals it is difficult or impossible to demonstrate the gluteal, epigastric and inter-scapular skin reflexes; the ankle clonus, too, is never present except in disease. Again, the reflex impulse may be 'blocked' outside the cord as the result of lesions of the centrifugal or centripetal fibres of the reflex arc. Possibly, too, the cerebral control may be so excessive as to completely inhibit the reflex movements.

§ 84. *Abolition of the reflexes.*—Spinal lesions which destroy the posterior roots, postero-external columns, that portion of the grey matter through which the reflex travels, the anterior root-fibres, or the anterior roots, will cause a break in the reflex arc.[1]

Indiscriminate lesions may involve any of these parts, and may therefore produce abolition of the reflex movements. The system lesions, in which the reflexes are abolished, are

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[1] It is probable that the superficial reflexes pass directly into the posterior horn of grey matter, while the deep reflexes pass through the postero-external column. It is possible in this way to explain the undoubted clinical fact, that in the earlier stages of locomotor ataxia, *i.e.*, while the lesion is limited to the postero-external column, the deep reflexes are abolished, but the superficial reflexes may be retained. (See page 25.)

locomotor ataxia (sclerosis of the postero-external columns), polio-myelitis anterior acuta, and progressive muscular atrophy. In the two latter affections the reflexes are only completely abolished when all of the multipolar nerve cells of the affected segment are destroyed. (See figs. 37 and 46.)

§ 85. *Exaggeration of the reflexes* is in the great majority of cases due to disease of the lateral columns, and is usually associated with secondary descending degeneration. It may, however, result from increased excitability of the grey matter.

It is important to remember that disease of the lateral columns of the cord usually produces exaggeration of both forms of reflex movement (superficial and deep). In cerebral lesions, on the contrary, the deep reflexes are exaggerated, but the superficial reflexes are diminished or abolished.

In some cases of spastic paralysis in which the deep reflexes are in marked excess, little or no movement results on tickling the soles. The usual flexion of the leg on the thigh, or thigh on the abdomen, does not occur in these cases, because of rigidity of the opponent muscles. Careful observation in such a case will usually show that the stiffness and rigidity of the limb are decidedly increased by the tickling.

§ 86. *The reflex functions of individual segments.—The method of testing the reflexes.*

The superficial reflexes.—Dr Gowers, who has given great attention to this subject, describes a plantar, gluteal, cremasteric, abdominal, epigastric, and interscapular skin reflex.

The plantar reflex is almost always present in health. The abdominal reflex can also usually be obtained. The cremasteric reflex is, as a rule, well marked in children, but often absent in adults. The gluteal, epigastric, and interscapular reflexes can seldom be demonstrated in the healthy individual.

1. *The plantar reflex* is obtained by irritating the skin of the sole, the result being a contraction of the foot muscles, or when the reflex radiates, as it often does, a contraction of the muscles of the thigh and leg. When confined to the foot muscles the reflex passes through the lower part of the lumbar enlargement.

2. *The gluteal reflex* is obtained by irritating the skin of the buttock, and consists in contraction of the gluteal muscles.

It passes through the cord at the level of the fourth or fifth lumbar nerves.

3. *The cremasteric reflex* is obtained by tickling the skin on the inner side of the thigh, and consists in retraction of the testicle on the same side. It passes through the cord at the level of the first and second lumbar nerves. This reflex is often absent in the adult.

4. *The abdominal reflex* is obtained by stimulating the skin of the side of the abdomen in the nipple line. It consists in contraction of the abdominal muscles, and passes through the lower part of the dorsal region of the cord from the eighth to the twelfth nerves.

5. *The epigastric reflex* is obtained by stimulation of the side of the chest in the fifth and sixth interspaces. It consists in the dimpling of the epigastrium from contraction of the (?) upper part of the rectus abdominalis. It passes through the cord at the level of the fourth, fifth, and sixth dorsal nerves.

6. *The interscapular reflex* is produced by stimulation of the skin between the scapulæ, and consists in contraction of some of the scapular muscles. When slight it is chiefly marked at the posterior axillary fold (teres); when more considerable, it involves almost all the muscles attached to the scapula (trapezius teres, serratus), and even moves the bone a little outwards. It passes through the cord at the level of the three upper dorsal and two lower cervical nerves.

To elicit the superficial reflexes, the skin should be gently scratched with a fine pointed instrument, such as the point of a pencil, or the end of a tuning fork. The finger does well enough in most cases, but a fine point is better.

§ 87. *The deep reflexes.*[1]—The best known are the patellar tendon reflex, and the ankle clonus. In diseased conditions, reflex movements can often be produced by tapping the tendons of the upper extremity.

The *knee-reflex* (*patellar-tendon-reflex*) is best obtained by striking the ligamentum patellæ when the knee is semiflexed and the foot at rest. The patient should be seated with his

[1] The exact character of the so-called tendon-reflexes is, as I have previously mentioned, still a matter of debate.

legs dangling over a high table in a pendulum-like manner, as shown in fig. 93; or, the leg may be crossed over its fellow, as represented in fig. 94. The tendon should be struck just below the patella with a percussion hammer, the side of the

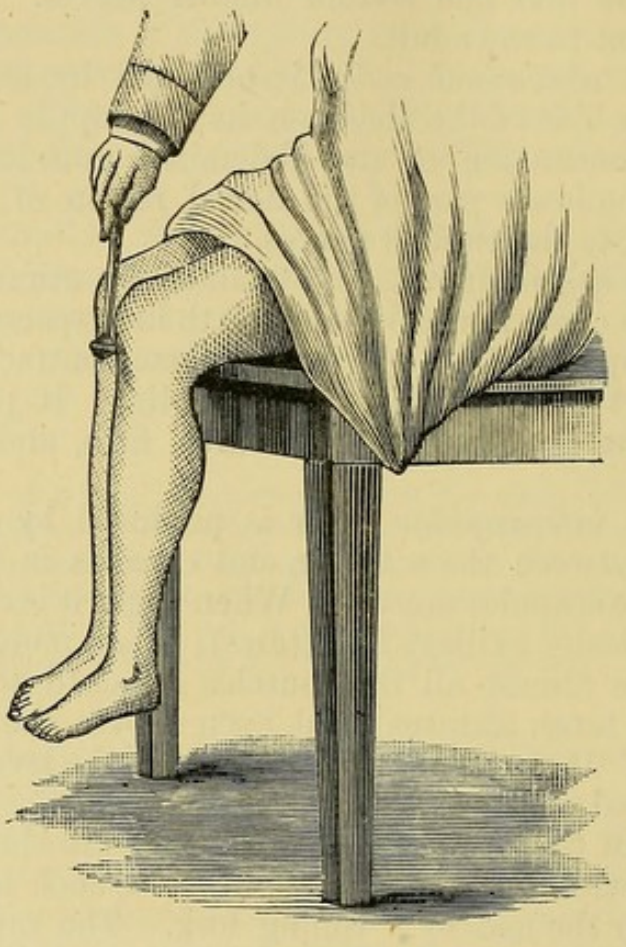


FIG. 93.

Method of obtaining the Patellar Tendon Reflex.

hand, or the ear-piece of a stethescope. The knee must be quite relaxed. When there is any doubt as to the presence of the phenomenon, I am in the habit of bandaging the patient's eyes; it is then possible to strike either tendon without the

patient being aware that you are going to do so. Care must be taken not to confound the movement of the leg, which results from the impulse of the blow, with the movement which depends upon contraction of the muscle. The reflex centre

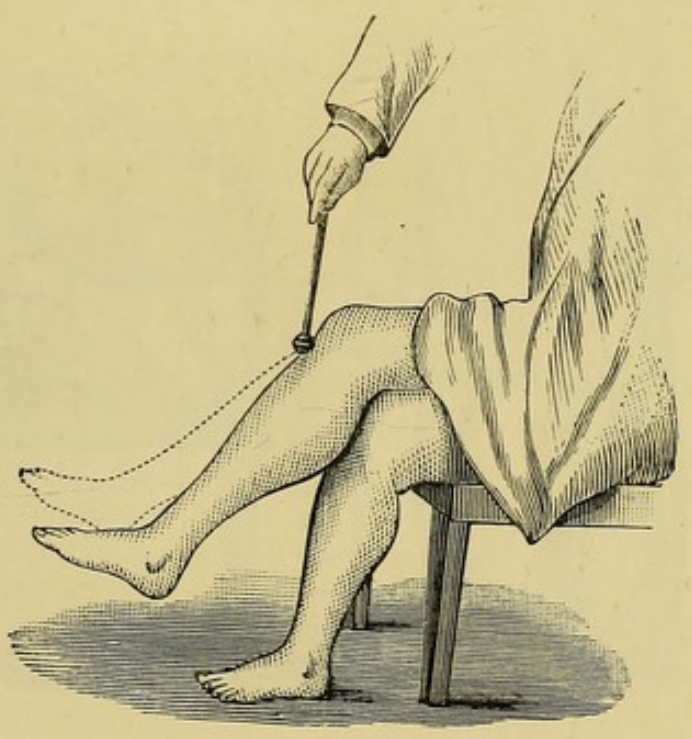


FIG. 94.

Method of obtaining the Patellar Tendon Reflex.

is situated in the lumbar region of the cord, in the segments corresponding to the second, third, and fourth lumbar nerves.

The ankle clonus is a rhythmical movement of the foot, due to rhythmical contraction of the calf muscles. In order

to obtain it the foot must be suddenly flexed at the ankle by pressure upon the balls of the toes; the leg should be extended, but not fully. (See fig. 95.)

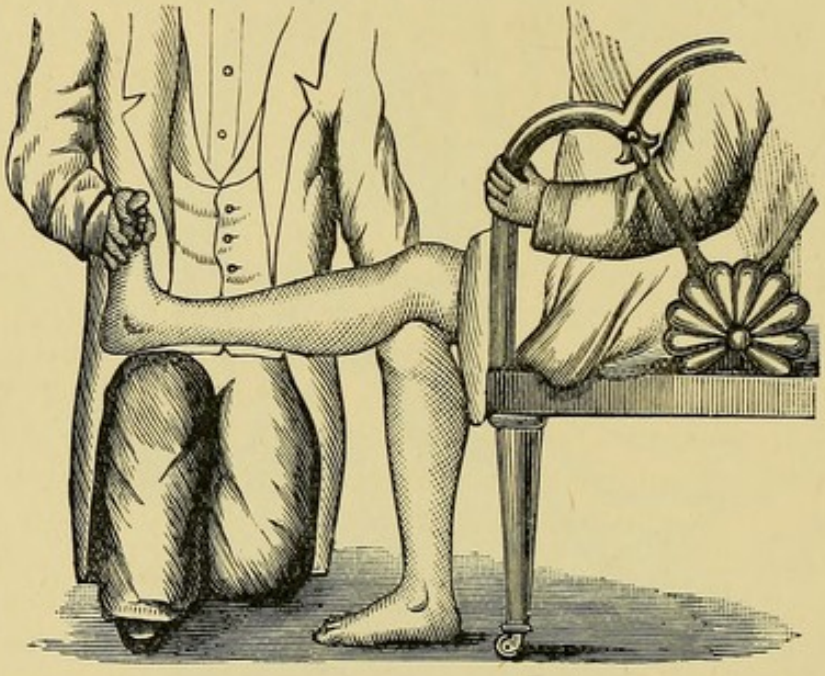


FIG. 95.

Method of obtaining the Ankle Clonus.

A modification of the ankle clonus has been described by Dr Gowers, and termed by him the *front-tap contraction.* He states that it is a very delicate test of morbid irritability, and can often be obtained when the ankle clonus cannot. To obtain it a tap must be made on the muscles of the front of the leg during passive flexion of the ankle.

The ankle clonus cannot be obtained in health. It is seen in cases of spastic paraplegia; and is very valuable evidence, though not pathognomonic, of organic disease, viz., sclerosis of the lateral columns. It is also seen in the hysterical (functional) form of spastic paraplegia; this point will afterwards be referred to in more detail. (See page 153.)

Deep reflexes can sometimes be obtained in the upper extremity. They are seldom, if ever, present in health, and are chiefly valuable as evidence of secondary descending degeneration resulting from cerebral lesions.

THE ORGANIC REFLEXES.

The more important organic reflexes which have their centres in the spinal cord, are the vesical and rectal.

§ 88. THE VESICAL REFLEX.[1]

Micturition is a complex process, the exact mechanism of which is not, perhaps, yet definitely understood. The nervous and muscular arrangements concerned are as follows:—

The neck of the bladder is surrounded by circular muscular fibres—the sphincter vesicæ muscle—which are supposed to be maintained in a constant state of contraction. By virtue of the action of the sphincter, and, to a less degree, of the resistance of the elastic fibres of the urethra, the exit of urine is prevented. The contraction of the sphincter is due to the action of a tonic centre which is situated in those segments of the spinal cord which correspond to the 2d, 3rd, and 4th sacral nerves. The action of the tonic centre can be inhibited, *i.e.*, the sphincter can be relaxed, by a voluntary effort. The course of the inhibitory fibres in the cord is undetermined, but they probably pass through the lateral columns. Dr Gowers supposes that the centre for the sphincter can be inhibited, *i.e.*, the muscle can be relaxed, *reflexly*, by sensory stimuli from the mucous membrane of the bladder itself. Such a supposition would undoubtedly well explain

[1] The mechanism of urination belongs, of course, to the physiology of the cord. The description of the process has been purposely deferred, in order that the derangements which occur in disease may be more readily understood.

some points of the process, and is adopted in the following description :

The expulsion of urine is effected by the contraction of the muscular fibres in the wall of the bladder—the detrusor urinæ —aided by the contraction of the voluntary muscles in the abdominal wall.[1] According to Goltz, the contraction of the detrusor is a purely reflex act. The reflex centre, which is

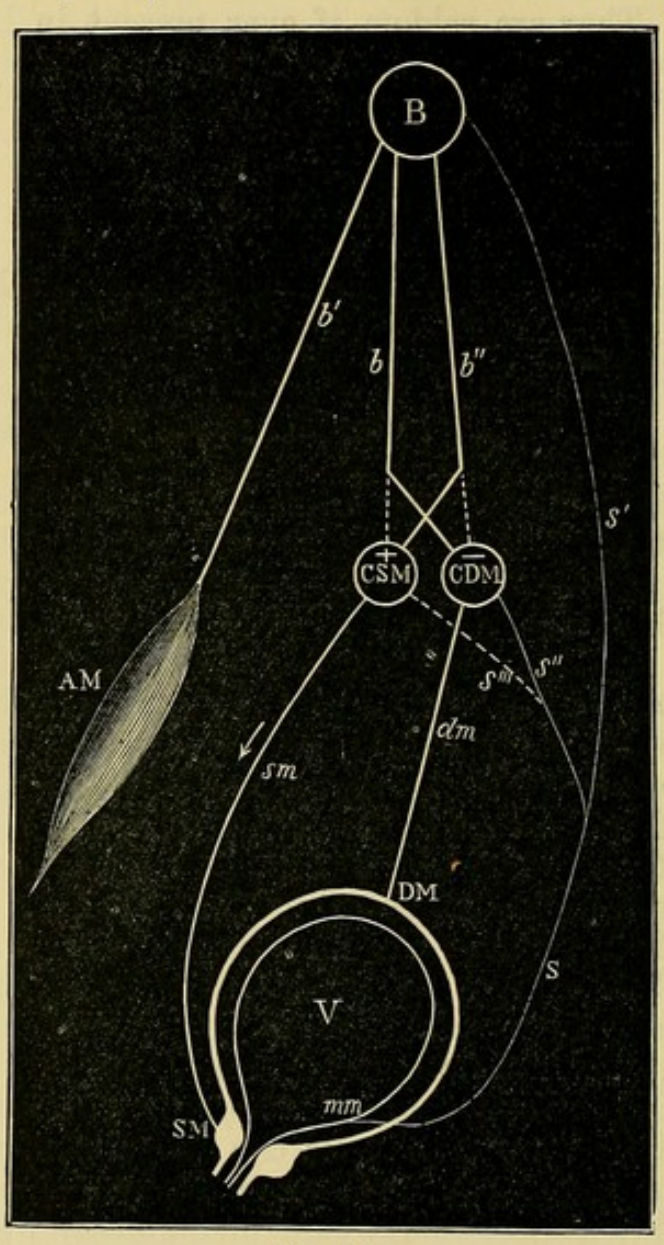


FIG. 96.

[1] Possibly the tonic contraction of the sphincter may be due to some local nervous mechanism.

situated in the segments of the spinal cord corresponding to the 3d, 4th, and 5th sacral nerves, is set into action by stimulation of the sensory fibres in the mucous membrane of the bladder.

Many authorities believe that the reflex process can be strengthened, and some say that it can be set into motion by a voluntary effort. Like many other reflex acts, it can be inhibited by an effort of the will.

It will be seen from this description that the centres for the detrusor and sphincter muscles are antagonistic, and that an impulse from the brain, which inhibits the action of the sphincter centre, of necessity excites the action of the detrusor; and *vice versâ*. The inhibitory fibres probably pass down the lateral columns of the cord.

The parts concerned in micturition, while at rest, are diagrammatically represented in fig. 96.

DESCRIPTION OF FIG. 96.

Diagrammatic representation of the parts concerned in the mechanism of micturition while at rest. (*After Gowers, but considerably modified.*)

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, sensory fibre 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 detrusorc entre, as shown in fig. 2. *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.

The mechanism of micturition then, so far as it is at present known, would seem to be as follows:—

1. When the bladder becomes sufficiently full of water, the sensory nerve filaments in the mucous membrane are stimulated, and an impression is conveyed along the sensory nerves to the reflex centres for the detrusor and sphincter muscles in the spinal cord and to the sensorium.

2. As a result of the sensory impression conveyed to the brain the desire to urinate is experienced.

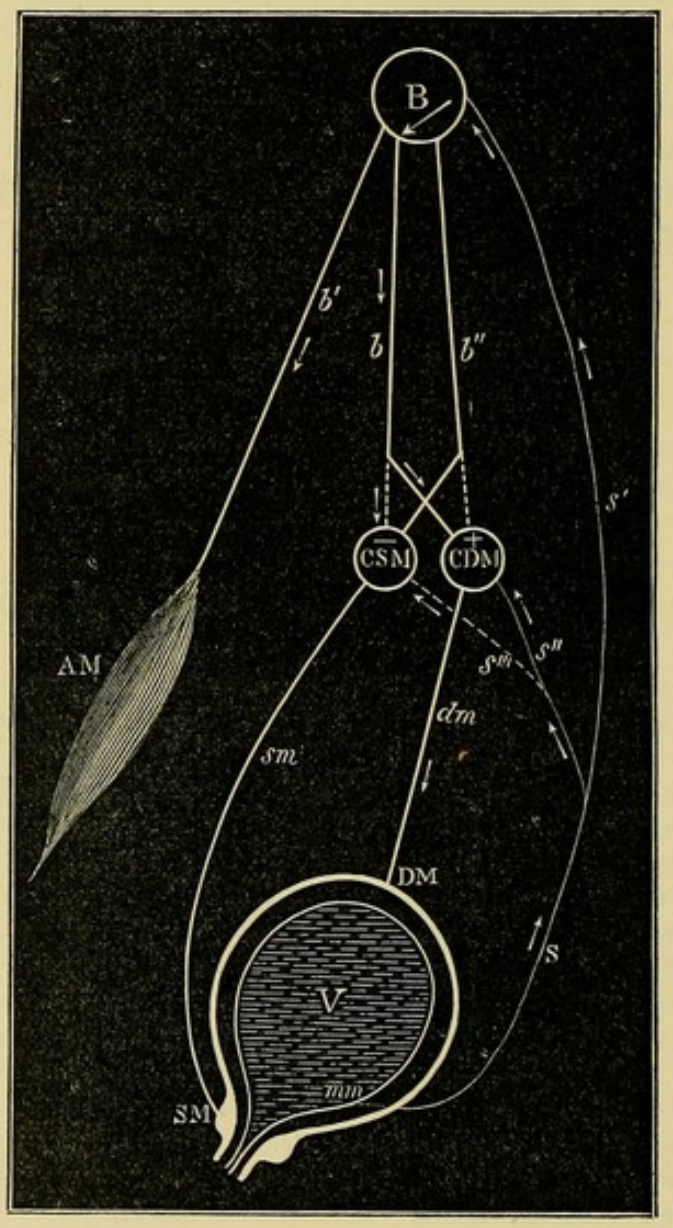


Fig. 97.

3. As a result of the reflex impulse carried to the spinal cord, the action of the detrusor centre is stimulated, while the action of the sphincter centre is inhibited.

If the circumstances for urination are favourable, an impulse is sent from the brain to: *(a)* The tonic centre for the sphincter, inhibiting its action, and causing relaxation of the sphincter muscle; *(b)* The centre for the detrusor, strengthening its action which has been already excited by the reflex impulse from the bladder; and to *(c)* The abdominal muscles, causing their active contraction. In health all these processes are simultaneously accomplished, and urination is the result. The mechanism of the process is diagrammatically represented in fig. 97.

If the circumstances are not convenient for urination, the process can be prevented by: *(a)* Voluntary inhibition of the centre for the detrusor; *(b)* The (voluntary) contraction of the urethral muscles at the neck of the bladder; and possibly, too, the strengthening of the tonic centre for the sphincter.

The manner in which the process is inhibited by a voluntary effort, is diagrammatically represented in fig. 98.

DESCRIPTION OF FIG. 97.

Diagrammatic representation of the parts concerned in the process of micturition while in action.

The bladder (v) is full of urine. The mucous membrane (m m) is stimulated, an impression is generated, and is carried by the sensory (centripetal) nerve (S) to the brain (B), and to the spinal centres for the detrusor (C D M), and sphincter (C S M). From the brain an impulse is sent (1) along the nerve (b) which strengthens the action of the detrusor centre (C D M), and inhibits the action of the sphincter centre (C S M); (2) along the nerve (b′), which throws the abdominal muscles (A M) into action.

The reflex impulse which has passed from the mucous membrane of the bladder to the spinal cord, excites the action of the detrusor centre, and inhibits the action of the sphincter centre.

The final result is contraction of the detrusor muscle (D M), relaxation of the sphincter muscle (S M); and the expulsion of urine. The arrows indicate the direction of the nerve 'currents.'

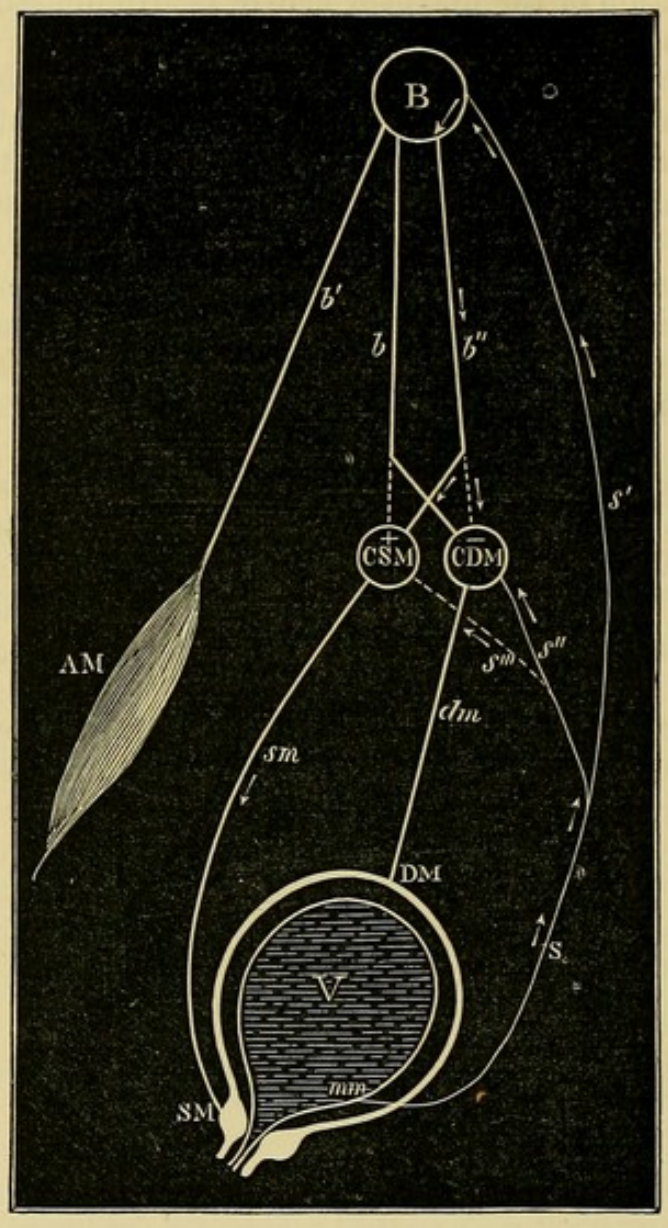


FIG. 98.

Diagrammatic representation of the mechanism of micturition, showing the process by which the act can be inhibited by a voluntary effort.

The bladder is full of urine. A reflex impulse passes from the mucous membrane to the spinal cord and brain; but the conditions for urination are not favourable. An impulse is therefore sent from the brain (B) along the nerve (b″), which inhibits the action of the detrusor centre (C D M), and strengthens the action of the sphincter centre (C S M). The final result being firm contraction of the sphincter muscle (S M), and *non*-expulsion of urine. The arrows indicate the direction of the nerve 'currents.'

§ 89. *Alterations of the process of urination which occur in disease.*—Disorders of urination are very frequent in disease, and result from derangement of the mechanism which I have just described; and since the chief part of that mechanism is situated in the lower portion of the spinal cord, it follows that the greatest disturbance in urination will occur when the lumbar portion of the cord is the seat of the lesion.

The following are the effects produced by lesions of the different parts of this nervous mechanism:

1. *Excessive stimulation of the centripetal nerves.*—When the sensory nerve filaments in the mucous membrane of the bladder are abnormally sensitive, as in cases of cystitis, a small quantity of urine will suffice to set up the reflex process; increased frequency of micturition and spasmodic contractions of the detrusor muscle result. A foreign body (such as a stone) will produce excessive stimulation of the sensory nerves, even when the mucous membrane is healthy, but as a matter of fact, cystitis is nearly always present in such cases. Irritation in the rectum may also excite the reflex mechanism. Some cases of nocturnal incontinence in children are probably in-induced in this manner.

2. *Destruction of the sensory (centripetal) nerve filaments* will, of course, prevent the reflex impulse passing to the spinal cord. Expulsion of urine can still to some extent be effected by voluntary effort, chiefly by means of the abdominal muscles. This condition is of more theoretical than practical importance. It might result from the pressure of a tumour or inflammatory products on the nerve trunk or posterior nerve roots.

3. *Destruction of the reflex centres in the spinal cord.*—Destruction of the reflex centres may result from myelitis, hæmorrhage into the cord, traumatic injuries, etc. The sphincter and detrusor muscles are, of course, paralysed. There is generally paraplegia and paralysis of the rectum.

Sudden injuries (traumatic and inflammatory, etc.) of the spinal cord above the lumbar region are also attended with arrested functions of the urinary centres. In cases of this description, the urinary reflex is re-established as soon as the effects of shock pass off (provided, of course, the lumbar cord remains unaffected). In many of these cases the conducting paths to and from the brain are interrupted with the results described below.

Destruction of the detrusor centre produces paralysis of the detrusor muscle, as a result of which urine collects in the bladder (retention): after a time the sphincter gives way, and incontinence occurs.

When the action of the detrusor centre is impaired but not destroyed, the paralysis is incomplete. In these cases the contraction of the abdominal muscles takes a larger share in the process than in health. The expulsive power is diminished; the patient cannot empty the bladder when lying on his back; in order to complete the process satisfactorily, he has to stand up, *i.e.*, to place the parts in the best hydrostatic condition for the exit of urine.

Paralysis of the detrusor from destruction of its reflex centre is almost invariably associated with paralysis of the sphincter.

Destruction of the sphincter centre produces paralysis of the sphincter; the urine dribbles away; this condition is termed incontinence of urine. When the sphincter is paralysed, any sudden movement on the part of the patient—laughing, coughing, etc.—forces away urine.

Paralysis of the sphincter is very rare *per se*. It is nearly always associated with a similar affection of the detrusor muscle and with paralysis of the rectum.

4. *Destruction of the centrifugal (motor) nerves.*—Destruction of the motor nerve to the detrusor muscle will, of course, produce paralysis of that muscle. As I have previously remarked, this condition is seldom, if ever, met with in practice.

5. *Interruption and destruction of the conducting paths to and from the brain above the reflex centre* are of frequent occurrence. If the lesion is a sudden one, the shock to the urinary centres may cause temporary arrest of function. In chronic cases the effects of the lesion vary with its position and extent. When the sensory conductors or sensory perceptive centres are affected, the desire to urinate is not perceived; the reflex arc is uninjured, and as soon as the bladder becomes sufficiently distended with urine, it is evacuated unconsciously. It must be remembered, therefore, that the involuntary discharge of urine and fæces in cases of coma does not necessarily imply any paralysis of the bladder or rectum.

When the motor and inhibitory fibres are alone interfered

with, the desire to urinate is perceived, but the act takes place quite independently of volition. It can neither be assisted nor prevented.

INCONTINENCE AND RETENTION.

Incontinence and retention of urine are the symptoms which result from the lesions I have just described.

§ 90. *Incontinence of urine.*—The escape of urine either against the will or without the knowledge of the patient, and without his being able to control its flow—may be due to:

1. *Mechanical causes*—Such as a vesico-vaginal fistula.

2. *Spasmodic contraction of the detrusor muscle.* (Spasmodic incontinence.) In spasmodic incontinence the involuntary discharge of urine occurs at intervals, and between times the patient has full control over his bladder. This is one of the most frequent causes of the nocturnal incontinence of children. The spasms may be due to a local source of irritation acting reflexly, as worms in the intestine; or, it may be part and parcel of a general spasmodic condition (epilepsy). Occasional nocturnal incontinence should always therefore suggest the possibility of epileptic fits.

3. *Atony or paralysis of the sphincter vesicæ.*—Where the sphincter is completely paralysed the incontinence is *constant*, the patient never has command over his bladder, the urine dribbles away, and is liable to be forced away by any sudden effort, such as coughing. The urine is often ammoniacal.

Where there is simply atony of the sphincter, the incontinence may only occur occasionally. This is probably another cause of nocturnal incontinence in children; the incontinence rarely occurs during the day; the atony becomes aggravated during sleep; these patients are frequently delicate, but there are no associated nerve symptoms such as are present in most cases of paralytic incontinence; the urine is normal; the condition is generally easily amenable to treatment. Paralysis of the sphincter sometimes results from local injury, as after the rapid dilatation of the female urethra, or inflammatory affections of the muscle. It frequently follows retention, the sphincter after a time

yielding to the pressure of the retained urine, and incontinence resulting. In these cases the important diagnostic point to be determined is,—What is the cause of the retention? Is it due to a local (generally temporary) or central (often permanent) condition?

In all cases of incontinence of urine, especially where there is no associated condition of the rectum and limbs, a careful examination of the bladder should be made with the catheter, etc.

4. *Cerebral Incontinence.*—Where the urinary reflex arc is uninjured, but where there is a lesion (in the cord above the lumbar region or in the brain), which interrupts the conductors or destroys the cerebral centres, the urine is discharged at irregular intervals, and in a good stream, but the desire to urinate is not perceived, and the act cannot be restrained. This form of incontinence may, for the sake of convenience, be termed *cerebral.*

The differential diagnosis of a case of incontinence.—The points to be determined are:—

1. Is the incontinence mechanical, spasmodic, paralytic, or cerebral?
2. If spasmodic, is it due to local or central causes?
3. If paralytic, is the paralysis due to local or central conditions?
4. What is the exact seat and pathological character of the lesion?

In trying to solve these questions, attention must be directed to the following circumstances:—

1. The local condition of the urethra, bladder, rectum, etc. (Careful local examination.)
2. The exact character of the incontinence. (See above.)
3. The history and mode of commencement of the attack.
4. The associated nerve symptoms, especially the condition of the other parts supplied by the lumbar and sacral nerves.

When the incontinence is due to central causes, an anatomical and pathological diagnosis must be made, in accordance with the usual plan of diagnosis in spinal and cerebral affections.

The chief points of differential distinction between spasmodic and paralytic incontinence are shown in the following table:

Tabular Statement showing the Differential Diagnosis of Spasmodic and Paralytic Incontinence.

	Occurrence.	Effect of Effort, Coughing etc.	Age.	Condition of Urine.	Associated Nerve Symptoms.	Effect of Treatment.
Spasmodic.	Occasional and Intermittent.	Nil.	Generally young.	Clear, acid, normal.	None, unless hysteria.	Good.
Paralytic.	Constant.	Forces away Urine.	Any age, but generally old.	May be ammoniacal and purulent.	If central, generally, a similar affection of rectum and paraplegia.	Very often unfavourable.

§ 91. *Retention.*—The accumulation of urine in the bladder, as the result of inability on the part of the patient to expel it—may be due to :—

1. *Mechanical causes,* such as stricture, enlarged prostrate, tumours in the pelvis, traumatic injuries of the urethra.

2. *Spasmodic contraction of the sphincter vesicæ.*—This condition may result from local (direct) irritation, after ligature for piles, etc., or it may be due to central nervous causes, as in some cases of hysteria.

3. *Paralysis of the detrusor muscle.*—Paralysis of the detrusor sometimes follows excessive distension of the bladder, as in those cases in which the urine has been voluntarily long retained; occasionally in these cases, though I suspect seldom if ever in perfectly healthy individuals, the paralysis becomes permanent.

Temporary paralysis of the detrusor also occurs in many cases of acute disease of the spinal cord and brain, in which a severe shock to the nervous system temporarily arrests the function of the detrusor centre.

Paralysis of the detrusor may also result from lesions of the detrusor centre, and from lesions of the centripetal (motor) nerves passing from that centre to the muscle. Myelitis, hæmorrhage, or traumatic injuries of the lumbar cord, pressure on the nerve roots or nerve trunks, are the chief causes

of this condition. In these cases the paralysis is often permanent, and is generally associated, as has been mentioned above, with paralysis of other parts supplied by lumbar and sacral nerves. Feeble action of the detrusor muscle is seen in some cases of general spinal weakness. In these cases the action of the detrusor is partly compensated by excessive action of the abdominal muscles.

4. *Excessive cerebral control.*—Theoretically, retention will occur where the cerebral influence which inhibits the action of the detrusor centre, and strengthens the action of the sphincter centre, is in excess. Possibly this is the cause of the retention which is met with in some cases of hysteria, and in some other cerebral affections.

The differential diagnosis of a case of retention.—The steps in the diagnosis are :

1. Is the retention mechanical, spasmodic, or paralytic ?
2. If spasmodic, is the spasm due to local or general causes ?
3. If paralytic, what is the seat and pathological character of the lesion ?

The points to be relied upon in making a diagnosis are much the same as those in cases of incontinence, viz.: —

1. The local condition of the urethra, bladder, rectum, and pelvic organs. (Catheter, rectal, and pelvic examination.)
2. The associated nerve symptoms, especially the condition of the other parts supplied by lumbar and sacral nerves.
3. The history and mode of commencement of the attack.
4. The general condition, age, etc., of the patient.

When the retention is due to spinal or cerebral causes an anatomical and pathological diagnosis is to be made in the usual manner.

§ 92. THE RECTAL REFLEX.

The mechanism of defæcation is very similar to that of urination. It must, however, be remembered that the fæces are solid or semi-solid ; that they are not discharged into the rectum in the same continuous stream, as it were, as urine is discharged into the bladder ; and that large hard fæcal masses may meet with a mechanical difficulty in passing through the sphincter. Possibly, too, the intensity of the reflex stimulus

conveyed to the spinal cord by fæces of different degrees of consistency may vary.

The reflex centre for the rectum is situated in the lower part of the spinal cord, in close proximity to the urinary centre.

After the very full description of the urinary reflex given above, it is unnecessary to say more regarding the rectal reflex. The reader is referred to that description.

§ 93. THE SEXUAL REFLEX.

Erection and ejaculation are reflex processes, the centre for which is situated in the lumbar portion of the spinal cord.

Erection.—The sexual centre is put into action by:

(*a*) Irritation of the sensory nerves in the glans penis.

(*b*) Cerebral (emotional) influences.—Though the action of the sexual centre can be excited and strengthened by cerebral impressions, neither erection nor ejaculation can be directly brought about by an effect of the will.

As the result of the stimulation of the sexual centre, an impulse is generated which travels along the nerve erigentes and inhibits the local nervous mechanism in the blood-vessels of the corpora cavernosa; vascular dilatation, engorgement, and erection follow. The course of the fibres through the cord is not known.

Ejaculation results from a more powerful and prolonged peripheral irritation, while the activity of the sexual centre is further strengthened by emotional impulses.

Inhibition.—The sexual, like other reflex acts, can be inhibited by an effort of the will, emotional impressions, etc.

Alterations in disease.—Destruction of the reflex centre (lumbar portion of the spinal cord), or of the nerve erigentes, destroys the power of erection and ejaculation, and causes impotence.

General spinal weakness, a condition which sometimes results from sexual excess, is frequently attended by a condition of irritable weakness of the sexual centre; erections are more readily produced by emotional influences than in health, but soon pass off; ejaculation is frequently premature.

§ 94. *Priapism.*—Erection without sexual desire—is usually incomplete. Occasionally the erection is complete; it may then be painful. The condition may persist for several days.

Priapism may result from—

1. Excessive stimulation of the sensory peripheral nerves, going to the sexual centre, as in gonorrhœa.

2. Direct irritation of the sexual centre; this cause is probably very rare.

3. Irritation of the fibres which conduct exciting (emotional) influences from the brain. This is the cause of the great majority of cases of priapism which are due to central disease. The lesion is usually situated in the lower cervical or upper dorsal region of the cord.[1]

4. Theoretically priapism may be due to irritation of the cerebral centres, which generate the emotional influences that excite the sexual centre. Possibly this is the cause of the sexual derangements which occur in some cases of insanity.

§ 95. *Satyriasis.*—In some cases of locomotor ataxia, a condition of satyriasis is met with. It probably results from irritation, in the lumbar region of the cord, of the sensory nerve filaments, which proceed from the sexual organs upwards to the brain.

§ 96. *The reflex functions of individual segments.*

In the following table the segments of the cord, with which the different reflexes are connected, is shown:

Table showing the segments of the cord with which the different reflexes are connected.

Cervical	7	Interscapular.
,,	8	
Dorsal	1	
,,	2	
,,	4	Epigastric.
,,	5	
,,	6	
,,	7	

[1] It is an interesting fact in this connection, that violent palpitation due to irritation of the (spinal) fibres of the sympathetic in the same region, is sometimes seen in young subjects as the result of sexual excess.

Dorsal	8	Abdominal.
,,	9	
,,	10	
,,	11	
,,	12	
Lumbar	1	Cremasteric.
,,	2	
,,	3	Patellar tendon.
,,	4	
,,	5	Gluteal.
Sacral	1	Plantar.
,,	2	
,,	3	Vesical.
,,	4	Rectal.
,,	5	Sexual.

§ 97. THE CONDITION OF THE PUPIL.

Alterations in the condition of the pupil are frequent in diseases of the spinal cord. In some cases the alteration is due to disease in the cilio-spinal region, which corresponds to the two lower cervical and upper three dorsal segments; from this cilio-spinal region sympathetic fibres pass to the radiating muscular fibres of the iris; irritation of the sympathetic fibres produces dilatation, while their sudden destruction is attended by contraction of the iris. In others, and these are probably the majority, the derangement is due to an associated lesion above the cord; in locomotor ataxia, for example, the loss of the pupil reflex to light is a very constant and important symptom, and is probably due to a lesion in the neighbourhood of the corpora quadrigemina. The mechanism of the pupil reflex and its derangements belong rather to cerebral than spinal pathology. I shall not enter into the subject now, further than to say that irritative lesions in the cilio-spinal region of the cord may be attended with dilatation, while destructive lesions may produce contraction of the pupil.

§ 98. CO-ORDINATION AND THE MUSCULAR SENSE.

Locomotor ataxia is the disease *par excellence* in which spinal co-ordination is deranged. Before the days of Duchenne, this affection was included with the various forms of motor

paralysis, under the common term *paraplegia;* we now know that there is no paralysis, but that the difficulty in walking is entirely due to inability to combine (co-ordinate) the muscular movements of the lower extremities. In the great majority of cases, the inco-ordination only involves the lower extremities. In the later stages of the disease, the upper limbs may also be affected.

The inco-ordination results from the fact, that peripheral impressions passing from the skin, muscles, tendons, ligaments, and joints of the lower extremities, are arrested in the postero-external column of the cord: the reflex grouping of muscles is interfered with, and the perceptive and co-ordinating centres are not informed, or are incorrectly informed, of the manner in which the muscular movements are being carried out. The patient endeavours to remedy the defect by the aid of sight.

§ 99. *Mode of testing co-ordination.* — The co-ordinating power is tested by making the patient perform different muscular movements, and by observing the regularity and order with which they are executed. In testing the condition of co-ordination in the lower extremities, the patient should first be made to walk in a straight line. (In hospitals where the floor is uncarpeted, he may be told to follow the line of one of the boards). This test is, as a rule, satisfactorily accomplished, provided that he is allowed to look at his feet and on the ground; the movements are, however, markedly irregular, the legs are thrown out with a jerk, and the heels brought to the ground with a stamp. He should then be made to raise his eyes from the ground, and told to walk across the room; the movements now become extremely unsteady, indeed, in many cases, the patient dare not, or cannot, walk at all.

The power of balancing the body in the erect position is also impaired or lost in cases of locomotor ataxia. In testing the balancing power, the patient should be made to stand upright, with the heels close together. When the inco-ordinating power is seriously deranged, he is unable to maintain this position, even with his eyes open, but requires to separate his feet in order to make his base of support as broad as possible. In slight cases, closing the eyes when the heels are together,

always makes him totter and fall. Inability to maintain the erect position, when the eyes are closed, though always present in locomotor ataxia, is not, as was formerly supposed, pathognomonic of that affection; any lesion which arrests the upward passage of peripheral impressions from the skin, muscles, ligaments, and joints of the lower extremities, will necessarily disturb the power of maintaining the body in the erect position when the eyes are closed.

§ 100. *The muscular sense* is tested. (1.) By observing the power of discriminating different weights. In applying this test in the case of the upper extremity, the test objects must be identical in every respect except their weight, in order to avoid the possibility of the patient getting at the result by the aid of sight or touch. Metallic balls of a uniform size, covered with leather, answer the purpose well. In testing the muscular sense of the lower extremity, the weights must be suspended from the foot in a handkerchief or by some other means.

(2.) By directing the patient, the eyes having been closed, to touch a given spot [1]—say, the tip of the nose—on the surface of his own body, or to perform certain movements when the eyes are closed. In applying this test to the muscles of the lower extremity, the patient should be placed on his back in bed, his eyes should then be bandaged, and he should be directed to place his legs in various positions. He may be told, for example, to raise his foot to a given height, and then to lower it slowly until the heel rests on the opposite great toe.

§ 101. THE CONDITION OF THE SENSORY FUNCTIONS OF THE SKIN.

In testing the condition of the sensory nerve apparatus, the exact character and distribution of any subjective

[1] In health we can touch any given spot on the surface of the body (the eyes being closed) with quickness and certainty; but when the muscular sense is impaired, the patient is no longer kept informed of the manner in which the muscles are contracting, and the movement is more or less imperfectly performed.

alterations of sensibility, such as pain, numbness, heat, etc., must be carefully noted; and the objective sensibility to touch, pain, and temperature, in the different sensitive areas must be investigated. The mode of investigating the sensory functions of the skin is as follows:

§ 102. *Method of testing the tactile sensibility.*—In testing the sense of touch, and also the sense of temperature, the following preliminary precautions must be adopted, if accurate results are desired:

1. The nature of the test must be clearly explained to the patient, whose intelligent co-operation is absolutely necessary.

2. His eyes must be carefully blindfolded, so as to eliminate the possibilty of his getting at the results by aid of vision.

3. His statements must, from time to time, be checked by blank experiments. This precaution is very necessary. Where, for example, the sensibility of the skin is quite normal, the patient frequently answers carelessly and erroneously. Blank experiments aid us in detecting this error. Again, in cases of anæsthesia, patients who are doing their best to answer correctly, often profess to feel contact impressions when no contact has been made. Such erroneous statements can only be detected by means of blank experiments.

The following points are then to be investigated:

1. *The capacity of perceiving contact impressions.*

In order to test this point, the skin is to be gently touched with some light object, such as a hair or feather, and the patient interrogated as to whether the contact is perceived or not.

Any marked departure from the normal rapidity with which contact impressions should be perceived, must also be noted.

2. *The power of localising contact impressions.*—Different parts of the skin are touched, and the patient is told to state the exact point where he feels the contact.

3. *The degree of sensibility in different localities.*—The sensibility of the skin may be measured in the following ways:

(*a*) Objects of different shapes, such as keys, coins, different weights, etc., are placed on the skin, and the patient is asked to describe them as accurately as possible. The observer should

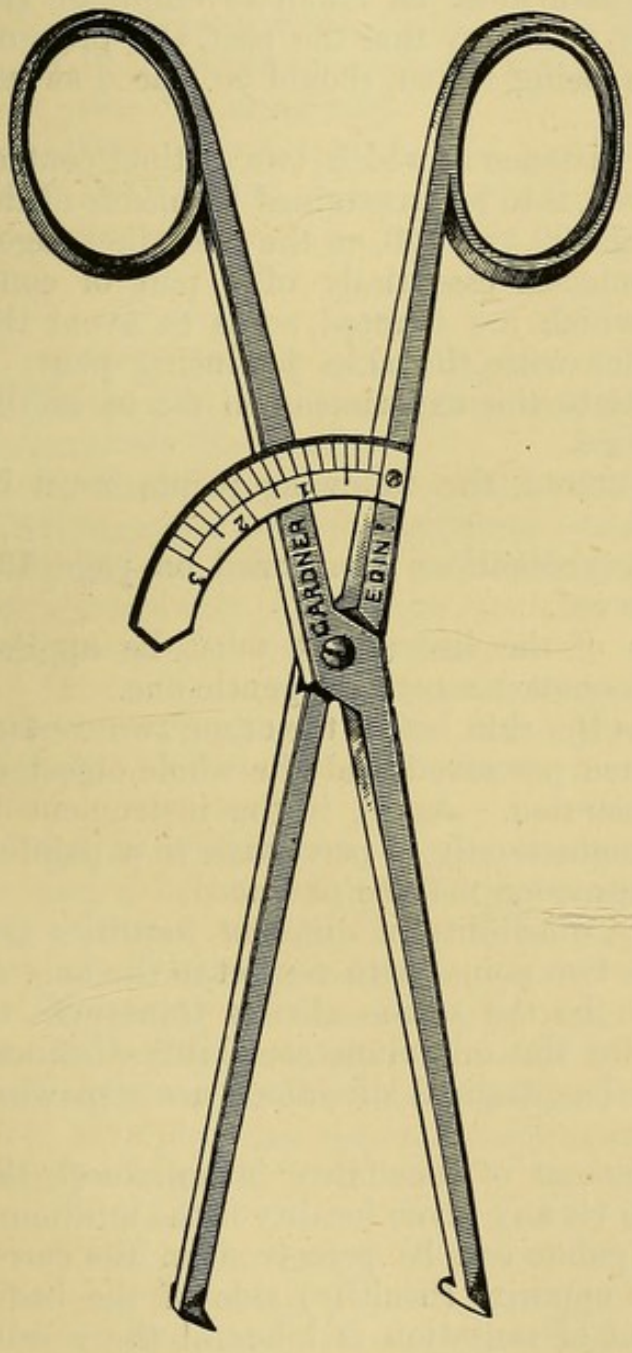


Fig. 99.

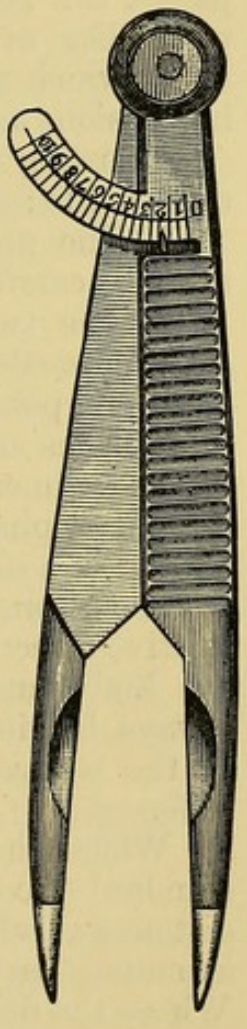


Fig. 100.

Figs. 97 and 98.—*Different Forms of the Æsthesiometer.*

gauge the results by similar contact experiments on his own person.

In estimating the sense of pressure, weights of known capacity may be placed on the skin, as proposed by Weber. In applying this test, care must be taken to eliminate the muscular sense. It is necessary that the part, the pressure sensibility of which is being tested, should be placed at rest and *supported.*

(*b*) The minimum distance at which two distinct contact impressions are perceived, is to be ascertained by means of the Æsthesiometer[1] (see figs. 99 and 100, on the preceding page).

This instrument consists essentially of a pair of compasses, the points of which are blunted, so as to avoid the possibility of a contact with the skin producing pain; a result which would vitiate the experiment so far as tactile impressions are concerned.

In using the instrument, the following points must be attended to:

1. The preliminary precautions mentioned on page 134 must be carefully observed.

2. The two points of the instrument must be applied *simultaneously*, and the contact must be a gentle one.

If one point touches the skin before the other, two contact impressions are of course perceived, and the whole object of the experiment is frustrated. Again, if the instrument is forcibly applied, one contact only is perceived, or a painful, instead of a contact impression may be produced.

3. In comparing the sensibility of different localities, the relative direction of the two points with respect to the axis of the limb must always be the same—always transverse, or always longitudinal—for the minimum sensibility-distances in the transverse and longitudinal directions are somewhat different.

Where the derangement of sensibility is unilateral, the standard of comparison for any given locality is the minimum distance at which two points can be perceived on the corresponding part of the opposite (healthy) side of the body. Where the derangement of sensation is bilateral the results obtained must be compared with those given in the following table:

---

[1] The Æsthesiometer test is a very unsatisfactory one at the bedside.

Tip of tongue,	1.1	m. m.
Palm of last phalanx of finger,	2.2	,,
Palm of second phalanx of finger,	4.4	,,
Tip of nose,	6.6	,,
White parts of lips,	8.8	,,
Back of second phalanx of finger,	11.1	,,
Skin over malar bone,	15.4	,,
Back of hand,	29.8	,,
Forearm,	39.6	,,
Sternum,	44.0	,,
Back,	66.0	,,

§ 103. *Method of testing sensibility to temperature.*— 1. The preliminary precautions mentioned on page 134 must be carefully observed.

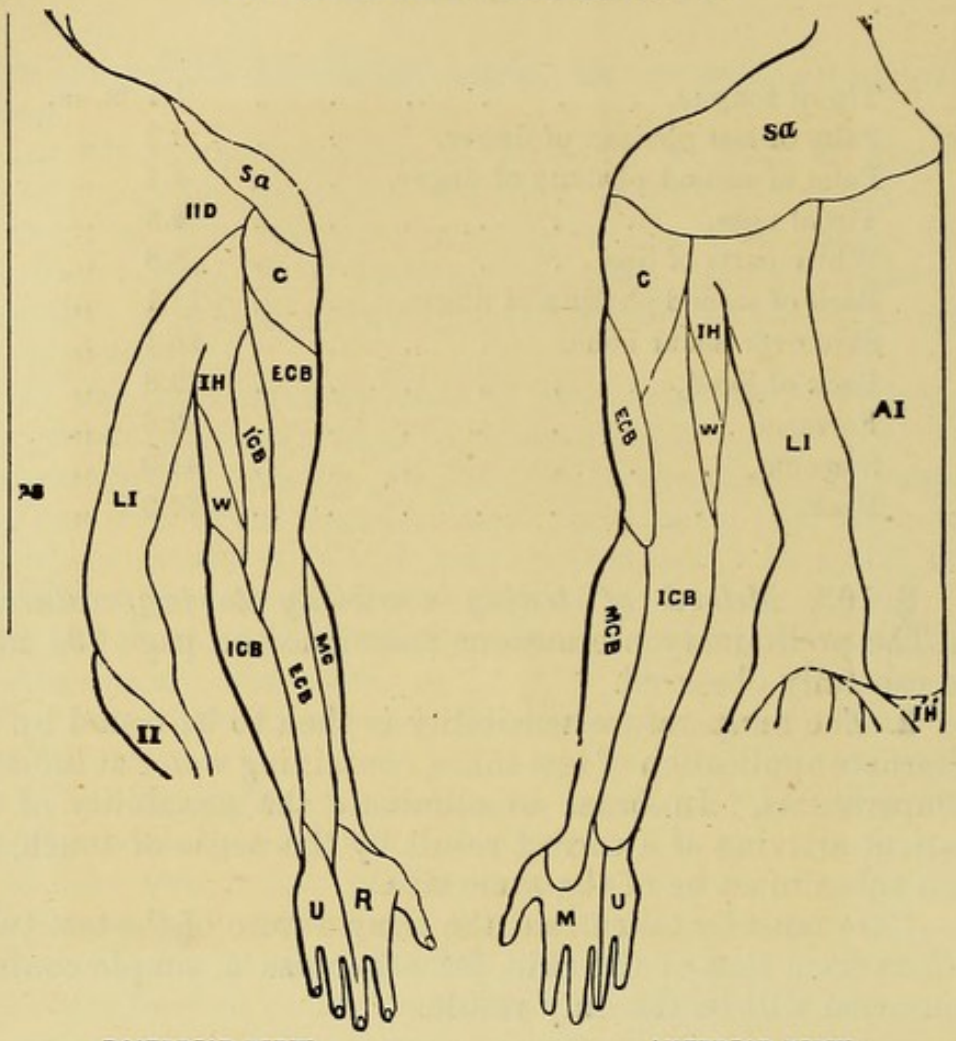
2. The temperature sensibility is then to be tested by the alternate application of test tubes, containing water at different temperatures. In order to eliminate the possibility of the patient arriving at a correct result by the sense of touch, the test tubes must be of the same size.

Care must be taken that the temperature of the test tubes differs from that of the skin, for otherwise a simple contact-sensation will be the only result.

§ 104. *Method of testing the sensibility to pain.*—Sensibility to pain is best tested by pricking or pinching the skin, applying a hot test tube, or a powerful interrupted current to the dry skin with dry electrodes.

In testing the sensibility to pain, the nature of the test should *not be previously explained*, but the patient should be taken unawares; for, if he is prepared for the test, he may be able to inhibit any external manifestation of suffering, and to falsify the result.

§ 105. *The sensory functions of individual segments.*— The exact sensory functions of individual segments have not, as yet, been definitely determined, but by referring to figs. 101, 102, 103, and 104, an approximate idea, at all events, can be obtained of the segments with which the sensory nerves supplying different tracts of skin are connected.



POSTERIOR VIEW. ANTERIOR VIEW.

FIG. 101.

Cutaneous Nerves of the Trunk and Upper Extremity. (*After Flower.*)

Sa, Supra clavicular nerve.
IID, Second dorsal.
PS, Posterior branches of the spinal nerves.
LI, Lateral branches of the intercostal nerves.
AI, Anterior branches of the intercostal nerves.
II, Iliac branch of ilio-inguinal nerve.
I'H', Ilio-hypogastric nerve.
C, Circumflex nerve.
IH, Intercosto-humeral.
W, Nerve of Wrisberg.
I'CB, Internal cutaneous branch of musculo-spiral nerve.
ECB, External cutaneous branch of musculo-spiral nerve.
ICB, Internal cutaneous nerve.
MC, Musculo-cutaneous.
R, Radial nerve.
U, Ulnar nerve.
M, Median nerve.

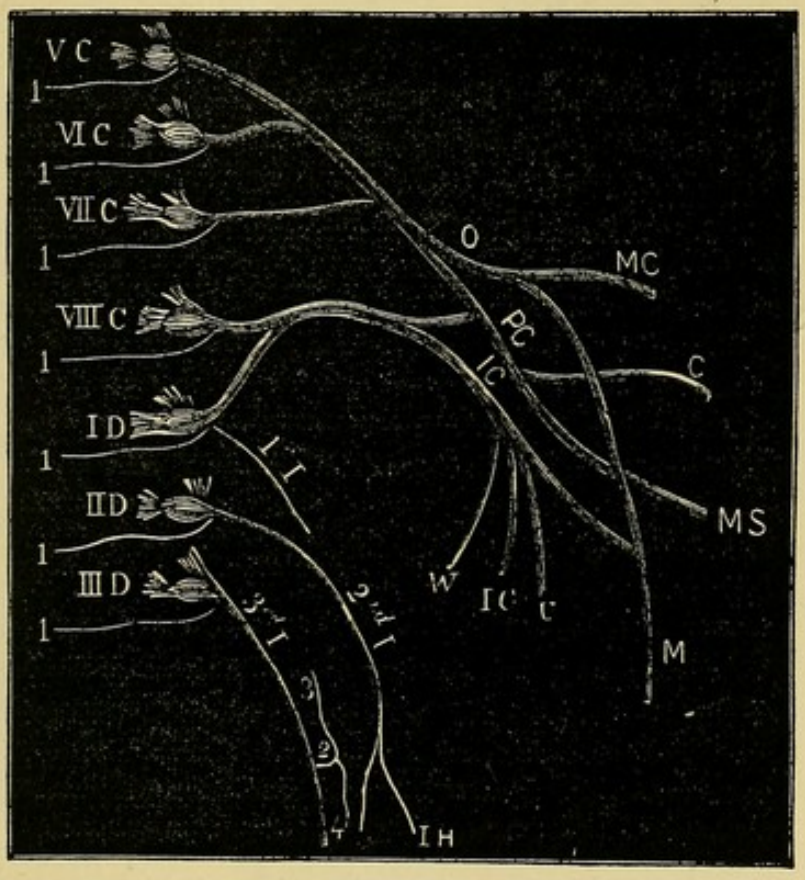


FIG. 102.

Diagram showing the mode of origin of the Sensory Nerves of the Upper Extremity.

V C, VI C, VII C, VIII C, Fifth, sixth, seventh, and eighth cervical nerves

I D, II D, III D, First, second, and third dorsal nerves.

O Outer, PC posterior, and IC inner, cords of the brachial plexus.

MC, Musculo-cutaneous nerve.

C, Circumflex.

MS, Musculo-spiral

M, Median.

U, Ulnar.

IC, Internal cutaneous.

W, Nerve of Wrisberg.

1st I, First; 2nd I, second; and 3rd I, third intercostal nerves.

1. Dorsal cutaneous.
2. Lateral cutaneous.
3. Posterior branch of lateral cutaneous.
4. Anterior branch of lateral cutaneous.

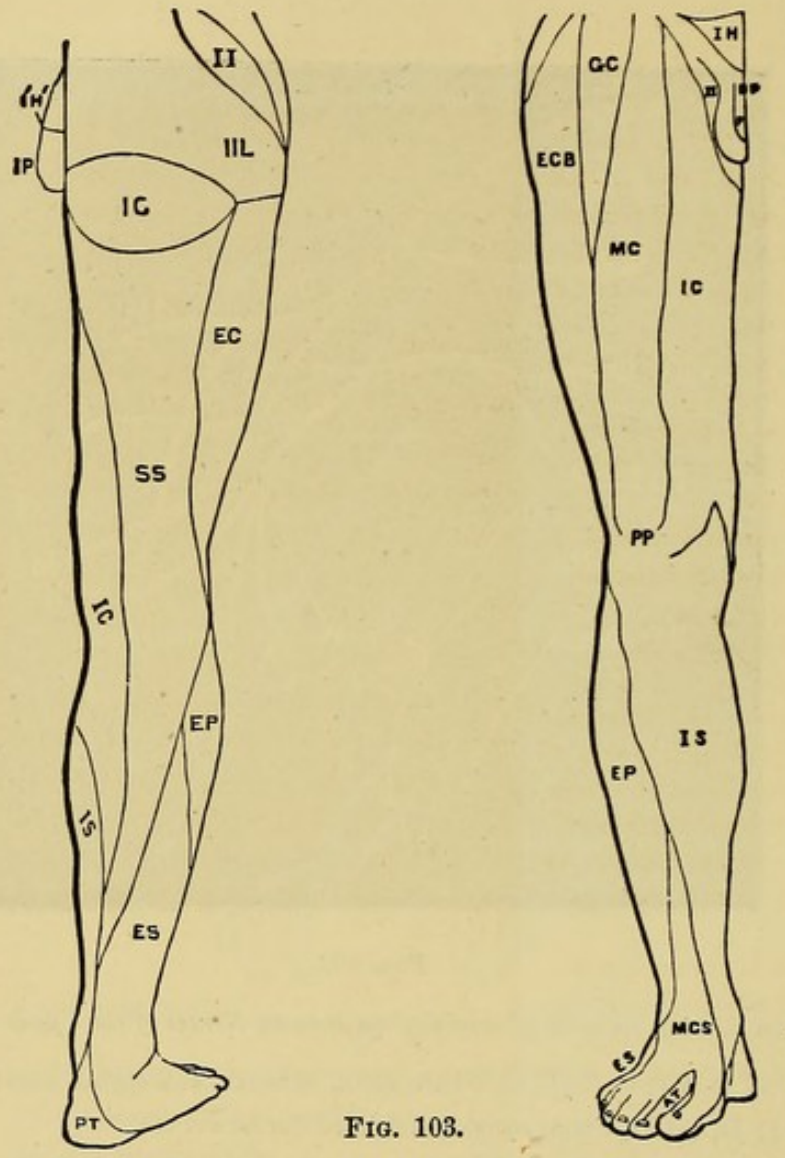


FIG. 103.

POSTERIOR VIEW. ANTERIOR VIEW.

Cutaneous Nerves of the Lower Extremity. (*After Flower.*)

Lumbar Plexus.

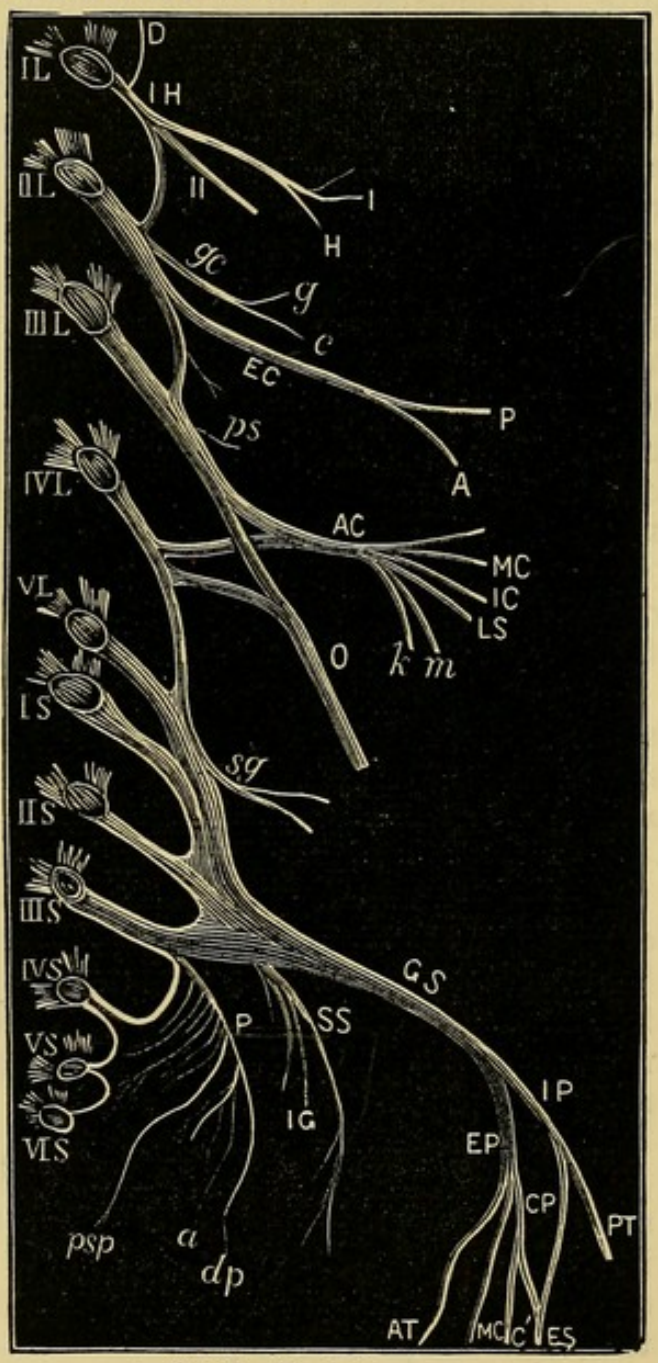
IH, Ilio-hypogastric nerve.
II, Ilio-inguinal.
IIL, Second lumbar nerve.
GC, Genito-crural.
EC, External cutaneous.
MC, Middle cutaneous.
IC, Internal cutaneous.
IS, Internal saphenous.
PP, Plexus patellæ.

Sacral Plexus.

DP, Dorsalis penis of pudic.
IP, Inferior hæmorrhoidal of pudic.
P, Superficial perinæal of pudic and inferior pudental of small sciatic.
IG, Inferior gluteal of small sciatic.
SS, Small sciatic.
EP, Branches from external popliteal.
ES, External saphenous.
MCS, Musculo-cutaneous.
AT, Branches of anterior tibial.
PT, Branch of posterior tibial.

FIG. 104.

Diagram showing the mode of origin of the Cutaneous Nerves of the Lower Limb and Lower Part of the Trunk.



I L, first; II L, second; III L, third; IV L, fourth; and V L, fifth lumbar nerves.

I S, first; II S, second; III S, third; IV S, fourth; V S, fifth; and VI S, sixth sacral nerves.

D, Communicating branch from last dorsal.

I H, Ilio-hypograstric.

I, Iliac branch to skin of gluteal region.

H, Hypogastric branch to skin of hypogastric region.

II, Ilio-inquinal.

Gc, Genito-crural.

g, Genital branch to spermatic cord or round ligament.

c, Crural branch to skin of upper part of front of thigh.

EC, External cutaneous.

P, Posterior branch to skin of upper and outer part of thigh.

A, Anterior branch to skin of front of thigh.

AC, Anterior crural.

MC, Middle cutaneous to front of thigh.

IC, Internal cutaneous to inner part of thigh and leg.

LS, Internal or long saphenous. m, Muscular branch.

k, Branch to knee-joint.

O, Obturator nerve.

Sg, Superior gluteal.

GS, Great sciatic.

IP, Internal popliteal.

PT, Posterior tibial.

EP, External popliteal.

CP, Communicans peronei.

AT, Anterior tibial.

MC, Musculo-cutaneous.

C', Cutaneous to outer side of leg.

ES, External or short saphenous to outer side of foot.

SS, Small sciatic.

IG, Inferior gluteal.

P, Pudic.

dp, Dorsalis penis seu clitoridis.

a, Anterior superficial perineal.

psp, Posterior superficial perineal.

§ 106. *Spinal anæsthesia*, like spinal paralysis, is generally bilateral and symmetrical. The anæsthesia is seldom absolute. In some cases, all forms of cutaneous sensibility are impaired; in others, one only is affected, or one escapes; thus, in many cases, sensibility to pain is lost, while sensibility to touch and sensibility to temperature remain. Occasionally tactile sensations are not perceived, but sensibility to pain and to temperature is retained; much more rarely temperature sensations are lost or diminished, while tactile and painful impressions are preserved. Subjective sensations, such as numbness, tingling, 'pins and needles,' are usually experienced. Where the lesion involves the posterior root-fibres, before they have joined the grey matter, 'lightning' pains are often prominent. In the great majority of cases other signs of spinal-cord disease, such as disturbances of motion, are associated with the anæsthesia. The sensibility of the face is not affected.

In unilateral lesions the anæsthesia is situated on the opposite side to the lesion in the cord, as is represented in fig. 73.

The most frequent clinical causes of spinal anæsthesia are sclerosis of the posterior columns (locomotor ataxia), myelitis, compression of the cord (acute and chronic), and cerebro-spinal sclerosis.

§ 107. *Spinal Hyperæsthesia* may be functional, as in cases of spinal irritation; or organic. The latter form (organic hyperæsthesia) is often associated with anæsthesia. The most frequent clinical causes of organic spinal hyperæsthesia are meningitis, compression of the sensory nerve roots and locomotor ataxia.

§ 108. *Pain referred to the spinal column.*—Pain in the spine is a prominent symptom of spinal meningitis: but it is by no means pathognomonic, for it is met with in many other conditions. It is, however, important to remember that this form of pain (*i.e.*, pain referred to the spinal column) rarely results from organic lesions of the cord itself. The spinal cord, like most other organs—as the liver or the brain—may be affected with severe inflammatory conditions without the slightest pain being experienced by the patient. This statement only holds good, so long as the lesion is confined to the interior of the organ. Should the inflammatory process

extend to the serous coverings, which are richly supplied with sensory nerves, or should it involve the posterior root-fibres before they have entered the grey matter, severe pain will be felt. Now, in many affections of the spinal cord, the membranes are involved; and in such cases pain in the back may be experienced.

It will perhaps be well to mention here the more important conditions which give rise to pain referred to the spinal column. They are as follows:

1. *Inflammatory affections of the skin and subcutaneous cellular tissue over the spine.*—In these cases there is tenderness on pressure, swelling, and other evidence of local disease.

2. *Muscular rheumatism* (lumbago).—The commencement of the attack is acute; the course rapid. Any movement of the affected muscles aggravates the pain, rest and support relieve it; there is tenderness on pressure. There is seldom any affection of the general health; there is no marked pyrexia.

3. *Small-pox, yellow fever, and many other infectious diseases* are ushered in by severe back-ache.—The associated symptoms, which I need not here describe, indicate the nature of the case.

4. *Disease in or about the kidney.*—Inflammation of the peri-nephritic cellular tissue, a renal calculus, and acute congestion of the organ are the chief diseases of the kidney in which pain in the back is experienced. The condition of the urine, the character of the pain, and the physical examination of the lumbar region, are the chief points to which attention must be directed in making a diagnosis.

5. *Disease of the vertebræ.*—This is a prominent cause of pain in the spinal column. The bone disease may be primary, or it may result from the pressure of an aneurism or cancerous tumour. In Pott's disease the general health is, as a rule, profoundly affected. Displacements of the vertebræ often cause curvature of the spinal column. Tenderness on percussion, but especially on movement of the diseased surfaces, is highly characteristic; to avoid jarring of the bones, the patient instinctively keeps the spinal column rigid and still; he will not stoop down to the floor, nor jump on his heels. Symptoms due to associated meningitis, pressure upon the nerve roots or cord itself, may be prominent features of the case.

In aneurism, abdominal cancer, and other affections implicating the spinal column, a careful physical examination can alone decide the exact nature of the case.

6. *Uterine disease, ulcer of the stomach, etc.* In these cases the pain is probably reflex. There is usually no difficulty in making a diagnosis. Assuredly, pain in the spine, due to these causes, is not likely to be confounded with the pain of meningitis or of cord disease.

7. *Neuralgic affections, spinal exhaustion, spinal irritation.* —In all of these cases pain referred to the spine may be a prominent symptom.

In 'spinal irritation' the patient usually is a young woman; marked hyperæsthesia is present; no apparent cause for the pain can be discovered; there is no evidence of organic disease of the cord, membranes, or bones; symptoms of hysteria are often present; the general health is usually below par; anæmia or uterine derangements are common.

8. *Spinal meningitis* both acute and chronic. In this condition, pain in the back is usually well-marked. It is generally aggravated by movements of the spinal column. In the earlier stages, *i.e.*, during the period of irritation, hyperæsthesia of the skin, and sharp shooting pains, radiating in the sensitive areas of affected nerves, are usually present. Symptoms of motor irritation (twitchings, cramps, spasms, contractions, etc.) in the muscles supplied by the affected (compressed) anterior roots are also observed, and are due, either to irritation of the anterior or of the posterior nerve roots. In the latter case the muscular spasms are reflex. Paralytic symptoms (anæsthesia and muscular paralysis) subsequently occur. When the inflammation extends to the spinal cord itself, symptoms of myelitis, or slow compression, are developed.

9. *Tumours within the spinal cord.*—The symptoms are typical of slow compression. The diagnosis of a tumour is sometimes easy, as, for example, where there is associated malignant disease elsewhere (in the liver, pelvis, etc.). In other cases a positive opinion can hardly be given, and the diagnosis can only be arrived at by exclusion:—the absence of the symptoms and signs of Pott's disease; the absence of any apparent cause of meningitis; the very gradual onset and steady advance of the symptoms, etc., etc.

§ 109. THE VASO-MOTOR AND TROPHIC CONDITION OF THE SKIN AND JOINTS.

Irritation of the fibres of the sympathetic in the cilio-spinal region produces pallor, diminution in the temperature and in the secretion of sweat on the same side of the head and neck, together with contraction of the pupil. Destructive lesions in the same region may produce dilatation of the blood-vessels, redness of the skin, increased temperature, increased secretion of sweat, and dilatation of the pupil. Alterations in the vaso-motor condition of the skin of the limbs and trunk are also seen in some cases of cord disease. Lesions of the anterior nerve roots are sometimes attended with œdema of the parts to which they are distributed, in addition to the motor paralysis.

Alterations in the trophic condition of the skin are not uncommon. Sloughing is the most frequent and important. In cases of paraplegia, in which the vitality of the skin is lowered, any external irritation is apt to be followed by destructive inflammation. In cases of paraplegia, therefore, cleanliness must be scrupulously attended to, and the greatest care taken to avoid any undue pressure. Bed-sores occasionally form with great rapidity and in spite of the most careful attention. Professor Charcot, to whom we are indebted for much information on this subject, believes that the acute form of bed-sore, results directly from the spinal lesion, and is due to irritation of the grey matter.

Trophic alterations in the bones and joints occur in a small proportion of cases of locomotor ataxia. The affected joints rapidly become disorganised; spontaneous fractures of the bones occur; the affection is a painless one. Professor Charcot believes that the joint affection is due to extension of the lesion from the postero-external columns to the anterior cornua; but this explanation is disputed by other authorities. Dr Buzzard suggests, that the seat of the lesion is the medulla oblongata.

§ 110. THE CONDITION OF THE SPINAL COLUMN.

The condition of the spinal column must be carefully investigated in all cases of cord disease, by inspection, palpation, and percussion.

Inspection.—Irregularities are seen in most cases of fracture and luxation of the vertebræ. Curvatures and irregularities are also observed in some cases of Pott's disease.

Palpation and percussion.— The spine should be percussed from behind, and the bodies of the vertebræ should be palpated from the front: pain sometimes is elicited, and swelling occasionally perceived, by this method of examination, when the examination of the spinal column from behind reveals no abnormal condition. In cases of Pott's disease, movement of the spinal column almost invariably gives rise to complaints of pain; this is a very valuable diagnostic, but it is not pathognomonic, for in other cases in which there is no bone disease, primary meningitis, for example, the pain is usually increased by movement of the spinal column.

The hot sponge test.—A sponge wrung out of water, not sufficiently hot to produce a sensation of pain in a healthy individual, is drawn slowly over the vertebral spines. A painful sensation is sometimes experienced at the seat of the disease. It was formerly thought to be indicative of myelitis, but is probably only a test of hyperæsthesia. A piece of ice may be substituted for the hot sponge, with the same result.

§ 111. The Condition of the other Parts of the Nervous System.

The mental faculties are usually clear in cases of cord disease, but pontine, medullary, and cerebral complications are not uncommon. *Polio-myelitis anterior acuta,* like all acute affections in the child, may be ushered in by an epileptiform convulsion; and during the febrile stage, mild delirium may occur. *Spinal meningitis* is frequently associated with inflammation of the cerebral meninges; in such cases the cerebral symptoms are usually the most prominent. In *cerebro-spinal sclerosis,* headache, vertigo, alterations in the mental condition, disturbances of speech, and derangements of the special senses, may be marked symptoms; but in some cases the symptoms are for a time chiefly spinal, and a separate variety of the disease (disseminated sclerosis limited to the cord) is accordingly described by some writers.

But in those affections which are usually classed as

distinctly spinal, derangements of the parts supplied by cranial nerves, are also observed. In *locomotor ataxia,* abolition of the pupil-reflex to light, is one of the earliest and most constant symptoms, while temporary paralyses of the muscles of the eyeball, ptosis, and grey atrophy of the optic discs, occur in a large proportion of cases. Lightning pains occasionally radiate in the sensitive area of the fifth nerve. It is particularly important, therefore, to observe the condition of the muscles of the eyeball and of the iris, and the state of the optic discs in supposed cases of locomotor ataxia. The symptoms of *locomotor ataxia* are not unfrequently developed in the course of general paralysis of the insane. The characteristic sense of well-being and other delusions occur in cases of this description; but in ordinary cases of locomotor ataxia, such as one meets with in private practice, the mental faculties are usually quite clear.

In *amyotrophic lateral sclerosis,* and *Landry's paralysis,* and in some cases of *progressive muscular atrophy,* the morbid process has a strong tendency to extend to the medulla oblongata—an event which is followed by bulbar symptoms, and (usually) by speedy death.

§ 112. The Condition of the other Systems and Organs.

Alimentary System.—In the great majority of chronic cord diseases, the appetite is good, the digestive process normal, and the nutrition of the body well preserved.

Acute diseases of the spinal cord are attended with the same derangements of the digestive system (loss of appetite, thirst, constipation, etc.) which occur in other acute affections.

Constipation is a common symptom in cases of paraplegia, and is in many cases due to paralysis of the intestine.

Attacks of vomiting and gastric pain—the so-called *gastric crises*—occur in some cases of locomotor ataxia, and are probably analogous to the lightning pains. These attacks occur more frequently in women than in men, and, as Dr Buzzard has pointed out, are often associated with the peculiar joint affection described by Charcot. Violent attacks of vomiting are also occasionally observed in cases of hysteria.

Meteorismus, depending upon paralysis of the intestine, sometimes occurs. When it appears towards the end of a case of myelitis it is a serious indication.

§ 113. The Circulatory Organs.—As a general rule the heart and great blood-vessels are healthy in cases of spinal cord disease. Aortic regurgitation and aortic disease (atheroma) are occasionally associated with locomotor ataxia. In functional derangements of the cord, palpitation of the heart with or without irregularity, is a frequent symptom; and neurotic disturbances of the heart also result from lesions in the upper cervical region.

§ 114. Respiratory Organs.—Complications on the part of the respiratory organs, such as phthisis, bronchitis, and pneumonia, are frequently the cause of death in cases of cord disease.

Spinal lesions which involve the nerve supply of the diaphragm or intercostal muscles may be attended with severe dyspnœa; a trivial bronchial catarrh may cause serious symptoms, and prove fatal under such conditions.

In progressive muscular atrophy, and still more frequently is amyotrophic lateral sclerosis, serious respiratory disturbances and speedy death may, as I have previously remarked, result from extension of the morbid process to the respiratory centres in the medulla.

§ 115. Urinary System.—In some cases of acute myelitis, involving the lower portion of the cord, the urine rapidly becomes ammoniacal and loaded with phosphates, muco-pus and vibriones. In some cases, the inflammatory affection, which has originated in the bladder, extends to the kidneys, and death quickly follows; Professor Charcot thinks that the cystitis is, in these cases, a trophic disturbance, analogous to the acute bed-sore, and that it is a direct result of the spinal lesion. In other cases—and they constitute the large majority—the decomposition of the urine, and the cystitis are caused by the introduction of septic matters from without. The infective process thus established may subsequently extend to the kidneys. This danger must always be kept in view in the treatment of cases of paraplegia, and the greatest attention paid to the cleansing and disinfection of bougies and catheters.

Excess of phosphates occurs in many cases of cord disease, independently of bladder paralysis; the quantity of urine is often excessive; its reaction acid or neutral; its specific gravity low.

In some cases of hysterical paraplegia large quantities of limpid urine are evacuated; occasionally the urinary secretion is entirely suppressed.

§ 116. The Integumentary System.—The trophic alterations of the skin, which result *directly* from spinal lesions, have been already alluded to. The ordinary forms of skin eruption are, of course, occasionally met with, as accidental complications. Syphilitic eruptions are of great importance, and indicate a specific treatment for the spinal lesion.

THE DIAGNOSIS.

§ 117. *Step No. 1.* Presuming that the symptoms suggest disease of the spinal cord, the first step in the diagnosis is to determine whether the lesion is actually situated in this part of the nervous system.

Typical cases present no difficulty. As the result of experience — clinical examination verified by *post-mortem* examination—we know that, certain symptoms, or rather certain combinations of symptoms, indicate a spinal lesion. But cases are not always typical, and the most experienced may sometimes hesitate before forming a definite opinion.

Now, the only true and scientific method of coming to a correct conclusion is to make a thorough and accurate examination of all parts of the nervous system, and then to draw a logical conclusion from the whole facts of the case. Due weight must be given to individual symptoms; and it is here that experience and a large practical acquaintance with disease give such essential information.

In some cases we have recourse to the '*method of exclusion.*' We endeavour to satisfy ourselves that there is no other lesion, either above or below the cord (*i.e.*, no peripheral or cerebral lesion), capable of producing the symptoms.

Cerebral paraplegia, peripheral paraplegia, spinal hemiplegia, and *spinal monoplegia,* may be instanced as some of the cases in which the difficulties, I have just alluded to, may occur.

§ 118. *Cerebral paraplegia.*—This rare condition may be caused by a lesion in the pons, or medulla. Derangements of the parts supplied by some of the cranial nerves will almost certainly be present, and when present are quite distinctive.

Cerebral paraplegia may also result from two separate and distinct cerebral lesions, as, for example, two independent hæmorrhages in the motor areas of both hemispheres. There is in such cases a history of two distinct attacks of hemiplegia: the lower facial muscles of the side most recently affected, will probably be paralysed: there is usually some disturbance of the mental condition, loss of cerebral control, etc.: the condition of the reflexes corresponds to a cerebral lesion, the superficial reflexes being abolished or diminished, the deep reflexes exaggerated.

§ 119. *Peripheral paraplegia.*—Where a peripheral lesion is so situated as to interfere with the motor nerve supply of corresponding parts on both sides of the body, paraplegia may result, and the case may be mistaken for one of spinal cord disease. A tumour pressing upon the cauda equina, or a cancerous tumour within the pelvis, pressing upon the great nerve trunks which supply the lower limbs, are good examples of such lesions. In cases of peripheral paraplegia severe pain is generally a prominent symptom; indeed, the term '*paraplegia dolorosa*' is almost equivalent to paraplegia, resulting from a peripheral lesion: a tumour pressing upon the cauda equina irritates the sensory fibres of the posterior roots; a tumour within the pelvis produces pain, by irritating the sensory fibres of the sensory-motor nerve trunks. In many cases of peripheral paraplegia the condition termed *anæsthesia dolorosa*, is also observed. Irritative phenomena, cramps, spasms, etc., are sometimes seen in the paralysed muscles. The reflexes may be at first increased, but are usually diminished or abolished. The affected muscles become markedly atrophied. The diagnosis of peripheral paraplegia must be based upon: (1) the nature of the symptoms; and (2) the fact that there is evidence of local (peripheral) disease, such as caries of the vertebræ, a tumour within the pelvis, etc.

§ 120. *Spinal hemiplegia.*—This condition has already been alluded to in speaking of unilateral lesions. It usually results from traumatic injuries or compression of the cord. Occasionally it is produced by a lesion arising within the cord itself, such as myelitis, polio-myelitis anterior acuta, or an intra-medullary tumour.

Spinal hemiplegia is easily distinguished from cerebral hemiplegia, by attention to the following points:—In spinal hemiplegia the face and tongue are not paralysed. There is no derangement of the cerebral functions. Anæsthesia is generally much more marked than in cerebral hemiplegia, and is situated on the opposite side to the motor paralysis. The skin on the paralysed side may be hyperæsthetic. The paralysed muscles may undergo rapid atrophy.[1]

§ 121. *Spinal monoplegia.*—This condition may result from traumatic injury, or compression of the cord, or from poliomyelitis anterior acuta. It is to be distinguished from cerebral and peripheral paralyses affecting one limb only.

§ 122. *Cerebral monoplegia.*—The distinction from cerebral monoplegia is not difficult. In cerebral cases there is no sensory derangement; the paralysed muscles do not undergo rapid atrophy; there are no qualitative electrical alterations. In the later periods, the paralysed muscles, especially the flexors, become rigid. The alteration of the reflexes corresponds to that met with in cerebral lesions. The history and mode of most of the attacks may be distinctive. Other cerebral symptoms are usually present. Localised epileptiform convulsions, headache, vomiting, and optic neuritis, are often observed, for a cerebral monoplegia very generally depends upon a 'coarse' cortical lesion.

§ 123. *Peripheral monoplegia.*—This condition may result from traumatic injury, or compression of the large nerve trunks. The sensory functions are profoundly affected; in the earlier stages there is usually pain and hyperæsthesia, later anæsthesia and analgesia occur. In severe cases the paralysed muscles are markedly atrophied. The 'reaction of degeneration' is present in acute cases. The reflexes are diminished or abolished.

A tumour within the spinal canal may produce a monoplegia. Sensory disturbances, due to pressure on the posterior nerve roots, are usually well marked, and symptoms due to pressure on the cord itself are generally present.

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[1] This of course depends upon the position of the lesion with regard to the nerve nuclei. In cerebral cases rapid atrophy does not occur

§ 124. *Spinal monoplegia the result of polio-myelitis anterior acuta.*—Spinal monoplegia occasionally results from acute destruction of one anterior horn of grey matter, but the diagnosis in these cases presents no difficulties; in the great majority of cases the patient is a child; the onset is acute, and is attended with fever; the affected muscles are flaccid; some of them undergo atrophy, and present the 'reaction of degeneration;' their reflexes are diminished or abolished. There are no marked sensory disturbances; the functions of the bladder and rectum are not interfered with.

§ 125. *Step No 2.*—Supposing that the symptoms indicate disease of the spinal cord, we must next determine whether they are genuine or not.

Symptoms are seldom deliberately manufactured, but patients frequently exaggerate their complaints; sometimes deliberately, with the intention to deceive; in most cases more or less unconsciously.

Deliberate exaggeration or imposture may be *suspected* when there is no evidence of organic disease, and when there is some obvious inducement for deception. But a diagnosis of feigned disease should never be based on negative evidence alone. Such an opinion is only to be given when it can be supported by some positive fact or facts. Positive evidence of imposture may be forthcoming in the shape of anomalous symptoms; such, for example, as the unnatural distribution of the paralysis, or some marked peculiarity in the mode of onset or course of the case; but the facts which lead to a definite opinion of imposture are usually *non*-medical.

§ 126. *Step No. 3.*—Having come to the conclusion that the symptoms are genuine, we must next determine *whether they are caused by functional derangement or by organic disease.*

The decision is, in some cases, easy, in others difficult or impossible. Local muscular atrophy, the 'reaction of degeneration,' true spinal inco-ordination, and paralysis of the sphincters, are proof positive of organic disease; but the great majority of spinal symptoms occur both in organic and functional derangements. In doubtful cases, the differential diagnosis can only be made by taking a comprehensive view

of all the facts; by paying particular regard to the mode of development and grouping of the symptoms, and to the general features of the case. The presence or absence of disease in the other parts of the nervous system must be carefully noted. The condition of the other systems and organs must, of course, be observed.

In some cases it is impossible to give a decided opinion at the first visit. We must then be content to make a provisional diagnosis; to await the course of events; and to throw light upon the case by particular modes of treatment (therapeutic diagnosis).

The functional conditions which are liable to be mistaken for organic disease, are for the most part characterised by derangement of the motor nerve apparatus: cases in which the sensory functions are alone perverted, seldom give rise to difficulty. *Hysterical paraplegia, paralysis depending upon idea, reflex, malarial,* and *alcoholic paraplegia,* are some of the best marked types.

§ 127. *Hysterical Paraplegia.*

Before entering upon the differential diagnosis of hysterical paraplegia, it is necessary to insist upon the fact, that all cases of paraplegia occuring in hysterical patients, are not necessarily functional.

Hysteria is frequently associated with organic disease of the nervous system. It is essential, then, to remember that the presence of hysteria, or a history of hysterical fits, is only corroborative evidence, and that a diagnosis of hysterical paraplegia should not be made unless the most scrupulous examination has failed to detect the signs and symptoms of organic disease.

The condition of the muscles.—There is nothing distinctive in the condition of the paralysed muscles in cases of hysterical paralysis. In some, the loss of motion is incomplete; in others, all motor power is abolished. The paralysed muscles are sometimes flaccid; in other cases they are rigid, the reflexes are exaggerated, the ankle clonus is present, and the condition exactly resembles the spastic paraplegia, which results from organic disease. Where the paralysis is of long duration, the muscles may become wasted, but there is never local atrophy. The 'reaction of degeneration' is never present.

The condition of the muscles at the upper level of the paralysis must be carefully noted; for, in some cases of spastic paraplegia, it enables us to decide whether the paralysis is due to organic causes or functional disease. In cases of spastic paraplegia depending upon a transverse myelitis, the muscles supplied by the affected segment, *i.e.*, the muscles at the upper level of the paralysis, become markedly atrophied—a condition which is never observed in purely hysterical cases.

In primary lateral sclerosis there is no local atrophy at the upper level of the paralysis, but that rare condition could hardly be confounded with spastic paraplegia, the result of functional (hysterical) disease. The differential diganosis of these two conditions will be afterwards considered.

The mode of onset and course.—In some hysterical cases the onset is gradual; in others sudden. The *immediate* development of spastic rigidity would be distinctive of the hysterical nature of the case; for rigidity, the result of organic disease, is never developed suddenly. Sudden improvements and relapses are also characteristic of hysterical and other forms of functional paralysis.[1]

The condition of the sensory functions.—In hysterical cases sensory disturbances are usually present. All forms of skin sensibility may be abolished. In some cases there is complete insensibility to pain, but sensibility to touch and temperature remains. Dr Drummond[2] thinks this condition distinctive of hysteria. In others, again, there is hyperæsthesia. According to Duchenne, paralysis, with abolition of the muscular sensibility, is characteristic of hysteria. This opinion is, however, disputed by some recent writers.[3]

The condition of the bladder and rectum.—The sphincters are never completely paralysed in hysterical paraplegia: retention of urine is common: spasmodic incontinence also occurs. After a 'fit' or emotional disturbance, the evacuation of large quantities of limpid urine, of low specific gravity, not unfrequently occurs.

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[1] In some cases of cerebro-spinal sclerosis sudden variations occur. The rhythmical tremor on voluntary effort, and the other features of the case would probably be sufficiently distinct to prevent an error in diagnosis.

[2] *Transactions of the Northumberland and Durham Medical Society*, February 1881.

[3] *A Treatise on Therapeutics.* Dr Wood. Page 678.

The age, sex, and general appearance of the patient.—Hysterical paraplegia is usually seen in *young women.* The peculiar expression of countenance, which Todd called the *facies hysterica,* may be present.

The condition of the uterus and ovaries.—There is often some disturbance of the uterine or ovarian functions.

The history.—A history of previous attacks of paralysis, which have been rapidly recovered from, is very suggestive of functional (hysterical) disease.

Other symptoms and signs of hysteria are generally present, and are of great diagnostic value *in those cases in which there is no evidence of an organic lesion.*

§ 128. *Paraplegia depending upon Idea.*

Under the term 'Paralysis depending upon Idea,' Dr Russell Reynolds has described certain cases of paralysis, depending upon imagination. In these cases, there is no intention to deceive; the patients really believe that they are the victims of serious organic disease. Cases of this sort are quite distinct from ordinary cases of hysterical paralysis, and from ordinary cases of hypochondriasis. The patients are, for the most part, of a highly nervous, and often very active mental temperament; their general health is usually below par, but they do not, so far as my experience goes, exhibit the usual symptoms of hysteria. The fixed belief that they are paralysed; and the concentrated attention which the affected parts receive, induce functional disturbances, which are often of a striking character; startings, twitchings, and fibrillary tremors occur in the limbs and other parts of the body; aching, muscular (myalgic) pains are common; the heart is easily excited; palpitation is frequent; exertion or mental excitement is followed by a feeling of exhaustion and fatigue. The patient becomes impressed with the idea that he is unable to do anything; that he is paralysed, etc. There is often sleeplessness and restlessness; he wakes up towards the early morning hours, and rises unrefreshed. The stomach may be deranged; constipation is common. On physical examination the reflexes—superficial and deep—are generally found to be exaggerated, the muscles supposed to be paralysed are usually soft and flabby; in chronic cases there may be considerable

atrophy, but it is not limited to special muscles or groups of muscles—a point of importance, for, in some cases which have come under my notice, the diagnosis lay between this condition (ideal paralysis) and progressive muscular atrophy. *Sensibility* is not affected. The *bladder* is healthy: the *bowels*, as already mentioned, are usually constipated.

The loss of motor power is never complete, and often presents anomalous characters: for example, a patient, who can neither stand nor walk, will move the legs in any direction when in bed.

'Ideal paraplegia' often lasts for a long time, it may be years; and it is sometimes most difficult to cure.

The diagnosis of it is to be made by attention to the following points:—

1. *The condition of the paralysed parts.*—There is no positive evidence of organic paralysis. The increased reflex excitability is general, and is not confined to the paralysed parts. The startings, jerkings, and fibrillary twitchings are not limited to special muscles; the muscles in which they occur do not present any special atrophy. There is no affection of the bladder or rectum.

2. *The mental condition and temperament of the patient.*—The fact that the patient's mind is concentrated on his condition; that he is always thinking of his symptoms, examining his limbs, etc.; and that he is thoroughly impressed with the idea that he is the subject of serious organic disease is, in the absence of symptoms and signs of organic disease, a strong point in favour of 'ideal' paraplegia.

3. *The history and progress of the case.*—In chronic cases the fact that there are no marked alterations in the condition of the affected muscles, is another point in favour of 'ideal' paraplegia.

In some cases there is a family history of spinal paralysis. The fact, of course, tells in two ways; for, on the one hand, we know that nerve affections are frequently hereditary; on the other, it must always be remembered that persons of a susceptible nervous temperament, who are brought in contact with disease, are very apt to imagine that they themselves are the subjects of it.

4. *The effects of treatment.*—A favourable opinion, confidently expressed, and appropriate treatment, sometimes effect rapid and remarkable improvement.

§ 129. *Malarial Paraplegia.*

Paraplegia sometimes occurs as the result of malarial infection. The pathology of the condition is unknown. The condition is a rare one, even in malarial districts, and no case has come under my personal observation. The characteristic feature of the paralysis is, that it is intermittent, and that it occurs at regular intervals, just as the ordinary febrile paroxysms (quotinian, tertian, etc.) do.

The diagnosis is to be based upon:

1. The absence of the signs and symptoms of organic disease.
2. The intermittent character of the paralysis.
3. The fact that the patient has been exposed to malaria, and that the paralysis is cured by anti-malarial remedies (quinine).

§ 130. *Reflex Paraplegia.*

Paraplegia occasionally results from peripheral irritation acting reflexly. The source of the irritation is usually in the bladder or urethra. Brown-Sequard supposes that the immediate cause of the paralysis is anæmia of the cord, and that the anæmia is due to spasmodic contraction of the vessels of the cord, produced reflexly.

In many cases of so-called reflex (urinary) paraplegia, there are organic changes in the cord, of an inflammatory character. In some, probably the majority, the inflammatory changes result from direct extension to the cord, of similar conditions in the distant (peripheral) parts. The extension may take place along the nerves (neuritis ascendens), or through the blood-vessels. In other cases in which organic lesions have been found in the cord, the intermediate parts have appeared to be healthy, and it is supposed by Benedict, that organic spinal lesions can actually be induced reflexly, independently of direct extension. Cases of this description, in which there is an organic lesion of the cord, cannot correctly be termed *reflex*, if we understand by that term, a *functional* paralysis due to peripheral irritation acting reflexly.

True reflex paralysis is undoubtedly rare, but that it does

occasionally occur in man seems beyond dispute; and that it can be induced in the lower animals seems proved by the remarkable chloroform experiments which Brown-Sequard has lately published. The diagnosis of reflex paralysis is always hazardous, and should only be made when:

Firstly. There is no evidence of organic disease.

Secondly. There is a manifest source of peripheral irritation.

Thirdly and *chiefly.* The removal of that irritation is followed by the disappearance of the paralysis.

§ 131. *Alcoholic paraplegia.*—A temporary form of paraplegia is sometimes produced by alcoholic excess. The paralysis is probably due to some alteration in the vaso-motor condition of the cord. It is often difficult to give a positive opinion at the first visit. There is no evidence of organic disease, but a commencing subacute myelitis, which is not uncommon in persons given to alcoholic excess, may be attended by exactly the same conditions. These are the cases in which we must await the course of events. Functional alcoholic paraplegia is only temporary: in myelitis the paralysis, of course, lasts for some time.

§ 132. *Anæmic paraplegia.*—Paraplegia may also result from sudden stoppage of the blood supply to the lower end of the cord. This condition may be due to plugging of the abdominal aorta, an accident which occurs in the course of some abdominal aneurisms.

In some cases of aortic regurgitation, weakness in the lower extremities—never, so far as I know, amounting to true paralysis—is seen. The motor weakness probably results from anæmia of the lumbar region of the cord, but this point has already been referred to. (See page 54).

§ 133. *Step No. 4.—The differential diagnosis of extra-medullary and intra-medullary lesions.*—Having come to the conclusion that the symptoms are due to organic disease of the cord, the final step in the diagnosis is to determine the exact nature of the affection which is present. We must not only give a name to the disease, but must endeavour to determine the special features which it presents in each individual case.

We first decide whether the lesion is *extra*-medullary or

intra-medullary. This point is determined by the nature of the symptoms.

The *characteristic* symptoms of an *extra*-medullary lesion result, as we have previously seen (see page 66), from irritation of the sensory nerves in the membranes and periosteum, and from pressure on the anterior and posterior nerve roots. They consist of:—pain referred to the region of the spinal column, and increased on movement of the vertebræ; eccentric pains, hyperæsthesia and anæsthesia in the sensitive areas of the compressed posterior roots; spasms and paralysis in the areas of the compressed anterior roots.

When these symptoms are present, we may conclude that the spinal symptoms are secondary, and that the primary lesion is *extra*-medullary.

§ 134. *The pathological diagnosis of extra-medullary lesions.*—Having determined that the lesion is an *extra*-medullary one, we must next endeavour to ascertain its pathological character. In seeking to decide this question, we must regard—

1. *The history.*—The duration of the case, whether acute or chronic, is of great importance. Where the symptoms develop suddenly, we have to deal with a displacement of the bones, or with a hæmorrhage into the spinal canal. Where the onset is rapid, but not instantaneous, the case is probably acute meningitis, but it may be displacement of the vertebræ. In chronic cases, the symptoms may be due to chronic meningitis, or to slow compression. The supposed cause of the attack should always be enquired into: a history of traumatic injury is especially important.

2. *The condition of the spinal column.*—In fractures and displacements of the vertebræ, either from violence or disease, irregularities in the spinal column are sometimes, but not always present. Pain and tenderness on percussion, and on movement of the vertebræ, are valuable evidence of bone disease. In some cases of vertebral cancer, a tumour or enlargement of the bones can be detected.

3. *The associated diseased conditions* are often distinctive. In a case of slow compression of the cord, the presence of malignant disease in the liver or pelvis would point to similar disease within the spinal canal.

4. *The family history* may also give some information. In a case of meningitis, without obvious cause, a strong family

tendency to scrofula, would suggest the tubercular character of the lesion.

§ 135. *The pathological diagnosis of intra-medullary lesion.* —Where the lesion is *intra*-medullary, we must first decide whether we have to do with a 'system disease,' or an 'indiscriminate' lesion. By this means we at once narrow the enquiry, for as the result of experience, we know that certain morbid conditions affect certain physiological tracts. Where, for example, the symptoms show that the lesion is *confined* to the anterior cornu, we know that we have to deal either, with acute or sub-acute inflammation *(polio-myelitis anterior acuta or sub-acuta)*, or with the chronic degenerative process which constitutes the patholological substratum of *progressive muscular atrophy.*

Having decided that the disease depends upon a system lesion, we must next determine the pathological character of the morbid process. We regard the history and the *mode of onset* of the attack. When, for instance, the lesion is *confined* to the region of the anterior cornu, and when the onset is acute, we at once decide in favour of *polio-myelitis anterior acuta.* When the onset is very gradual, the probability is, that we have to deal with the typical form of progressive muscular atrophy. To be quite certain, we must carefully ascertain the *mode of development of* the symptoms, for there are two forms of this affection; a typical form, which commences in the great majority of cases in the upper extremity and usually first affects the small muscles of the hand; and an irregular form, which first affects the muscles of the trunk, or those of lower extremity. The typical form undoubtedly results from slow destruction of the motor nerve cells of the anterior cornu. The irregular form is supposed by some observers to result from a primary lesion of the muscles.

It is always advisable to *check* the diagnosis, by observing the mode of development of the attack, and the exact character of the symptoms. In locomotor ataxia, for example, the motor inco-ordination is almost invariably preceded by lightning pains. Certain eye symptoms are also very frequent in the early stage of that disease. Inco-ordination occurring *per se*, without lightning pains or eye symptoms, would probably be due to some other morbid process than the extremely

chronic lesion, which constitutes the anatomical substratum of locomotor ataxia.

§ 136. *The differential diagnosis of indiscriminate lesions* is to be determined by regarding :—

1. *The mode of onset.*—*Sudden* paralysis is due to a vascular lesion, such as hæmorrhage, or embolism. *Rapid*, but not instantaneous lesions, may be vascular, but are usually inflammatory. *Chronic* lesions may be due to chronic inflammation, simple softening, or some degenerative process. Under the last head we may include the morbid process, which I have previously described as sclerosis, commencing in the connective tissue (interstitial sclerosis). In rare cases new formations arise within the spinal cord itself; the symptoms usually develop slowly; in some cases acute symptoms, due to hæmorrhage or inflammation around the growth, occur.

2. *The associated diseased conditions*, which sometimes give a clew to the nature of the morbid process; in myelitis, for example, the presence of associated syphilitic symptoms would suggest a 'specific' character for the cord lesion.

3. *The effects of treatment*, which may also afford information as to the nature of the morbid process, especially in syphilitic cases.

4. *The grouping of the symptoms.*—Having *'named'* the disease, we can in many cases tell the nature of the morbid process with which we have to deal. In cerebro-spinal sclerosis, for instance, the clinical picture is usually quite distinct; and we know, as the result of experience, that the anatomical substratum of that affection is an interstitial sclerosis of a very chronic and incurable character.

To sum up, then, the plan of the diagnosis is as follows:—

Step No. 1.—Make certain that the symptoms do not depend upon a cerebral or a peripheral lesion.

Step No. 2.—Where the symptoms indicate disease of the spinal cord, determine whether they are genuine or not.

Step No. 3.—Where the symptoms are genuine, ascertain whether they are due to functional derangement or to organic disease.

Step No. 4.—Having decided that the symptoms are due to organic disease, next determine—

a. Whether the lesion is *extra*-medullary or *intra*-medullary.

b. If *extra*-medullary, the pathological character of the morbid process.

c. If *intra*-medullary, whether the lesion is a 'system' or 'indiscriminate' one; and the pathological character of the morbid process.

§ 137. THE PROGNOSIS.

In a case of spinal cord disease, we must be prepared to express an opinion as to: (*a*) the probable termination of the case; (*b*) its duration; and (*c*) whether it will unfit the patient for his business occupation or not.

Our opinion on these points must be guided by:

1. *The particular form of disease, and the nature of the morbid process.*

As a matter of experience, we know that 'functional' disorders are never fatal; and that, in most cases, they are quickly recovered from. We also know that, some affections, such as infantile paralysis, lead to permanent paralysis, but do not tend to destroy or shorten life; and that other diseases are of long duration, but sooner or later prove fatal. Locomotor ataxia, and progressive muscular atrophy, are good examples of the last group.

Some affections, which are incompatible with an active outdoor occupation, do not necessarily unfit the patient for literary or clerical work. The late distinguished Professor of Anatomy in the Edinburgh University—John Goodsir—was affected, for many years, with locomotor ataxia, and, until very shortly before his death, carried on the onerous duties of the professorship.

The exact pathological character of the morbid process is often of considerable importance from a prognostic point of view. A syphilitic myelitis, for example, is often recovered from; while a tubercular, or cancerous meningitis, is (?) invariably fatal.

2. *The severity of the affection in the special case under observation; the effects which it has already produced; the rapidity with which the morbid process is being developed, and the direction in which it is extending.* All of these points are of great importance, and must be carefully observed in each individual case.

In infantile paralysis, for instance, the severity of the lesion varies immensely in different cases. The prognosis, in that affection, is chiefly determined by observing the extent of the paralysis, and the number of muscles which have undergone 'rapid atrophy,' and which present the 'reaction of degeneration.'

Again, a myelitis which involves the lower end of the cord, and which is attended with paralysis of the sphincters, and with cystitis, is very much more serious than a myelitis in the lower dorsal region. Where the upper cervical region is affected, the prognosis is still worse, for the diaphragm will probably be paralysed, and serious complications on the part of the respiratory organs will then result.

The fact that the disease is developing rapidly, makes the opinion more serious. Where the morbid process is extending towards the medulla, a fatal termination is probably close at hand.

In each case, the individual symptoms must be taken into account. In locomotor ataxia, for example, the early occurrence of optic atrophy unfits the patient for any business occupation, and so makes the prognosis more serious.

3. *The result of treatment.*—This sometimes gives valuable information as to the future course of events. Rapid improvement under anti-syphilitic treatment is an eminently hopeful indication.

4. *The mental temperament of the patient; his circumstances and surroundings.*—These are also points which must be carefully considered.

Some patients will not submit to a long continued course of treatment, and the restraints in sexual and other matters, which are often necessary. Others cannot place themselves in the best possible position for recovery; they cannot guard themselves against cold and exposure; they must, of necessity, submit to business worries, and the anxieties which are entailed by an arduous struggle for existence. Some persons bear these mental anxieties fairly well, but, speaking generally, the presence of any mental trouble or worry makes the prognosis more unfavourable.

5. *The presence of complications.*—This is a very important element in the prognosis. Each case must, of course, be judged in accordance with the special nature of the complication which is present.

To sum up, the opinion as to the future course of the case, is determined by the following points:

1. The particular form of cord disease, and the pathological nature of the morbid process.

2. The severity of the affection in the special case under observation; the effects which it has already produced; the rapidity with which the morbid process is advancing, and the direction in which it is extending.

3. The effects of treatment.

4. The mental temperament, the circumstances and surroundings of the patient.

5. The presence or absence of complications, and their nature.

Such are the general grounds on which the prognosis is to be based. The special circumstances which determine the opinion in the individual affections of the cord, will be afterwards considered. (See chap. iv.)

TREATMENT.

It must be confessed that the treatment of many affections of the cord is eminently unsatisfactory. The treatment of the individual diseases will be afterwards considered. *The general indications for treatment and the mode of carrying them out are* as follows:

§ 138. *1st Indication. Remove the cause.*

It is seldom possible to carry out this indication in practice. In some functional disorders it can be accomplished, as, for instance, in the derangements of the cord which result from sexual excess, from alcohol, from malaria, or from reflex irritation. In some cases of hysterical paraplegia the treatment of the general condition is followed by a cure of the local condition, *i.e.*, the paralysis.

In some cases of displacement of the vertebræ, we can, by attention to the position of the parts, and by the application of rigid supports (Sawyer's jacket, etc.), remove the pressure from the cord, and so relieve many of the nervous symptoms.

Abscesses near the spinal column should always be evacuated, under strict antiseptic precautions. In several cases, I

have seen very remarkable improvement in the nervous symptoms follow the opening of an abscess which was pointing at some little distance from the spinal column. The abscess sac had evidently, in these cases, pressed upon the cord and its membranes.

The removal of extra-medullary tumours is not yet a recognised plan of treatment. This is partly owing to the uncertainty of diagnosis, and partly to the serious nature of the operation. The difficulties in diagnosis are disappearing, and, thanks to antiseptic surgery, we can now undertake operations which were formerly unjustifiable. I would advise an operation in any case in which the symptoms were urgent; in which the diagnosis clearly indicated the presence of a tumour; in which there was no evidence of malignant disease; in which the exact position of the growth could be localised; and in which a vigorous anti-syphilitic treatment had failed to produce beneficial results.

§ 139. *2d Indication.—Endeavour to arrest and allay the morbid process; and aid nature to repair the damage.*

Hæmorrhage.—Where the symptoms indicate hæmorrhage into the substance of the cord, or into the spinal canal, the usual plan of treatment for an internal hæmorrhage is to be adopted. The patient should be placed in the prone position; perfect rest should be enjoined; ice-bags applied over the spinal column; and full doses of ergotine given subcutaneously.

In practice it is seldom possible to carry out this treatment sufficiently early to produce any decided effect upon the hæmorrhage itself. In most cases the damage is already done before we see the case. In some instances it is possible to mitigate the after effects by reducing the subsequent inflammatory changes.

Inflammatory affections.—In *acute* inflammation either of the cord or its membranes, the patient should be placed at perfect rest in the prone position; leeches or cups, and then ice-bags should be applied to the spinal column; ergotine, in full doses, should be given subcutaneously, with the object of producing contraction of the blood-vessels; belladonna is also employed for the same purpose. Where there is much

pyrexia, febrifuge remedies, especially full doses of quinine, should be prescribed.

In *sub-acute* and *chronic* inflammation, rest is to be enforced; counter-irritants (iodine, blisters, or the actual cautery) are to be applied on each side of the vertebral column; ergotine and belladonna given internally. When there is reason to suspect a syphilitic taint, iodide of potassium and mercury must, of course, be given. In tubercular cases the general state of nutrition must be carefully attended to; in chronic cases, cod-liver oil, lactophosphate of lime, and the compound syrup of phosphates may be given.

After the acute stage of the inflammatory process has subsided, when secondary degenerative changes are progressing, and in the very chronic spinal affections, such as locomotor ataxia, we do what we can to arrest the morbid process, and to aid nature to repair the damage.

The general health must be carefully regulated; the diet should be simple and nutritious, the patient should live out of doors as much as possible; a locality should be selected in which the air is pure and dry; extremes of heat and cold are to be avoided. It is particularly important to attend to the proper ventilation of the sleeping and sitting apartments.

Functional excitement of the cord, must, so far as possible, be prevented. Sexual indulgence should generally be prohibited altogether. Reflex irritation is also to be avoided: patients sometimes find this out for themselves; in a case of spastic paraplegia, for example, which was recently under my care, the patient informed me that the rigidity, girdle-sensation, and difficulty in walking, were always much increased when the bladder became distended. When spastic symptoms are prominent, a minimum of exercise is to be allowed. Remedies which are supposed to favour the absorption of thickened tissues, and to aid in the restoration of the nervous elements[1] are to be given. Iodide of potassium, mercury, and the constant (galvanic) current are the most valuable remedies for this purpose. Arsenic is very beneficial in some chronic cases; it seems to act as a nervine tonic, but is, I think, chiefly useful in those cases in which there is general anæmia.

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[1] Nerve tubes can certainly be restored. Nerve cells which have once been completely destroyed, probably remain so for ever.

Remedies, which experience has found to be beneficial, such as nitrate of silver, should also be administered. Counter-irritation, especially the actual cautery, is useful in many of these chronic cases.

Where the affection of the cord is secondary, the primary disease (Pott's disease of the vertebræ, aneurism of the aorta, etc.) must be treated in the usual manner.

The galvanisation of the spinal cord.—The application of the constant (galvanic) current to the spinal cord, seems to favour the absorption of inflammatory products, and to aid in the restoration of the spinal functions.

In applying the constant current to the spine, with the object of obtaining its so-called catalytic (tonic and restorative) effects, the following points must be attended to:[1]

The position of the electrodes.—Both poles should be placed over the spinal column, in such a manner as to include the diseased portion of cord between them. Where a long portion of cord is involved, as in most system diseases, one pole should be placed over the upper cervical region, the other over the lumbar enlargement. When the lesion is circumscribed, as in most indiscriminate lesions, the poles should be approximated: both poles may be applied over the spine behind, or one may be placed on the front wall of the abdomen or on the sternum. When both electrodes are applied to the vertebral column, one pole should be kept stationary, while the other is slowly moved up and down the spinal column, care being taken to keep it in firm contact with the skin. By this means all parts of the cord can be brought under the influence of the current, while variations in the strength of the current, and the resulting pain and muscular spasms (shocks) are avoided.

The strength of current.—The current should be the strongest which can be comfortably borne by the patient. In order to avoid pain and irritation of the skin, the electrodes must be large. Professor Erb recommends electrodes measuring at least four by two inches. The skin must be made thoroughly moist. All sudden variations in the strength of the current, especially interruptions and reversals, are to be avoided. When the electrodes are first applied, the current should be a feeble

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[1] For further details the reader is referred to special works on medical electricity (Ziemssen's Cyclopædia, vol. xiii. p. 178), and to Erb's article on this subject, to which I am indebted for many of the following details.

one; it should then be gradually increased until the required strength is reached.

The direction of the current is not of much importance.

Duration and frequency of application.—The current may be applied every alternate day. Each sitting should last for about five minutes. The treatment, in order to be effectual, must be continued for several weeks, or even months.

§ 140. 3*d Indication. To relieve the symptoms.*

This is a most important indication. In many cases the relief of symptoms constitutes the main part of the treatment, for, as I have previously remarked, the therapeutic measures which we at present possess, are often inadequate to effect a cure.

Pain must be allayed by the usual anodyne remedies. Opium and morphia are of course the chief. In cases of spinal meningitis, these drugs should be combined with remedies which allay spasm. Bromide of potassium alone, or in combination with opium or morphia, is useful in the pains of locomotor ataxia; the constant current also exerts a decided anodyne effect in some cases. Stretching the sciatic nerve has recently been recommended in the treatment of locomotor ataxia, but I have no personal experience of it. The cases which have been reported seem to show that it relieves the lancinating pains, but does not produce any decidedly beneficial effect upon the course of the disease. In some cases, serious, even fatal results have followed the treatment.

Spasm.—Where spasm is a prominent symptom, the patient should be kept at perfect rest in that position which he himself instinctively selects. All movement and external irritation is to be avoided. The bladder must be regularly evacuated. Warm applications to the spine are, as a rule, pleasant to the patient. Bromide of potassium, opium, hydrate of chloral, are the most useful drugs.

Paralysis.—The nutrition of the paralysed muscles must be carefully maintained. Friction, shampooing, and, above all, the electric current, are the means which we employ.

The electrical treatment of paralysed muscles and nerves.—In using electricity for this purpose, the rule is to employ that form of current to which the paralysed muscles most readily respond. When the muscles react to the interrupted

current, it is the best form. When the 'reaction of degeneration' is present, the slowly interrupted galvanic current must be employed. Each muscle should be regularly exercised. One electrode may be placed over the motor point (see page 91, *et seq.*), the other slowly moved up and down over the affected muscle. Three sittings a week are sufficient in most cases.

Where the paralysed muscles are flaccid, strychnine may be given. Iron, arsenic, and cod-liver oil, are also useful in the same class of cases. Strychnine should never be given where there is the slightest tendency to spasm or rigidity; it is absolutely useless, and often hurtful under such circumstances.

Where spastic symptoms are present, ergot, belladonna nitrate of silver, and iodide of potassium, are the most useful drugs. The rigidity itself is best relieved by chloral hydrate, and bromide of potassium.

In cases of partial paralysis, and especially in those cases in which the muscles are flaccid, the patient should be encouraged to make voluntary efforts. Where attempts at voluntary movement cause an increase of spasm or rigidity, rest should be enjoined.

Paralysis of the bladder and rectum.—In cases of retention (paralysis of the detrusor), the urine must be regularly drawn off by the catheter. Obstinate constipation must be relieved by purgatives and enemata. Paralysis of the bladder or intestine is to be treated in exactly the same manner as any other form of paralysis. When the sphincters are paralysed, great care must be taken to keep the patient clean and dry; when he is able to move about, an india-rubber urinal should be worn; when he is confined to bed, Mr Chiene's syphon arrangement[1] may be tried.

In applying electricity to the bladder, one pole may be placed over the pubes, the other on the perineum, just in front of the anus. If this fails to effect a cure, one pole may be introduced into the bladder, a properly insulated catheter electrode being used.

In employing the constant current in this manner, care must be taken to prevent any injurious action on the mucous

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[1] For a description of this apparatus, and the mode of using it, see *The Edinburgh Medical Journal*, December 1880.

membrane of the bladder. The current should be a weak one. It is generally desirable to inject a small quantity of water into the bladder before introducing the catheter electrode, so as to diffuse the current over the interior of the organ.

Priapism seldom calls for special treatment. Camphor and bromide of potassium may be given in severe cases.

Bed-sores must be treated on ordinary surgical principles.

Deformities and contractures. — Deformities may sometimes be removed by the division of rigid tendons. Professor Volkmann has recently proposed to resect the knee-joint in those cases of old standing infantile paralysis, in which the muscles of the leg and thigh are paralysed and atrophied, and in which the muscles of the hip are only slightly affected. The object of the operation is to weld the bones of the lower limb into a firm continuous support on which the patient can walk, in the same manner as a patient walks on a wooden leg after amputation above the knee.

§ 141. *4th Indication. To prevent complications.*

Bed-sores.—It is not possible to prevent the occurrence of the acute bed-sore, which, as we have previously seen, is a direct result of the cord lesion. The ordinary form of bed-sore, which is due to irritation of and pressure on skin, the vitality of which is lowered, can generally be avoided. The main objects are to prevent continuous pressure on any one point, by placing the patient on a water-bed, and to attend to cleanliness. If decomposing urine and fæces are allowed to remain in contact with the skin, gangrenous inflammation will almost certainly result. It must be remembered that, in consequence of the anæsthesia, the patient may be quite unconscious of his condition: it is the duty of the nurse to feel for him in such circumstances: the back and hips should be frequently examined: the skin may be daily bathed with a spirit lotion: if a blush appears, the patient must be so placed that all pressure is removed from the affected part.

Cystitis.—In some cases the urine decomposes, and cystitis results within a few hours after the cord lesion. This form of cystitis is exactly analagous to the acute bed-sore, and cannot be avoided.

To prevent the ordinary form of cystitis, the greatest atten-

tion must be paid to the condition of catheters and bougies. No instrument should be introduced into the bladder unless it has been thoroughly disinfected.

Pulmonary complications.—Bronchitis, pneumonia, and other pulmonary complications are very apt to arise towards the end of chronic cases. To avoid these accidents, the patient must be guarded against exposure to wet and cold, and other depressing influences.

Contractures and deformities.—The position of paralysed limbs must be carefully attended to: passive movements should be regularly made: by these means contractures and deformities can often be prevented.

Mechanical appliances are sometimes required. In some cases it is necessary to have recourse to tenotomy, but this is rather a remedial than a preventative measure.

§ 142. *5th Indication. To treat complications.*

Phthisis, kidney disease, and *other general complications,* must be treated on ordinary medical principles. The treatment of those complications which result from the cord lesion, has been already alluded to.

§ 143. *6th Indication. To prevent the recurrence of the disease.*

Organic affections, which are recovered from, seldom recur: functional affections often do so. Provided that we know the cause of the attack, and can get the patient to submit to proper preventative measures, we can often, in functional affections, ward off a second attack.

To sum up, the indications for treatment are as follows:—

1. Remove the cause.
2. Endeavour to arrest and allay the morbid process, and aid nature to repair the damage.
3. Relieve the symptoms.
4. Prevent complications.
5. Treat complications.
6. Prevent the recurrence of the attack.

CHAPTER IV.

TABULAR CLASSIFICATION OF THE DISEASES OF THE SPINAL CORD—A SHORT DESCRIPTION OF THE INDIVIDUAL ORGANIC AFFECTIONS.

We are now in a position to take up the individual diseases of the spinal cord, and my treatment of this part of the subject will be brief. I shall, so far as possible, content myself with a concise statement of facts, avoiding repetition by refering to the description, which has been given in the second chapter, of the morbid histology of the different lesions, and of the *rationale* of the different symptoms.[1]

I shall, too, limit my description to the *organic* diseases of the spinal cord, for the chief functional affections have been already somewhat fully considered in the section on diagnosis. (See page 152, *et seq.*)

On the following page a tabular classification of the diseases of the spinal cord will be found.

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[1] The sections on the special pathology of the individual diseases would have been more appropriately included in this chapter than in chapter II.; but it was my original intention to publish the first three chapters only, and it was not until they had passed through the press that I determined to add a description of the individual diseases. Hence the somewhat anomalous arrangement, which will be rectified, should a second edition of the work be called for.

TABULAR CLASSIFICATION OF THE DISEASES OF THE SPINAL CORD.

- I. Functional Affections.
  - (a) Spinal irritation
  - (b) Hysterical paraplegia
  - (c) Paraplegia depending upon idea
  - (d) Reflex paraplegia
  - (e) Alcoholic paraplegia
  - (f) Anæmic paraplegia
  - (g) Malarial paraplegia
- II. Organic Diseases.
  - Intra-medullary
    - System Diseases
      - Primary
        - Of the Anterior cornu
          - Acute, . . *Polio-myelitis anterior acuta:* varieties—Infantile, Adult.
          - Sub-Acute, *Paralysie générale spinale antérieure subaiguë.*
          - Chronic, . (*a*) *Progressive muscular atrophy.* (*b*) *Pseudo-hypertrophic paralysis.*
        - Of the Lateral column (*a*) *Primary lateral sclerosis.* (*b*) *Amyotrophic lateral sclerosis.*
        - Of the Postero-external column . . . . . *Locomotor ataxia.*
      - Secondary
        - Of the Anterior Cornu
          - (*a*) Extension of the lesion, in cases of locomotor ataxia, from the postero-external column to the anterior cornu.
          - (*b*) Extension of the lesion from the lateral column in cases of amyotrophic lateral sclerosis, primary lateral sclerosis, and secondary degeneration of the pyramidal tract.
        - Of the Anterior column — Secondary descending degeneration of, after a brain or cord lesion.
        - Of the Lateral column — Secondary descending degeneration of, after a brain or cord lesion.
        - Of the Postero-internal column . . . . . — Secondary ascending degeneration of, after a lesion of the postero-external column, posterior roots, or posterior root-ganglia.
    - Indiscriminate Lesions
      - Acute
        - (*a*) *Intra-medullary hæmorrhage.*
        - (*b*) *Acute myelitis* (varieties; diffuse, transverse, focal, disseminated, etc.)
      - Chronic
        - (*a*) *Simple softening.*
        - (*b*) *Chronic myelitis.*
        - (*c*) *Cerebro-spinal sclerosis.*
        - (*d*) *Intra-medullary tumours..*
  - Extra-medullary
    - (*a*) Traumatic injuries, wounds, etc.
    - (*b*) Hæmorrhage into the spinal canal.
    - (*c*) Inflammation of the membranes — Acute. Sub-Acute. Chronic.
    - (*d*) Disease of the bones causing rapid or slow compression of the cord.
    - (*e*) Extra-medullary tumours, arising from the bones, membranes, or nerve roots.

TABULAR CLASSIFICATION OF THE DISEASES OF THE ANTERIOR CORNU.

- A. System affections.
  - I. Primary.
    1. Acute inflammation (Polio-myelitis anterior acuta.) — (*a*) Infantile. (*b*) Adult.
    2. Sub-acute inflammation.
    3. Progressive muscular atrophy.
    4. Pseudo-hypertrophic paralysis.
  - II. Secondary.
    1. Extension of the lesion from the lateral column, as in amyotrophic lateral sclerosis, primary lateral sclerosis, and descending degenerations of the crossed pyramidal tract.
    2. Extension of the lesion from the postero-external column in locomotor ataxia.
- B. Indiscriminate lesions.
  - The anterior cornu may, like any other part of the transverse section, be affected by indiscriminate lesions. Other parts of the transverse section are usually involved; the symptoms are then compound, being partly due to the lesion of the anterior cornu, partly to the lesion of the other parts which happen to be affected.

POLIO-MYELITIS ANTERIOR ACUTA.

Acute inflammation of the anterior cornu of grey matter (πολιὸς, grey, and μυελος, marrow). *Varieties* (*a*) infantile; (*b*) adult.

INFANTILE VARIETY.

Synonyms. — Infantile paralysis: Essential paralysis of children: Acute atrophic spinal paralysis of children.

§ 144. Pathology and Morbid Anatomy.—The morbid process, as the term *polio-myelitis anterior acuta* indicates, is an acute inflammation, which is sharply defined and practically limited to the region of the anterior cornu. The segments, composing the lumbar and cervical enlargements, are the parts of the cord in which the lesion is usually situated; in most cases the morbid process is confined to the lumbar region only, but it may involve one or both anterior cornua in one or both of these situations.

The function of the multipolar nerve cells (the essential constituents of the anterior cornual region) is *quickly* interrupted; some of the nerve cells are absolutely destroyed, the muscles, which they supply, undergo rapid atrophy, and are permanently paralysed; other nerve cells are for a time disabled, but ultimately recover; the muscles which they supply are temporarily paralysed.

The histological characters of the cord lesions have already been described in detail; and to that description the reader is referred (see pages 43, 44).

The morbid histology of the paralysed muscles.—The muscular fibres, which are most seriously affected, lose their transverse striæ, and are infiltrated with oil globules; their nuclei are more numerous than in health; the interstitial fibrous tissue is increased in amount; other muscular fibres are in a condition of simple atrophy; others again are normal.

In advanced cases the interstitial fibrous tissue becomes more dense and cicatricial, and the muscular fibres are reduced to thin transparent bands, dotted here and there with nuclei. In exceptional cases the interstitial fat is in excess.

§ 145. Etiology and General Considerations.—The disease is most common between the ages of one and four years. The majority of cases occur during warm weather (summer and autumn), but the exact cause or causes of the affection are obscure; in some cases it seems to result from a chill during dentition; it occasionally follows an attack of scarlatina, measles, or some other febrile affection; in a few cases it is apparently due to traumatic injury, but this is quite exceptional. Nurses are sometimes most unjustly blamed because the paralysis is supposed to be due to a fall.

§ 146. Mode of Onset, Symptoms, and Course.—In some cases *premonitory symptoms*, consisting of general malaise, irritability of temper, slight febrile disturbance, muscular twitchings and tremors, etc., are observed, but in most cases the onset is abrupt.

The affection being an acute inflammation, is usually ushered in by more or less *febrile disturbance.* In some cases, the elevation of temperature is rapid and considerable; and the *onset* is then attended with those symptoms which are associated with rapid elevation of temperature in the child, viz., heat of skin, quick pulse, flushing of the face, muscular twitchings and tremors, epileptiform convulsions, mild delirium or (exceptionally) coma. In other cases the pyrexia is slight, and the general symptoms comparatively trifling. The febrile process is usually of short duration, lasting from a few hours to a few days.

After the pyrexia has continued for a short time, the *characteristic paralytic symptoms* are developed. Occasionally the paralysis occurs during the night, and the febrile disturbance may be so slight as to escape notice; in other cases the febrile symptoms absorb attention, and the paralysis is not noticed until the pyrexia has subsided. The *essential feature of the paralysis* is that the highest degree of motor impairment is at once reached, any subsequent changes being in the direction of improvement.

The *distribution of the paralysis* varies in different cases, and depends upon the exact extent and situation of the lesion. In many cases the distribution is paraplegic (some muscles of both lower extremities being affected), in others, all four limbs are involved; occasionally the distribution is monoplegic, and still more rarely hemiplegic.

All the muscles of a limb are very rarely affected; some muscles or parts of muscles usually escape; and it is important to remember that the paralysis attacks muscles and parts of muscles which are functionally related. This is exactly what we should expect on the supposition that the motor nerve cells of any given segment represent a functional grouping of muscles; in other words, destruction of any given anterior cornu will not produce paralysis of one muscle, but paralysis of several muscles or parts of muscles which functionate in concert. Professor

Remak [1] has described several types; in the upper extremity, for example, an '*upper arm type*,' in which the supinator longus, a muscle of the forearm, is affected along with the brachialis anticus, biceps and deltoid (muscles of the upper arm); and a '*forearm type*,' in which the supinator longus escapes, but in which the extensors of the wrist (muscles of the forearm) are affected.

The condition of the paralysed muscles.—The paralysed muscles are flaccid; some of them (those connected with nerve cells which are seriously injured) undergo rapid atrophy, and present the 'reaction of degeneration.' The reflexes are much impaired or entirely abolished, as shown in fig. 105. Fibrillary twitchings are usually seen in the paralysed parts.

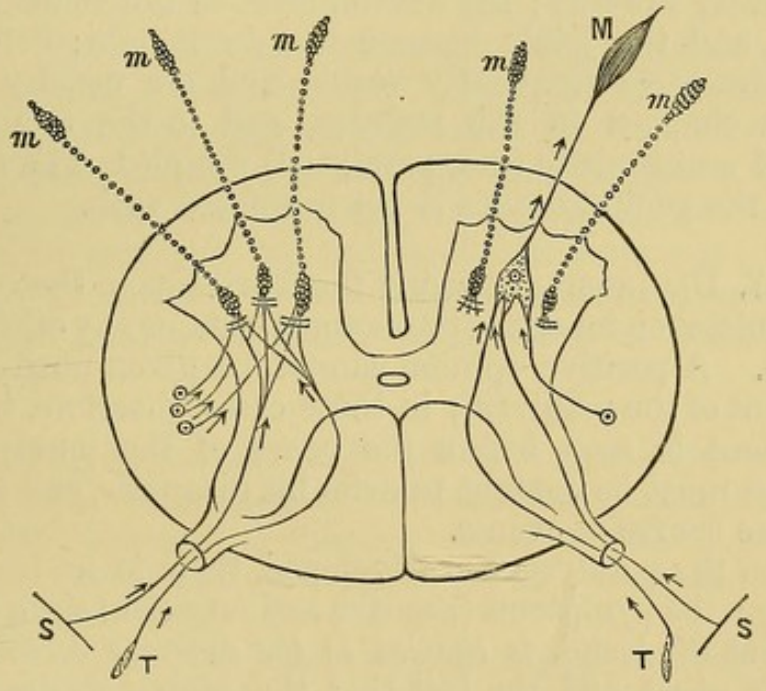


FIG. 105.

Diagrammatic representation of the symptoms which result from acute destruction of the anterior cornua of the spinal cord.

On the left side the destruction of the nerve cells is complete. The anterior nerve roots, motor nerve fibres, and muscles, which they supply, are all degenerated. There is a total 'block' to the passage of voluntary motor and reflex motor impulses. On the right side two-thirds of the motor cells are destroyed; two-thirds of the muscular area connected with the right anterior cornu are degenerated and atrophied; one-third (M) remains healthy, and can be made to contract by voluntary or reflex motor impulses.

[1] *Archives für Psychiatrie*, 1879.

There are no marked sensory disturbances; the functions of the bladder and rectum may for a few hours or days be temporarily arrested, but are never permanently interfered with; trophic disturbances in the skin do not occur.[1]

Exceptional cases.—Very exceptionally, as Dr Buzzard[2] has pointed out, muscles supplied by cranial nerves such as the facial, are affected.

Progress of the case.—A certain amount of improvement almost invariably occurs, but it is quite exceptional to meet with complete recovery; some muscles usually remain permanently atrophied and paralysed, and in severe cases a permanent paraplegia or monoplegia remains.

The temperature of the paralysed and atrophied limbs is permanently lowered; the development of the bones is often arrested, and the joints become unduly mobile; deformities and contractures frequently result, and are usually due to cirrhotic changes in the muscles, and to the unrestrained action of antagonistic (non-paralysed) muscles. The development of the genital organs is not interfered with.

§ 147. DIAGNOSIS.—During the febrile stage the condition may be mistaken for one of the exanthemata, or any other febrile affection. A positive opinion cannot be given until the development of the paralysis; in those cases, therefore, in which the patient is seen before the onset of the paralysis, the physician must be content to defer his diagnosis, and to watch the future course of events.

When the paralysis has developed, there is no longer any difficulty; the symptoms (positive and negative) show without doubt that the lesion *is limited to the anterior cornu;* while the *acute* onset, and the fact that the *highest degree of paralysis is at once reached,* distinguish the condition from all other affections of the same region.

It would be impossible to distinguish the 'system disease,' poliomyelitis anterior acuta, from an acute 'indiscriminate' myelitis, which happened to be *limited* to the anterior cornu. Practically such a limitation does not occur. It would, too, be impossible to distinguish the

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[1] For the explanation of the symptoms, positive and negative, see page 40.

[2] *Medical Press and Circular*, February 23, 1881.

disease from an *acute* destructive lesion *confined* to the anterior roots, but as a matter of fact, this difficulty does not occur; for it would be hardly possible for an acute extra-medullary lesion, such as meningitis, to seriously injure the anterior, without at the same time implicating the posterior roots, and without producing marked sensory disturbances.

When the patient comes under observation during the atrophic stage, it may be impossible from the mere condition of the paralysed parts to give a positive opinion—for the same condition of atrophy might result from *subacute or chronic destruction of the anterior cornu;* but the history of the case (the *acute* onset, and the *immediate* occurrence of paralysis) has only to be ascertained in order to settle the point.[1]

In some cases of *rickets* and *other wasting diseases in children,* the lower limbs are very much emaciated, and the condition at first sight may be taken for 'infantile paralysis;' but there ought to be no difficulty in coming to a correct conclusion, for the muscular atrophy is general, not local; there is no true paralysis, but only muscular weakness; the history usually gives decisive information, and the associated symptoms of rickets or some other cause of emaciation are present.

§ 148. Prognosis.—The disease very seldom proves fatal. When the acute stage is recovered from, as it is in the vast majority of cases, the affection does not tend to shorten life; sexual development is not interfered with; the disease does not tend to recur.[2]

A fatal result during the acute stage is to be dreaded when the febrile process is very severe, and does not quickly subside, and when symptoms, pointing to complications on the part of the cerebral nerve centres or other important organs, are present.

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[1] The differential diagnosis of acute and subacute inflammation of the anterior cornua is afterwards considered in more detail. (See page 185.)

[2] Cases have been reported in which there have been two, or even three exacerbations or separate attacks at the commencement, but these cases are quite exceptional. The statement in the text refers to the risk of future and entirely fresh attacks.

The prognosis as to the subsequent course of the paralysis, is to be guided by the extent and severity of the lesion, especially by the extent of rapid atrophy, and by the number of muscles which present the 'reaction of degeneration;' muscles which continue to react to the interrupted (Faradic) current will certainly recover; while those which do not respond to the electric current, after two months' treatment, will in all probability be permanently atrophied.

When the case comes under observation during the atrophic stage, and when some of the paralysed muscles still respond to the electric current, a certain amount of improvement may be confidently predicted. The amount of improvement will depend upon the number of muscular fibres which respond to electricity, especially to the interrupted (Faradic) current.

§ 149. TREATMENT.—When the patient is seen before the development of the paralysis, the indications for treatment are, to reduce the fever, and to relieve any urgent symptoms which may be present. A calomel purge should be administered, and a febrifuge mixture prescribed. When the attack is ushered in by a convulsion, and when the patient is seen in the fit, cold water may be applied to the head, and the child placed in a blanket-bath. If the convulsions recur, bromide of potassium or chloral may be combined with the febrifuge.

After the paralysis has developed, and the true nature of the disease is apparent, we must endeavour to arrest and allay the inflammatory process. Ergotine should be given in full doses, with the object of producing contraction of the blood-vessels of the affected part;[1] belladonna[2] has also been strongly recommended by Professor Brown-Sequard for the same purpose. Leeches or dry cups may be applied over the

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[1] Dr Althaus gives one-fourth of a grain for a child from one to two years of age; one-third of a grain for a child of from two to five years; half a grain for a child of from five to ten years; and one grain for patients above ten years of age. The drug may be administered subcutaneously, or by the mouth. It should be repeated twice daily.

[2] From one-twentieth to one-sixth of a grain of the extract of Belladonna may be given to a child, but the effects must be carefully watched.

region of the spine corresponding to the affected part. Provided that the patient is sufficiently old and docile, he should be placed in the prone position; and cold, in the form of Chapman's ice-bags, applied to the spine. The febrifuge mixture should be continued; where the fever is high, quinine or the salicylate of soda may be given. The diet should consist entirely of milk; the room must be well ventilated and not too hot.

After the fever has subsided, iodide of potassium may be combined with ergot. Some authorities recommend the inunction of mercury at this stage, but I doubt its utility. The galvanic treatment of the case should next be commenced: a current of medium strength should be passed through the cord at the seat of the lesion, in the manner already described (see 167); and the nutrition of the paralysed muscles maintained by means of friction and electricity; muscles which respond to the interrupted current should be faradized; others should be stimulated by the constant current.

At the end of a month or six weeks the iodide of potassium may be discontinued, and the syrup of iodide of iron, Parrish's compound syrup of phosphates, or arsenic, together with cod-liver oil, substituted. Dr Hammond[1] and some other authorities speak highly of strychnine, but many competent observers have not seen much benefit from its use. It is essential to maintain the general health in the highest state of efficiency. The diet must be light and nutritious. The patient must not be exposed to extremes of

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[1] The following is Dr Hammond's mode of prescribing it:—

℞ Strychniæ sulphatis . . . gr. i.
Ferri pyrophosph . . . ʒ ss.
Acidi phosphorici dil . . . ℥ ss.
Syr. Zingiberis . . . . ℥ iiss.
♏ ft. mist.

Dose, a teaspoonful or less, according to the age of the child.

A child of from three to five years of age can take half a teaspoonful of this mixture thrice daily; or, the strychnia may be given advantageously in the form of hypodermic injections in doses suitable to the age. In children under one year old, the ninety-sixth of a grain is as much as should be given at a dose, and under six months it should not be administered at all.'—*Diseases of the Nervous System*, p. 481.

heat or cold, but, provided that the weather is favourable, he should have plenty of carriage exercise. Sponging the back and limbs with tepid salt (sea) water is highly beneficial in some cases. Great care must be taken to prevent the paralysed parts becoming fixed in a vicious position. Passive flexion should be regularly practised; and in some cases it is necessary to overcome the tendency to contractures by mechanical supports.

This treatment should be continued for several months, until in fact the maximum of improvement has been obtained. Where deformities are already established, section of the tendons may be required. Professor Volkman has recently, as I have previously mentioned (see page 170), recommended resection of the knee-joint in those cases of paraplegia in which the muscles of the hip are not paralysed, and in which the patient is unable to walk for want of a firm support.

ADULT VARIETY.

§ 150. Polio-myelitis anterior acuta is a very rare condition in the adult. Its general features are the same as those which I have described as characteristic of the infantile variety, but there are some slight differences: muscles supplied by cerebral nerves are more frequently affected in the adult than in the child; headache is often observed at the commencement of the attack; in a case which came under my own observation recently, there was temporary aphasia; aching pains, occasionally associated with tenderness on pressure, may be experienced in the paralysed muscles; contractures and deformities seldom result in the adult in whom the bones and joints are fully formed.

SUBACUTE INFLAMMATION OF THE ANTERIOR CORNUAL REGION.

Synonym.—Paralysie générale spinale antérieure subaiguë (Duchenne).

This is an extremely rare affection, which, so far as is at present known, occurs exclusively in adults between thirty and fifty years of age. One case only has come under my personal observation. The following brief account of the disease is mainly taken from the works of Duchenne[1] and Erb.[2]

§ 151. PATHOLOGY.—In the few cases that have as yet been examined *post-mortem*, evidence of chronic inflammation in the region of the anterior cornua was found. The multipolar nerve cells were more or less extensively destroyed; the vessels dilated; the connective tissue thickened; the nuclei and Deiters' cells more prominent and abundant than in health.

§ 152. ETIOLOGY.—Nothing definite is known as to the causes of the condition. Erb throws out the hint that some of the cases may be due to lead poisoning, and, in support of this opinion, quotes the undoubted clinical fact, that paralytic symptoms exactly resembling those which occur in this affection do sometimes result from plumbism.

§ 153. MODE OF ONSET, SYMPTOMS, AND COURSE.—As a rule, the onset is gradual. In some cases *premonitory symptoms*, consisting of aching pains in the back and limbs, a feeling of excessive weariness and weakness, or slight febrile symptoms are observed.

The first characteristic symptom is motor weakness, which gradually increases, and ultimately becomes complete. In the large majority of cases the lower limbs are first affected (the *ascending type* of Duchenne); the muscles of the leg are first invaded, then those of the thigh, and finally those of the hip.

The paralysis presents all the characteristic features which result from a lesion of the anterior horn; the muscles rapidly become atrophied, and present the 'reaction of degeneration; the reflexes are at first diminished, and soon completely

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[1] *L'Electrisation localisée*, 1872, p. 458.

[2] *Ziemssen's Cyclopædia of Medicine*, vol. xiii. p. 712.

abolished; and it is important to observe that, the paralysis is the first event, and that the atrophy occurs subsequently. The sensory functions are practically intact; slight numbness may be present, but there is never much anæsthesia. The functions of the bladder and rectum are not interfered with.

The rapidity with which the disease is developed varies in different cases; the muscles first affected become rapidly paralysed and soon atrophied, but the *maximum extent* of motor impairment may not be reached for months, or even for years. After the lower extremities have become paralysed, the upper limbs are in turn involved; the extensor muscles of the fingers being the first to suffer, and subsequently those of the upper arm and shoulder. The muscles of the trunk may also be affected. In exceptional cases the paralysis first affects the muscles of the upper extremities, and then extends to the lower limbs. This is the *descending type* of Duchenne. The muscles on one side of the body may be more paralysed and atrophied than those on the other.

A stationary period now generally occurs, but in some cases the lesion continues to extend, ultimately reaching the upper part of the cord and the medulla oblongata; bulbar symptoms are then developed, and death results from respiratory complications.

In the majority of cases improvement occurs after the stationary period has lasted for a few weeks or more. Recovery takes place in the reverse order to the mode of onset, the muscles last affected being the first to recover. In exceptional cases the recovery is complete; but generally some paralysis remains. The total duration of the disease is usually from one to four years.

§ 154. Diagnosis.—The symptoms, negative and positive, clearly show that the morbid process is limited to the region of the anterior cornu. The fact, that the paralysis and atrophy are sometimes completely recovered from, seems to show that the destruction of the multipolar nerve cells is not usually complete, for nerve cells which are once destroyed are never restored.[1]

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[1] The affection presents many points of resemblance to some forms of paralysis which depend upon peripheral lesions. The points of distinction are, however, more marked than those of resemblance, and seem to me to differentiate clearly the

The affection has to be distinguished from *polio-myelitis anterior acuta, progressive muscular atrophy* and *lead paralysis.*

The differential diagnosis of subacute inflammation of the anterior cornua and polio-myelitis anterior acuta is given in the following table:—

	Subacute Inflammation of the Anterior Horn.	Polio-myelitis Anterior Acuta.
Age.	Always in adults, between the ages of thirty and fifty.	Very rarely in adults.
Onset.	Gradual; fever slight or absent; no cerebral symptoms.	Sudden; fever may be considerable; cerebral symptoms (epileptiform convulsions, etc.) at the outset are not uncommon.
The Paralysis.	The paralysis extends from point to point, usually from below upwards; the maximum is never at once reached.	The maximum is always at once reached. Any subsequent changes are in the direction of improvement.
Result.	Usually ends in recovery, which may be complete. When fatal, death occurs after two, three, or four years, and is usually due to extension to the medulla oblongata.	Very seldom fatal, but some paralysis and atrophy usually remain. When death does occur, the fatal termination takes place at the outset.

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two groups of cases. In peripheral paralyses depending upon lesions of sensory-motor nerves, sensory disturbances are of necessity much more prominent painful sensations are experienced in the affected parts, and there is usually considerable anæsthesia: the fact that the morbid process sometimes extends to the medulla oblongata, producing well-marked bulbar symptoms, is in favour of a central lesion; and these clinical arguments are confirmed by the few cases in which a careful examination of the cord has been made after death.

The differential diagnosis of subacute inflammation of the anterior cornual region, and progressive muscular atrophy.

	Subacute Inflammation of the Anterior Horn.	Progressive Muscular Atrophy.
Muscles First Affected.	In the large majority of cases the muscles of the lower extremities are first affected.	In the large majority of cases the muscles of one upper extremity (small muscles of the hand or deltoid) are first affected.
The Condition of the Affected Muscles.	The affected muscles are first paralysed, and subsequently become atrophied. Whole muscles, or groups of muscles, atrophy at once. They present the reaction of degeneration. The reflexes are abolished.	The atrophy is the first event; the loss of motor power follows, and is in direct proportion to the muscular wasting. The atrophy never invades a whole muscle at once, but destroys individual fibres and groups of fibres. The reaction of degeneration is not present. The reflexes are retained until all the muscular fibres are destroyed.
Termination.	The disease does not, as a rule, destroy life. The improvement is often considerable, and occasionally complete.	The disease is very often fatal; muscles which have once atrophied do not recover.

The differential diagnosis of subacute inflammation of the anterior cornual region (Paralysie générale spinale antérieure subaiguë of Duchenne) and lead paralysis.

There is of course no difficulty in distinguishing the typical form of lead palsy (*wrist-drop*) from the affection which we are now considering. Chronic lead poisoning does, however, sometimes give rise to a general form of muscular atrophy, which very closely resembles the *paralysie générale spinale antérieure subaiguë* of Duchenne. In fact, the symptoms are in some cases identical, and the lesion in cases of lead paralysis is thought by some to be a subacute inflammation of the anterior cornua. In such cases the distinction can only be made by observing the mode of development of the symptoms, and the presence or absence of associated evidence of lead impregnation, viz., the blue line on the gums, lead colic, lead rheumatism, etc.; the occupation of the patient should be ascertained, and, if necessary, his drinking water tested.

§ 155. PROGNOSIS.—In most cases the prognosis is favourable as regards life; in some recovery is complete; in the majority some paralysis remains; some prove fatal from extension to the medulla oblongata.

§ 156. TREATMENT.—The treatment is essentially the same as that which has been recommended in polio-myelitis anterior acuta after the febrile stage has subsided.

PROGRESSIVE MUSCULAR ATROPHY.

This affection is comparatively common. It is one of the most chronic, and incurable of all spinal affections. There are probably two varieties of the disease; (1.) a typical form, which commences in the upper extremity, usually in the small muscles of the hand, and in which individual fibres and groups of fibres are gradually destroyed; and (2) an irregular form, which usually commences in the muscles of the lower extremity, and in which whole muscles, or groups of muscles, rather than individual muscular fibres and parts of muscles, undergo atrophy. Many of the cases included in the latter group are doubtless identical with the *paralysie générale spinale antériéure subaiguë* of Duchenne. I shall therefore limit my remarks to the typical form of the disease.

§ 157. PATHOLOGY.—The exact pathology of progressive muscular atrophy is a subject which has given rise to endless discussion. Some authorities hold that the morbid process commences in the muscles; others think the primary lesion is a slow destruction of the multipolar nerve cells of the anterior cornu, and that the muscular atrophy is secondary to the spinal lesion. The latter view is the one which is now generally accepted, and there can, I think, be little doubt that the anatomical substratum of the typical form of progressive muscular atrophy is slow destruction of the motor nerve cells of the spinal cord.

Some authorities think the spinal lesion is an extremely chronic inflammation; others consider it rather as a degenerative process. Be this as it may, the effect of the process is to produce gradual destruction and disappearance of the motor nerve cells of the anterior horn.

On microscopic examination of well-marked cases the lesion

is seen to be practically confined to the anterior cornual region. The nerve cells are in all stages of atrophy and destruction, their processes wasted and degenerated. In two well marked cases reported by Pierret and Troisier,[1] the atrophy of the nerve cells of the anterior horn was simple, the cell processes were wasted, the atrophied cells, even the smallest, still contained a nucleus; exactly the same conditions were also found by Charcot and Gombault.[2] Luys, Lockhart Clarke, and others, have observed dilatation of the blood-vessels of the affected region, and thickening of the vascular walls. In some cases compound granule corpuscles and oil globules are scattered through the affected parts of the cord, apparently replacing the nerve cells, and adhering to the outer coats of the vessels.

The *anterior nerve roots* proceeding from the affected part of the cord are atrophied, but seldom to the same extent as in *polio-myelitis anterior acuta*. The anterior column of the cord through which the affected anterior roots pass, and the adjacent parts of the cord sometimes exhibit sclerotic changes.

The condition of the affected muscles.—To the naked eye the muscles are pale and fawn coloured. The interstitial connective tissue, and, in some cases, the interstitial fat, are increased. The atrophy of the fibres is, according to Pierret and Charcot (see fig. 117), a simple atrophy; the transverse striæ being preserved until the end; the nuclear proliferation and fatty infiltration of the fibres, which are seen in *polio-myelitis anterior acuta*, are not generally present, though in some cases these changes have been observed.

§ 158. General Considerations and Etiology.—The disease is essentially one of *adult* life; cases are seldom met with before the age of twenty-five. The period of greatest frequency is between thirty and fifty.

The pseudo-hypertrophic paralysis of children is thought by some writers to be identical with progressive muscular atrophy. The two affections undoubtedly present many analogies, but are probably distinct.

The *male* sex is much more frequently affected than the female.

Of 176 cases collected by Friedreich, only thirty-three, or about 19 per cent., were females.

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[1] *Archives de Physiologie*, vol. ii., 1875, page 243. [2] *Ibid.* page 747.

The disease is often *hereditary*, and cases have been reported in which the affection has been handed down through, as many as five, generations. Various exciting causes have been described; excessive muscular exercise, and the consequent exhaustion of certain muscles, is a well ascertained cause, and explains the tendency to the affection in persons who follow particular occupations; traumatic injuries[1] of peripheral parts seem occasionally to give rise to the condition; a chill is sometimes the only apparent cause; the affection has been known to follow an attack of typhoid, measles, or acute rheumatism; lead impregnation is sometimes followed by a general form of the disease, and I have seen one case in which it apparently was the cause of the typical variety.

§ 159. ONSET, SYMPTOMS, AND COURSE.—The onset is very gradual, and is not attended with febrile symptoms. In many cases the patient is unaware of his condition until the disease has made considerable progress. The first symptom is motor weakness; it attracts attention, and, on examination, the weakened muscles are found to have undergone considerable atrophy, the muscular weakness being in direct proportion to the muscular wasting. The atrophy gradually increases, and the muscular weakness becomes greater, but there is no true paralysis until the muscular fibres are entirely destroyed. The interossei or thenar muscles of one, generally the right, hand, are usually first affected; occasionally the deltoid is the first to suffer; exceptionally the disease commences in the muscles of the trunk, and still more rarely in those of the lower extremity.

Of 146 cases collected by Friedreich, 111 commenced in the upper extremity, 27 in the lower extremity, and 8 in the lumbar muscles. Duchenne only saw the disease commence twice, in the lower extremities, out of 159 cases.

The condition of the affected muscles.—The affected muscles are *flaccid*. There are no *qualitative* electrical alterations, the 'reaction of degeneration' is not present, and the force of the muscular contractions produced by the Faradic (interrupted) current is in direct proportion to the number of healthy

[1] Possibly this was the cause in a case which I have reported in Brain. Vol. xi. page 396.

muscular fibres which remain. The *reflexes* may be increased.[1] in the earlier stages, but soon become diminished; it is not, however, until the muscular fibres are entirely destroyed that reflex movements are entirely abolished. (See fig. 106.)

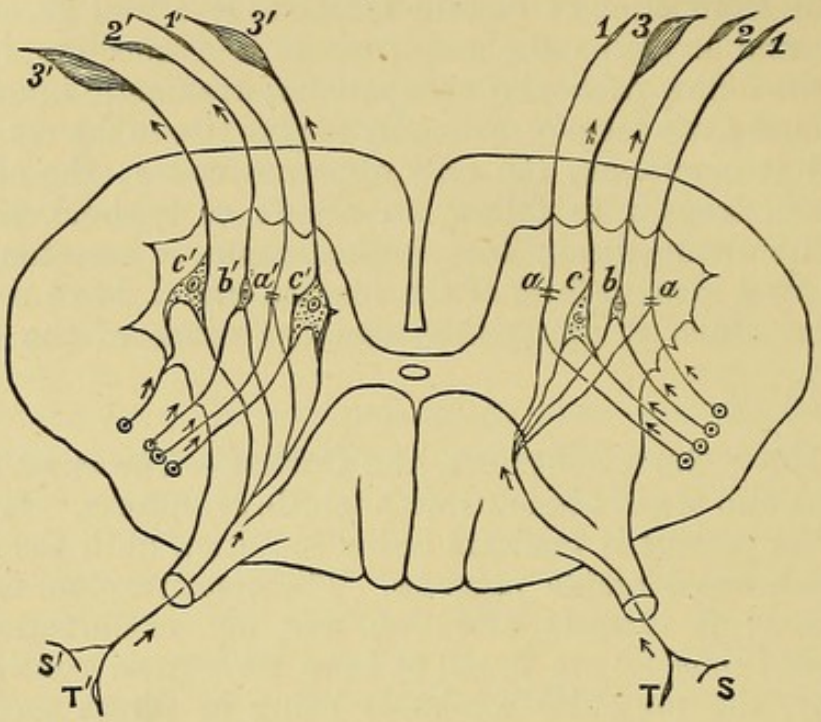


FIG. 106.

Diagrammatic representation of the symptoms which result from slow destruction of the multipolar nerve cells of the anterior cornu.

On the left side the disease is in an early stage. One nerve cell (a′) is completely destroyed. Its muscular fibre (1′) is completely atrophied. Voluntary motor and reflex motor impulses are 'blocked' at the seat of the lesion (a′). One nerve cell (b′) and its muscular fibre 2′ are very much atrophied, but feeble motor and reflex impulses can still pass through the cell to the muscle. Two nerve cells c′ c′ are healthy. Their muscular fibres are of normal bulk, and can be made to contract either by voluntary or reflex impulses.

On the right side the disease is much more advanced. The muscular area is three-fourths degenerated. There is a total 'block' at a and a. This condition represents a late stage of progressive muscular atrophy. The atrophy of the muscular fibres is represented as *simple*. There is not the same fatty change as is shown in figure 37.

Fibrillary twitchings occur in the affected muscles; they are, as I have previously stated, more frequent and constant

[1] In many wasting diseases, as for instance in phthisis, there is increased muscular irritability. Under such circumstances the reflexes, especially the deep reflexes, may be more easily obtained than in health.

in progressive muscular atrophy than in any other affection, but are by no means pathognomic. The *temperature* of the affected parts is lowered, and the patient almost always exhibits increased sensitiveness to cold.

Muscular (myalgic) and joint-pains, resembling rheumatism, are not uncommon, but the skin sensibility is not deranged to any noticeable degree. There are no trophic disturbances in the skin. The functions of the bladder, rectum, and sexual organs are all perfectly normal.

Atrophy of the interossei (the muscles of the forearm being unaffected) causes a peculiar alteration in the position of the fingers, to which the term '*clawed hand*,' or '*main en griffe*,' has been given (see figs. 107, 108, 110, and 111.)

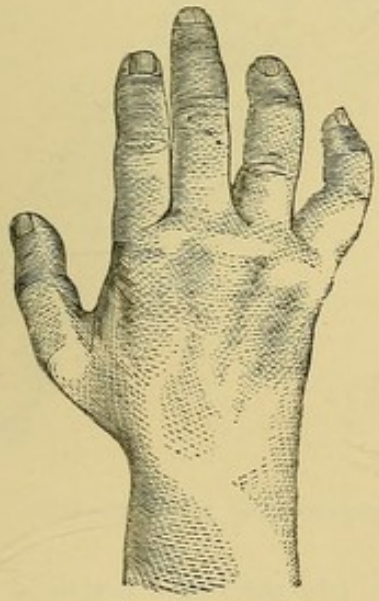


FIG. 107.

The *main en griffe* in progressive muscular atrophy. The interossei and thenar muscles are almost entirely destroyed. (*After Duchenne.*)



FIG. 108.

The *main en griffe* which results from paralysis of the interossei in consequence of a traumatic lesion of the ulnar nerve. (*After Duchenne.*)

The manner in which this alteration in the position of the fingers is produced is as follows:—

The combined action of the internal and external interossei produces (as Duchenne was the first to demonstrate), a

movement of the fingers in which the first phalanx is flexed on the meta-carpus, and the second and third phalanges are kept extended; in other words, the fingers are placed in the writing position. The direction of the tendon of the interossei perfectly explains this contradictory action upon the phalanges. In the first part of its course (from the meta-carpal phalangeal articulation to the upper surface of the first phalanx) the tendon is directed obliquely from above downwards and from before backwards (see fig. 109), there is therefore flexion of the phalanx on its meta-carpal bone during contraction of the muscle. The second part of the tendon, united by an aponeurotic expansion to the tendon of the common extensor, is placed on the back of the phalangeal articulation, and consequently produces their extension.[1]

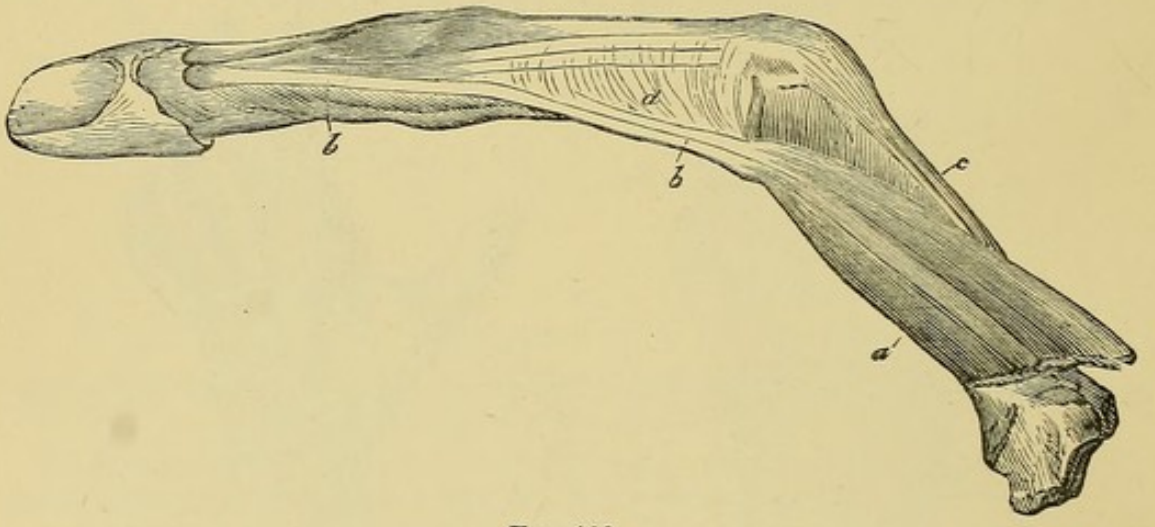


FIG. 109.

Ring finger of the right hand, with its dorsal interosseous or adductor muscle.
(*After Duchenne.*)

(*a*) Dorsal interosseous or adductor muscle; (*bb*) phalangeal tendon of the dorsal interosseous; (*c*) tendon of the extensor communis; (*d*) aponeurotic expansion, which unites the phalangeal tendon of the interosseous with the tendon of the extensor.

Now, when the interossei are paralysed, the opponent muscles have full play, the result being extension of the

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[1] *L'Electrisation localisée*, 1872, p. 966, *et seq.*

[2] According to Duchenne the interossei are the only flexors of the first phalanx, and the only extensors of the second and third phalanges. He adds, Les lombricaux sont, il est vrai, auxiliaires de ces mouvements; mais ils n'exercent point d'action de latéralité sur les doigts,' *loc. cit.*, page 967.



FIG. 110.

The appearance of the hand (dorsal aspect) after division of the ulnar nerve.

For a description of this plate see page 193.

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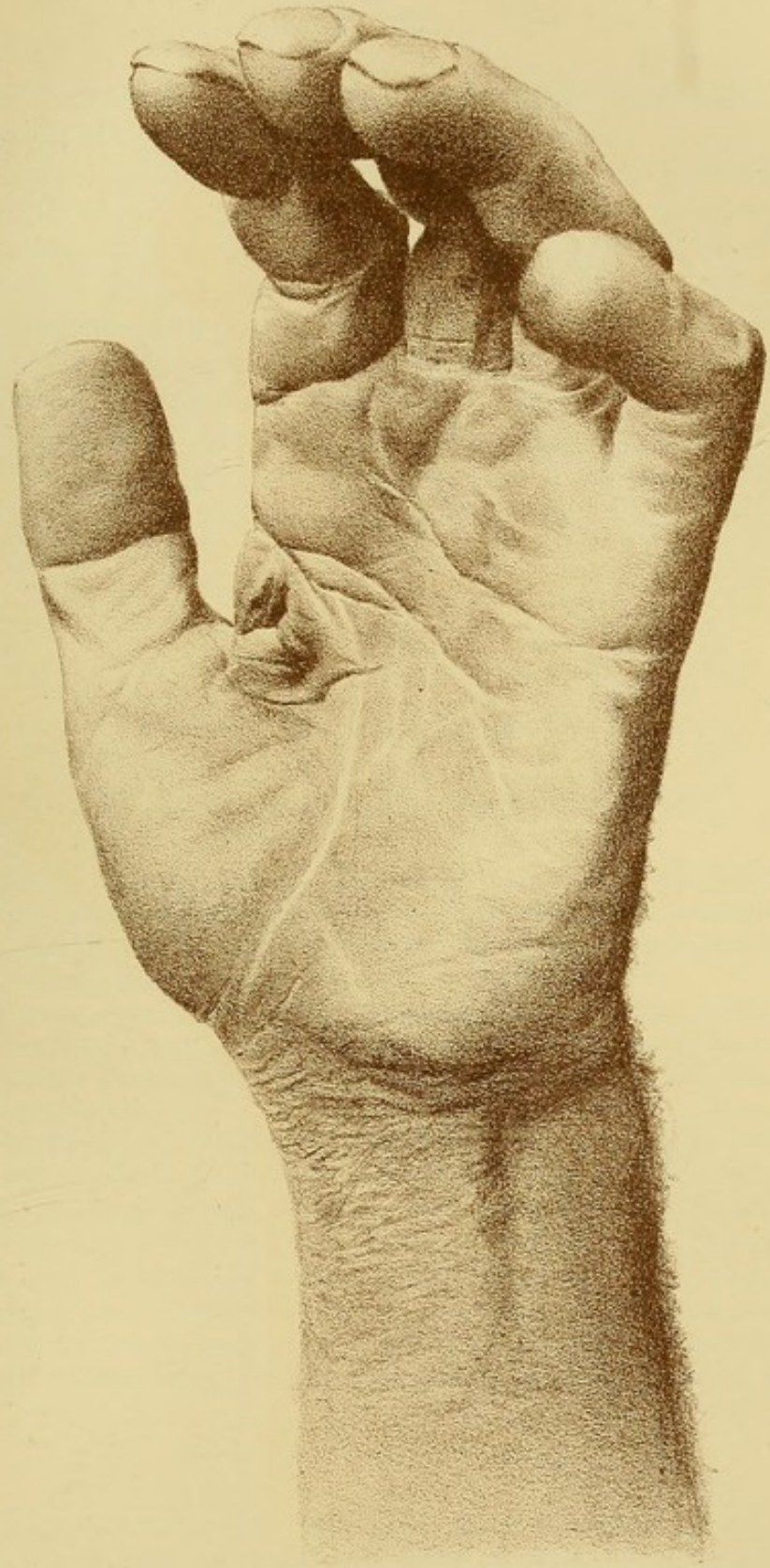


FIG. 111.

The appearance of the hand (palmar aspect) after division of the ulnar nerve.

For a description of this plate see page 193.

M^c^Lagan & Cumming, Lith. Edin^r^.

first, and flexion of the second and third phalanges; in short, the hand assumes the bird-claw position.

The *main en griffe* is not pathognomic of progressive muscular atrophy; it indicates paralysis of the interossei, a condition which may, of course, be due to any lesion of the ulnar nerve. The appearance of the hand in the two cases (progressive muscular atrophy and paralysis due to a lesion of the ulnar nerve) is, however, somewhat different, a fact to which attention was first directed by Duchenne. In progressive muscular atrophy, all the fingers are equally affected; in paralysis of the ulnar nerve, the ring and middle fingers are more particularly involved. (See figs. 107, 108, 110, and 111).

DESCRIPTION OF FIGS. 110 AND 111.

The condition of the Hand after a Lesion of the Ulnar Nerve.

The patient, a labourer, æt. 47, came under my notice in the year 1879 Eighteen years previously he received a severe cut with a cooper's knife in the region of the left elbow. The wound did not heal for eight or nine months, and it was fourteen months before he could use the hand at all.

A cicatrix two inches long extended from the tip of the oleranon to the interna condyle, which was much thickened. All the muscles supplied by the ulnar nerve were paralysed, the wasting of the interossei being well seen in fig. 110. The *main en griffe* was well marked.

Tactile sensibility was impaired in the little finger; sensibility to pain was completely abolished in the little finger, and much impaired in the ring finger.

Soon after the injury the nail of the little finger began to come off in hard, dry chips, and ultimately it was completely detached.—(See fig. 111.) The patient stated 'that every three or four months a bleb, containing 'water as clear as crystal,' forms on the tip of the little finger; it appears in the course of a few hours, usually through the night, and is sometimes as large as a grape. For some days before the bleb forms he feels a stinging sensation in the region of the cicatrix.'

The appearance which the hand and forearm present in progressive muscular atrophy are well shown in figures 112 113, 114 which are copied from Duchenne.

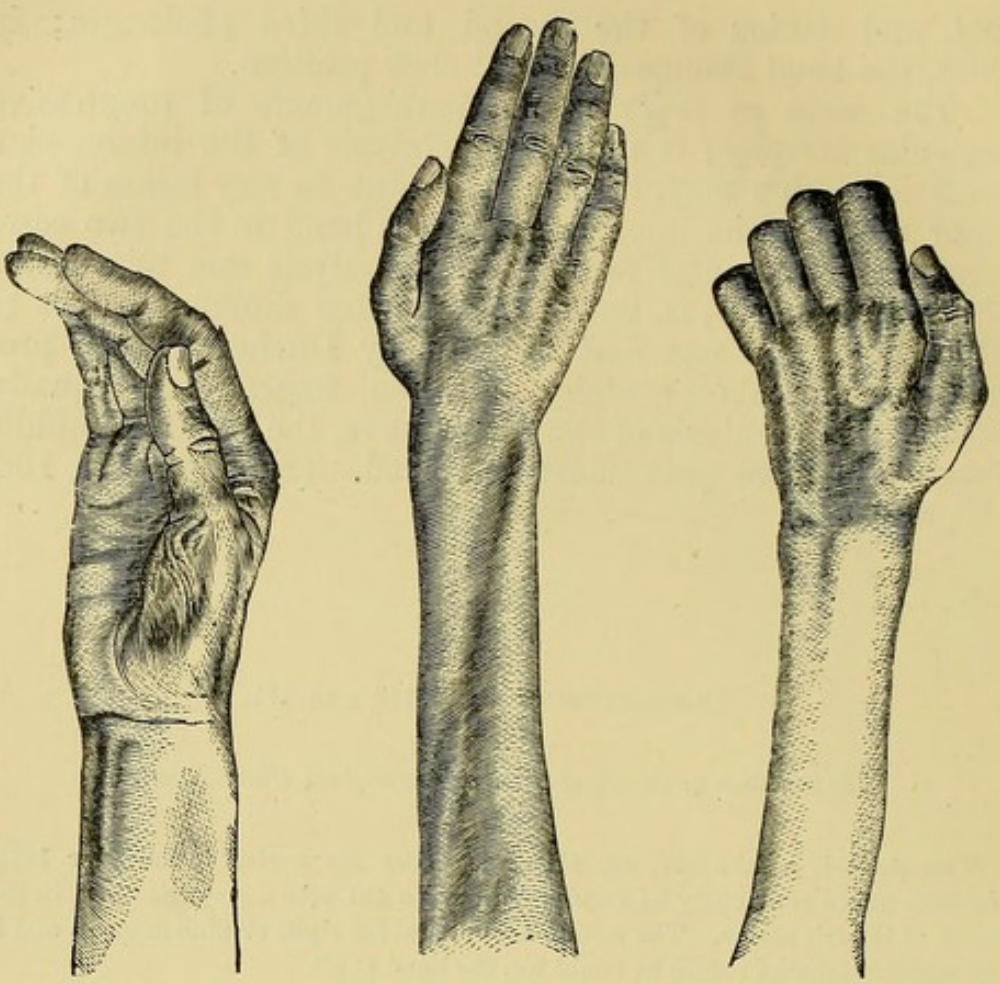


FIG. 112. FIG. 113. FIG. 114.

FIG. 112.—Hand in a case of progressive muscular atrophy which commenced in the muscles of the thumb. (*After Duchenne.*)

FIG. 113 AND 114.—Hands, the muscles of which are almost entirely destroyed, from a case of progressive muscular atrophy, which had become general at the end of two years. (*After Duchenne.*)

After the condition, just described, has continued for a longer or a shorter period, and after the atrophy in the parts first affected has made some progress, other muscles are attacked in a similar manner. The homologous muscles on the opposite side of the body are usually next attacked. When, for instance, the muscles of the right hand are the first to suffer, the muscles next invaded are those of the left hand; in other words, the morbid process extends to the opposite anterior cornu in the segment first affected. Ex-

ceptionally the lesion extends upwards in a vertical direction, and attacks the anterior cornu of another spinal segment on the same or on the opposite side of the body. In this manner the deltoid may be the muscle next invaded. The atrophy, then, progresses by leaps, and invades muscles or groups of muscles which are widely separated, but whose nerve nuclei are in close juxtaposition—a strong argument in favour of the central (spinal) theory of the lesion.

After the muscles, or rather some of the muscles, of both upper extremities have become invaded, the atrophy may extend to the muscles of the trunk, and then to those of the lower extremities; in the course of time almost all the voluntary muscles of the body may become implicated. In some cases the nerve-nuclei of the medulla oblongata are attacked, and bulbar symptoms[1] are developed.

In rare cases the atrophy commences in the trunk muscles, and produces characteristic alterations in the position of the trunk: where the lumbar muscles are affected the back is strongly arched, and the line of gravity falls behind the sacrum (see fig. 115); where the abdominal muscles are extensively atrophied the back is also arched, the result of the unopposed action of the sacro-lumbar muscles, but the line of gravity falls within the sacrum (see fig. 116). When the muscles of the lower extremities become involved, which is seldom until the last stage of the disease, the 'loosely strung' gait (see page 82) may be observed.

Death sometimes results from simple exhaustion, but is much more frequently due to extension of the disease to the medulla oblongata, or to some respiratory complication. When the intercostal muscles or diaphragm are atrophied and paralysed, a slight bronchial catarrh may prove fatal.

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[1] By the term 'bulbar symptoms,' I mean all the symptoms which are characteristic of glosso-labial or bulbar paralysis, viz., difficulty in deglutition and articulation as well as atrophy of the tongue and facial muscles. Very exceptionally we find atrophy of the voluntary muscles supplied by nerves arising from the pons and medulla, but no difficulty in deglutition or articulation. I have recorded such a case (see *Brain*, Part xi. p. 396), in which the muscles of one-half of the tongue were in an advanced stage of atrophy, but in which there were no 'bulbar' symptoms proper. The condition of the tongue had evidently lasted for some time, but the patient did not know that there was anything the matter with it.

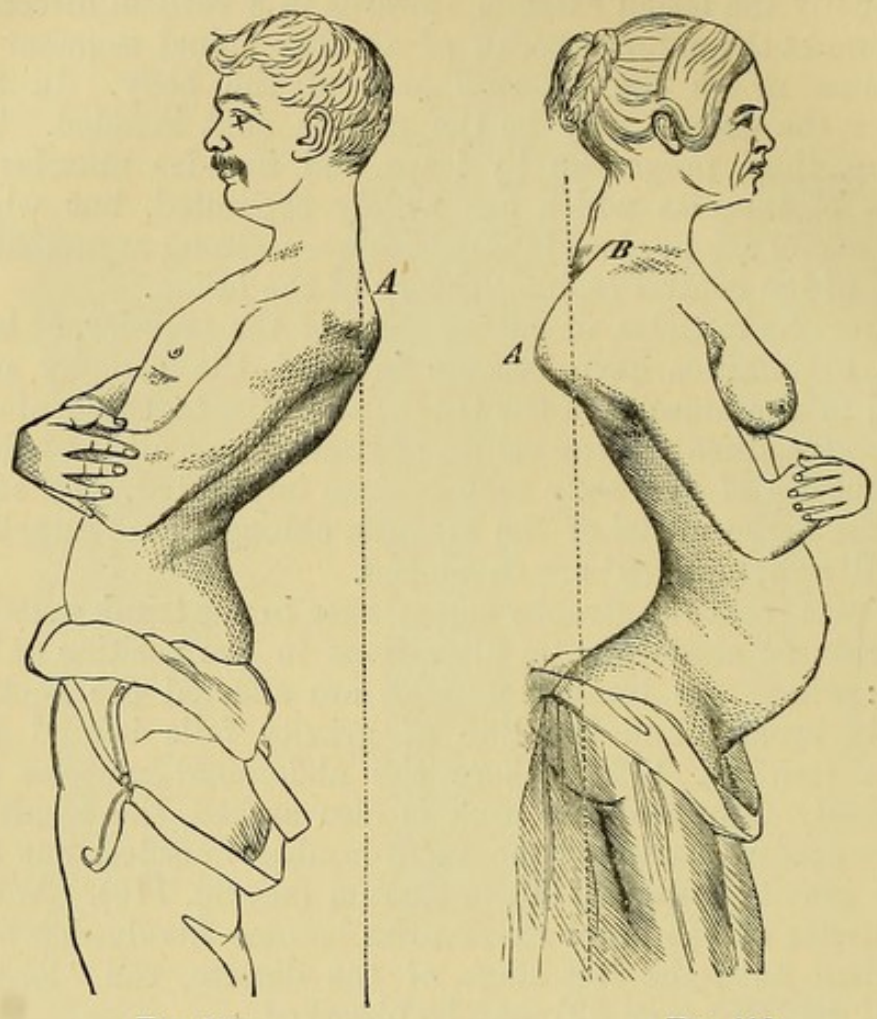


FIG. 115. FIG. 116.

FIG. 115.—*Atrophy of the lumbar muscles in Progressive Muscular Atrophy.* (*After Duchenne.*)

In the erect position the back is curved, so that a line, drawn perpendicularly downwards from the shoulders, falls behind the sacrum.

FIG. 116.—*Atrophy of the abdominal muscles in Progressive Muscular Atrophy* (*After Duchenne.*)

The back is bent backwards by the unopposed action of the lumbar muscles which are healthy.

§ 160. DIAGNOSIS.—The symptoms, negative and positive, clearly show that the lesion is *limited* to the anterior cornu.[1] The disease can only, therefore, be confounded with *poliomyelitis anterior acuta*, or with the *paralysie générale spinale antériéure subaiguë of Duchenne.*

[1] Granting of course that the disease is due to a spinal lesion, and that it is not a primary muscular affection.

The differential diagnosis of progressive muscular atrophy and polio-myelitis anterior acuta is self-evident. The mode of onset of the attack (very gradual in the former, acute in the latter); and the fact that in polio-myelitis anterior acuta the paralysis is the first event and the atrophy secondary, while in progressive muscular atrophy there is no paralysis, but only muscular weakness in proportion to the muscular atrophy, are, irrespective of the many other differences, absolutely conclusive.

The differential diagnosis of progressive muscular atrophy, and the *paralysie générale spinale antériéure subaiguë* of Duchenne has already been considered. (See page 186.)

The *main en griffe,* which results from lesions of the ulnar nerve, may be mistaken at first sight for progressive muscular atrophy in its early stage; but the appearance of the hand is somewhat different (see figs. 107, 108, 110, and 111); while the history of the case, the presence of an injury or cicatrix in the course of the ulnar nerve, and the condition of sensation, are quite distinctive. The points of difference are shown in the following table:—

Differential diagnosis of progressive muscular atrophy in its first stage, and paralysis resulting from a lesion of the ulnar nerve.

	Progressive Muscular Atrophy.	Lesion of Ulnar Nerve.
Onset.	Very gradual—the atorphy is the first event, the motor weakness secondary.	In traumatic cases the onset is sudden, and the paralysis precedes the atrophy.
Appearance of the Hand.	All the fingers are equally flexed, for all the interossei are equally atrophied.	The ring and little fingers are much more bent than the others, for the first two lumbrical muscles, which are supplied by a branch of the median nerve, escape.
Condition of Sensibility.	Unaffected.	Anæsthesia of the skin of both sides of the little, and of the ulnar side of the ring finger.
The Trophic Condition of the Skin.	No trophic alterations.	Trophic alterations in skin supplied by the ulnar nerve may be present. (See fig. 111).
Supposed Cause.	Often indistinct.	In traumatic cases there is a history of an injury, or the presence of a cicatrix in the course of the ulnar nerve.

Secondary lesions of the anterior cornual region, may produce a muscular atrophy, which, so far as the condition of the muscles is concerned, exactly resembles that seen in the advanced stages of progressive muscular atrophy. The history of the case, and the mode of onset of the atrophy are however quite different; in all the secondary forms of atrophy, such as muscular atrophy complicating lateral sclerosis; amyotrophic lateral sclerosis; muscular atrophy complicating sclerosis of the postero-external columns, etc., spinal symptoms of a distinctive character precede the atrophy, which does not follow the classical type.

§ 161. THE PROGNOSIS is unfavourable; most cases ultimately terminating in death. The duration is very variable: the majority of cases last for several years; some run a rapid course, and prove fatal within two years; occasionally the progress of the disease is arrested, and this happy result seems sometimes to be due to treatment.

Cases which progress rapidly, in which the atrophy tends to become general, and in which the disease is hereditary, almost invariably terminate in death; the prognosis is more favourable in those cases in which the disease is limited in distribution, and in which an exciting cause, such as over-use of the affected muscles can be clearly traced. Where the intercostal muscles or diaphragm are affected, or where the lesion has invaded the medulla oblongata and produced bulbar symptoms, a fatal termination is generally close at hand.[1]

§ 162. TREATMENT.—Almost all authorities are agreed that the internal remedies which we at present possess are of little or no avail. The treatment should consist essentially in attending to the condition of the general health, and in the systematic exercise of the affected muscles by gymnastics, kneading, and above all, by electricity; each muscle should be regularly exercised in the manner described on page 168, and the constant current may at the same time be passed through the affected portion of the spinal cord. Arsenic, strychnine,

[1] A limited atrophy of the tongue without bulbar symptoms, as in the case to which I have previously referred, does not of necessity indicate a speedy termination.

iron, and nitrate of silver may be given internally. In syphilitic cases iodide of potassium should of course be prescribed.

In persons who inherit a tendency to the disease, care must be taken to avoid all the exciting causes which we know by experience are apt to set up the condition; it is particularly important to avoid the over-use of any set of muscles; exposure to cold and wet must also be carefully guarded against.

PSEUDO-HYPERTROPHIC PARALYSIS.

This affection resembles *progressive muscular atrophy*, inas-much as certain muscles undergo slow and gradual atrophy; and that motor weakness, corresponding in degree to the amount of muscular tissue destroyed, is gradually developed. It differs from *progressive muscular atrophy;* (1) in the fact that it is essentially a disease of early life; (2) in the order of development of the symptoms — the atrophy commencing in the muscles of the lower extremity, and showing no tendency to invade the small muscles of the hand, which are almost *constantly* involved in progressive muscular atrophy; and (3) in the circumstance that *some of the affected muscles become increased in size.* This increase is not a true hypertrophy, but depends upon an increase of the interstitial fibrous tissue and fat, hence the name pseudo-hypertrophic paralysis which has been given to the affection.

§ 163. PATHOLOGY.—It is still undecided whether pseudo-hypertrophic paralysis is due to a spinal lesion or to a local affection of the muscles. Analogy would, I think, lead us to suppose that the disease is due to a lesion of the anterior cornu. Opportunities of examining cases *post-mortem* are not frequent, and the few cases which have been, as yet, thoroughly investigated have yielded contradictory results. Thus, Charcot and Cohnheim have found the cord perfectly sound, while Gowers and Drummond have observed alterations in the grey matter. In the case examined by Drs Gowers and Lockhart Clarke, 'changes were scattered throughout the entire length of the cord, the most extensive lesion being found in the lowest part of the dorsal region, where in each lateral grey substance was an area of disintegration, amounting to an actual cavity outside each posterior vesicular column, which, with the caput cornu posterioris, and anterior cornu, was

undamaged.'[1] In the case recently reported by Drummond[2] the grey matter of the anterior horn, at its junction with the posterior cornu and in its lateral part, was softened and disintegrated, so that a cavity was formed in the centre of the cord. This cavity, which had no proper cyst wall, was of large size in the lumbar enlargement, where it caused the cord to bulge out laterally, and extended through the dorsal and cervical regions. 'With a high power ($\frac{1}{4}$ inch), minute disintegration could be traced through the lateral grey net-work of both sides, the degenerative appearances being most conspicuous around the blood-vessels.' The nerve cells of the anterior horn were both numerous and healthy. In describing these appearances, Dr Drummond cautiously says, 'I do not mean to assert confidently that this lateral disintegration or tearing was of pathological significance; it may have been due to the manipulation, but I am inclined to think otherwise.

It is, then, still doubtful whether pseudo-hypertrophic paralysis is a disease of the cord or a disease of the muscles. But since the condition presents so many analogies with progressive muscular atrophy, it seems only proper to consider it along with that affection.

The condition of the Muscles.—The affected muscles undergo simple atrophy; the interstitial connective tissue is largely developed; fat cells are deposited between the muscular fibres. The increased size of the muscles is due to the increase of these interstitial products; the atrophy of the muscular fibres seems in great part secondary, and it probably depends upon the pressure of the interstitial fibrous tissue and fat. The changes in the muscle are well seen in figure 119, which is copied from Gowers.[3]

While these sheets were passing through the press, I received from Dr Milner Moore of Coventry, the spinal cord and a portion of the heart of the case of pseudo-hypertrophic paralysis (that of J. S.) reported by him in the *Lancet*, June 19, 1880.

Time has not permitted an exhaustive investigation, but I have been enabled to examine part of the cervical and dorsal

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[1] *Pseudo-Hypertrophic Paralysis*, page 43. [2] *Lancet*, Oct. 15, 1881, p. 661.

[3] In all organs an increase in the interstitial connective tissue is followed by atrophy of the parachyma (the proper elements) of the organ. The most striking illustration of this pathological fact is the atrophy of the liver cells, which occurs in cirrhosis of that organ.

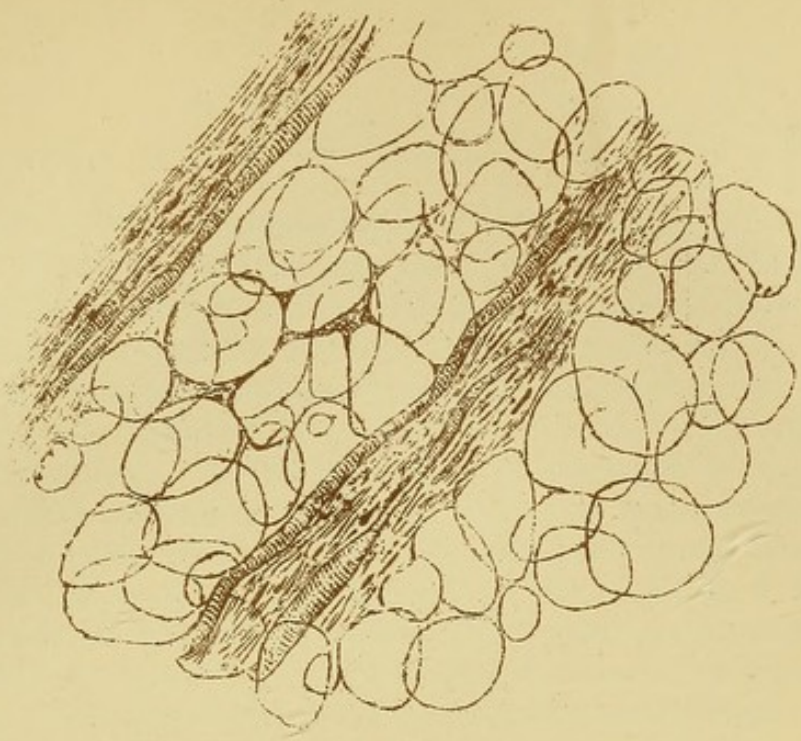


FIG. 119.

Section of the gastrocnemius muscle in a case of Pseudo-hypertrophic Paralysis.
(*After Gowers.*)

Fat cells separate two tracts of nucleated fibrous tissue, containing striated muscular fibres. The latter are much narrowed, and irregular in thickness, but preserve their transverse striation except in the narrowest portion.

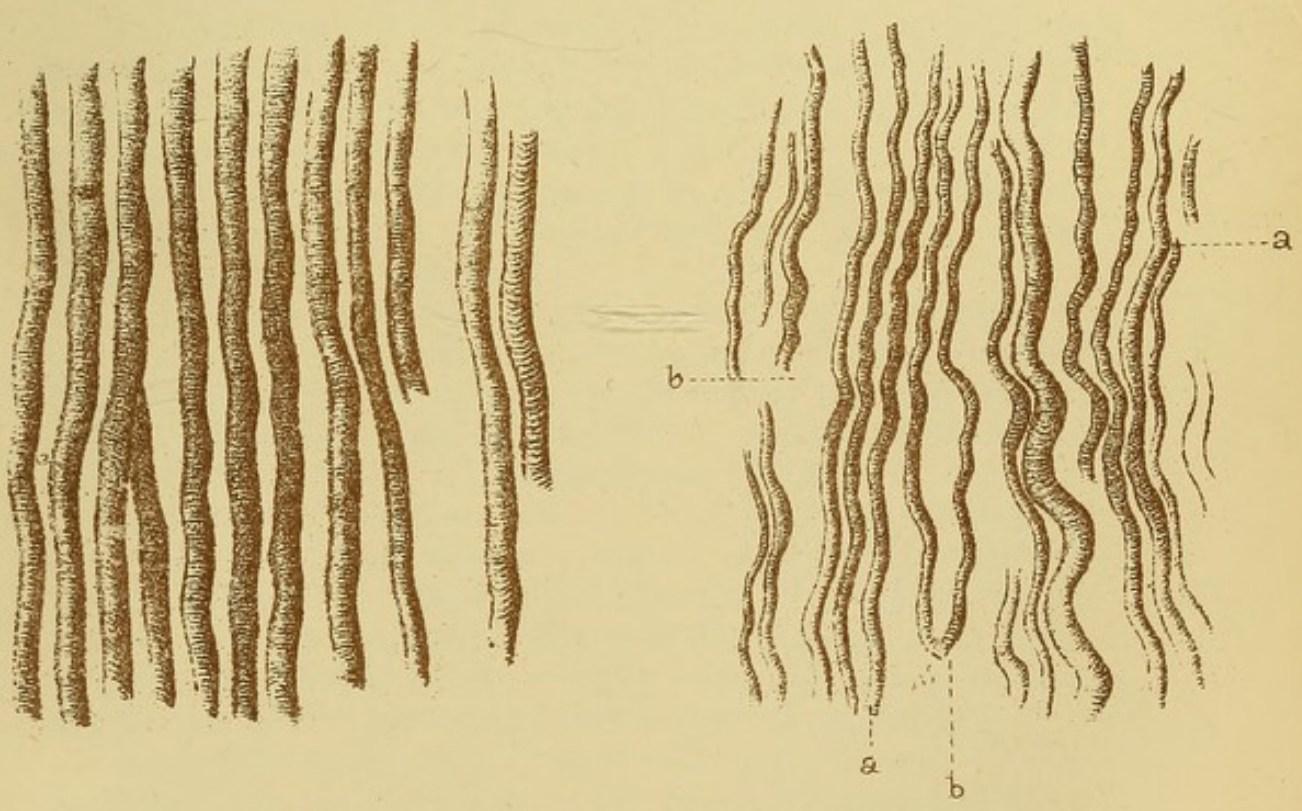


FIG. 117. *Muscular fibres from the diaphragm in a case of Progressive Muscular Atrophy.*
(*After Charcot.*)

The muscular fibres (a) are much atrophied, but preserve their transverse striæ. The connective tissue intervals (b) are enlarged.

FIG. 118. *Healthy muscular fibres from the diaphragm, showing the natural size.* (*After Charcot.*)

regions; and as the appearances are very striking, I now publish the following preliminary note, premising that when the examination is completed, Dr Milner Moore intends laying the full clinical particulars, together with my detailed report of the post-mortem appearances, before the profession.

The naked eye examination of the spinal cord.—The membranes were healthy. In the middle of the cervical enlargement the lateral column of the right side was misshaped by a projection or out-growth, which measured from above downwards a little more than half an inch. The surface of the projecting mass, at its most prominent point, was irregular and disintegrated, and exactly resembled nervous tissue in a state of softening. The tip of the filum terminale was swollen, and presented the same ragged, disintegrated appearance.

On cutting across the cord at the level of the bulging (the cord had been for several weeks in a four per cent. solution of bichromate of ammonium before I received it) the irregularity of the lateral column was rendered very evident, and in the lower part of the cervical region a distinct fissure was seen on each side of the cord in the central part of the grey matter.

The cord was cut up into small pieces, some of which were returned to a four per cent. solution of bichromate of ammonium; while others were placed in a solution of bichromate of ammonium and per-osmic acid.

After remaining, for five weeks in all, in the hardening fluid, three portions of the cervical and four portions of the dorsal regions were examined.

Microscopical examination of the cervical region showed:—

1. *A curious alteration in the shape of the right lateral half of the cord, and in the arrangement of its grey matter,* which reached its highest degree of development in the middle of the cervical enlargement (see fig 120).

In that section the central grey matter is seen to be split up by a band of white matter (*a*), which is continuous with the white matter of the posterior column, and by a fissure, which extends from the surface of the posterior column deep down into the grey matter of the anterior horn. (The size of this fissure was much increased in the process of mounting.)

A narrrow wedge-shaped mass (see figs. 120 and 121) is detached from the main portion of the grey matter, and runs right out to the surface of the cord, and into the projecting portion of the lateral column.

In the section shown in figure 120, the dotted line represents a portion of the projecting mass which was detached in the process of mounting. (The actual size of the projection was, however, considerably larger than the dotted line indicates, for a portion of it was detached before the specimen was placed in the microtome.) In figure 122 the relative positions of the cord and the outgrowth from the lateral column are seen.

At the upper level of the out-growth from the lateral column (see fig. 122), the grey matter is of normal shape, the detached wedge-shaped portion having disappeared; the projection from the lateral column is composed of a central grey nucleus containing numerous dilated blood-vessels and some nerve cells, and of an external layer of white matter, the nerve tubes being concentrically grouped round the central grey mass, and running horizontally and not vertically, as the fibres of the lateral column normally do.

At the lower part of the cervical enlargement, the out-growth from the lateral column has disappeared, but the grey matter on the right side is still split up by a band of white matter (see fig. 123) which passes into it from the posterior column.

This peculiar alteration in the shape of the grey matter, and the out-growth from the lateral column were probably, I think, congenital malformations, and not the essential lesion in the case. It is, however, important to remember that a very similar out-growth from the lateral column was present in Dr Drummond's case; it is therefore probable that in pseudo-hypertrophic paralysis there is a strong tendency to congenital malformations in the arrangement of the grey matter and in the shape of the cord.

2. *Collections of leucocytes and patches of inflammatory softening around the blood-vessels.*—Collections of leucocytes (see figs. 124, 125, and 126) were scattered throughout the grey matter in the cervical region, they were especially large and numerous in the district of the median branch of the central artery. The nervous elements, around some of the most recent extravasations, exhibited evidence of inflammatory irritation, the axis-cylinder processes being hypertrophied, the cellular elements extremely numerous, and the nerve cells themselves, every here and there, enlarged. In those parts of the grey matter in which the extravasations were extensive, the nervous elements were entirely destroyed. Collections of leucocytes were occasionally also seen around the blood-vessels in the white columns.

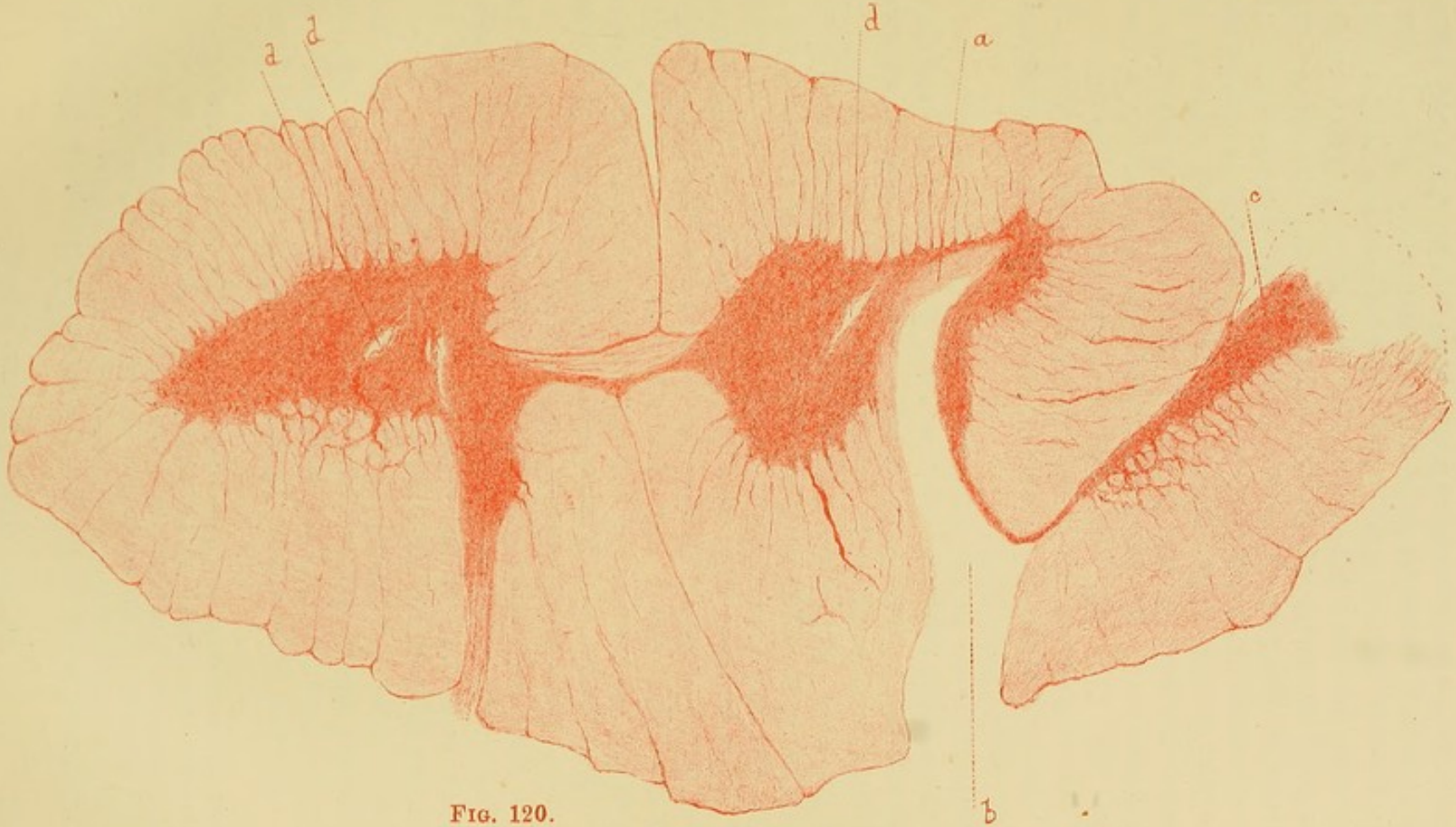


FIG. 120.

Transverse section through the middle of the cervical enlargement in a case of Pseudo-hypertrophic Paralysis. Stained with carmine, mounted in dammar, and magnified about 10 *diameters.*

The grey matter of the right lateral half is split up by a band of white matter (a) which passes into it from the posterior column, and by a deep fissure (b); c, detached portion of grey matter running out to the surface of the lateral column; d,d,d, fissures or tears in the grey matter.

B.B. del^{t} ad. nat^{m}.

McLagan & Cumming, Lith. Edin^{r}.



FIG. 121

The detached portion of grey matter (c in section 120) more highly magnified.

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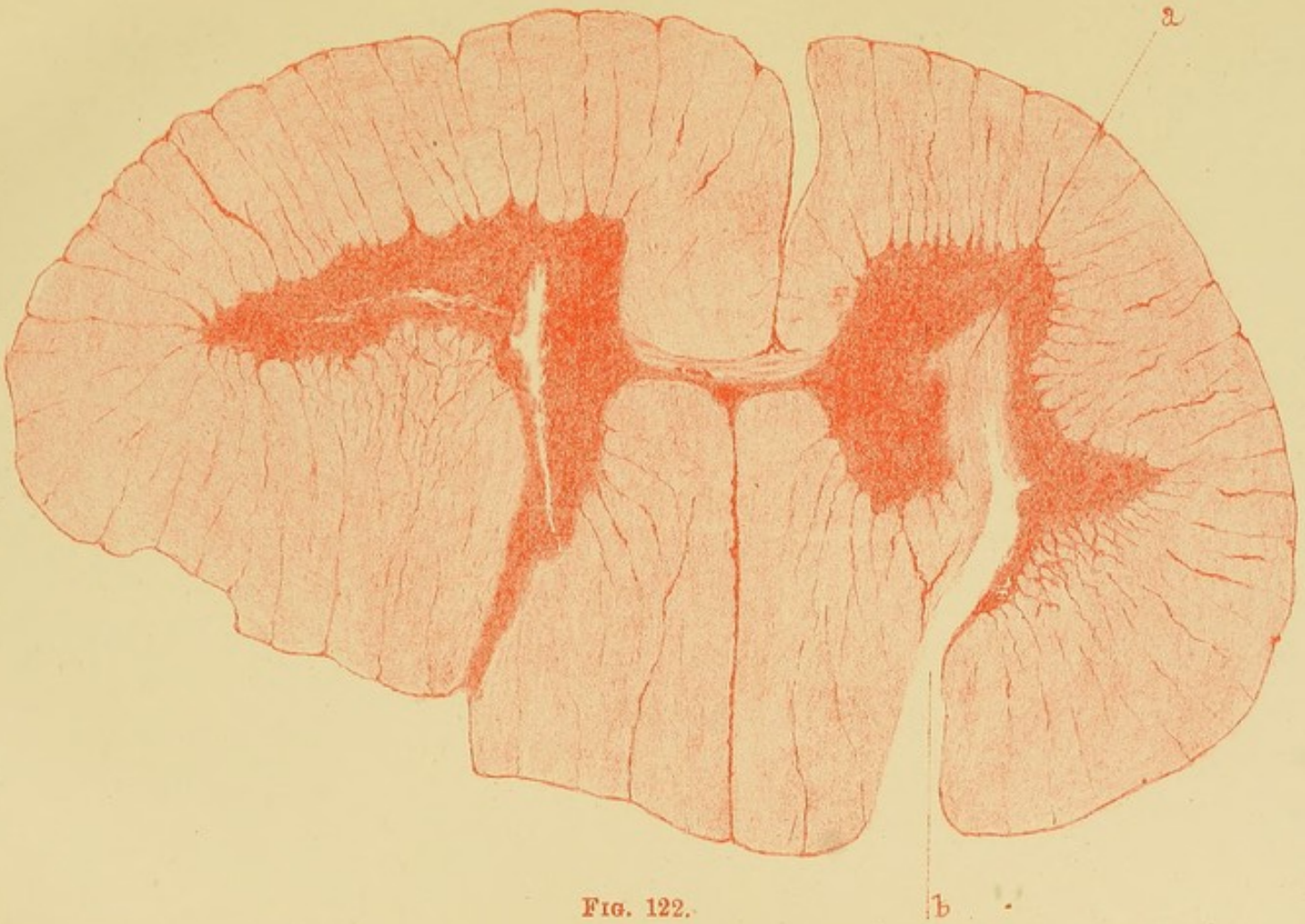


FIG. 122.

Transverse section through the lower part of the cervical enlargement in a case of Pseudo-hypertrophic Paralysis. Stained with carmine, mounted in dammar, and magnified about 10 *diameters.*

The grey matter of the right lateral half is split up by a band of white matter (a), which passes into it from the posterior column, and by a deep fissure (b); the grey matter of the left lateral half is also fissured.

B.B. delt. ad. nat.

McLagan & Cumming, Lith. Edin.



FIG. 123.

Transverse section through the upper part of the cervical enlargement in a case of Pseudo-hypertrophic Paralysis. Stained with carmine, mounted in dammar, and magnified about 10 *diameters.*

The anterior horn of grey matter is of normal shape; the outgrowth from the lateral column, with its central mass of grey matter, is well seen.

a, Terminal branches of the median division of the right central artery.

B.B. delt ad. nat.

McLagan & Cumming, Lith Edin.



FIG. 125.

Transverse section of an artery surrounded with leucocytes from the section shown in fig. 121 *Stained with carmine, mounted in dammar, and magnified about* 250 *diameters.*

Note.—In the preparation itself the leucocytes are of a rusty red colour; the same statement applies to figures 124 and 126.



FIG. 124.

Terminal branches of the median division of the right central artery of the cord, as it passes from the grey matter into the lateral column, in a case of Pseudo-hypertrophic Paralysis. The vessel is surrounded by large masses of leucocytes. Stained with carmine, mounted in dammar, and magnified about 50 *diameters.*

This drawing was made from a portion of the preparation shown in figure 123.

B.B. del. ad. nat.

McLagan & Cumming, Lith. Edin.



FIG. 126.

Right anterior horn of the cervical enlargement, in a case of Pseudo-hypertrophic Paralysis. Stained with carmine, mounted in dammar, and magnified about 50 diameters.

Numerous collections of leucocytes (a a) are scattered through the grey matter, which is split across by a deep fissure (b).

B.B.del^{t} ad. nat^{m}

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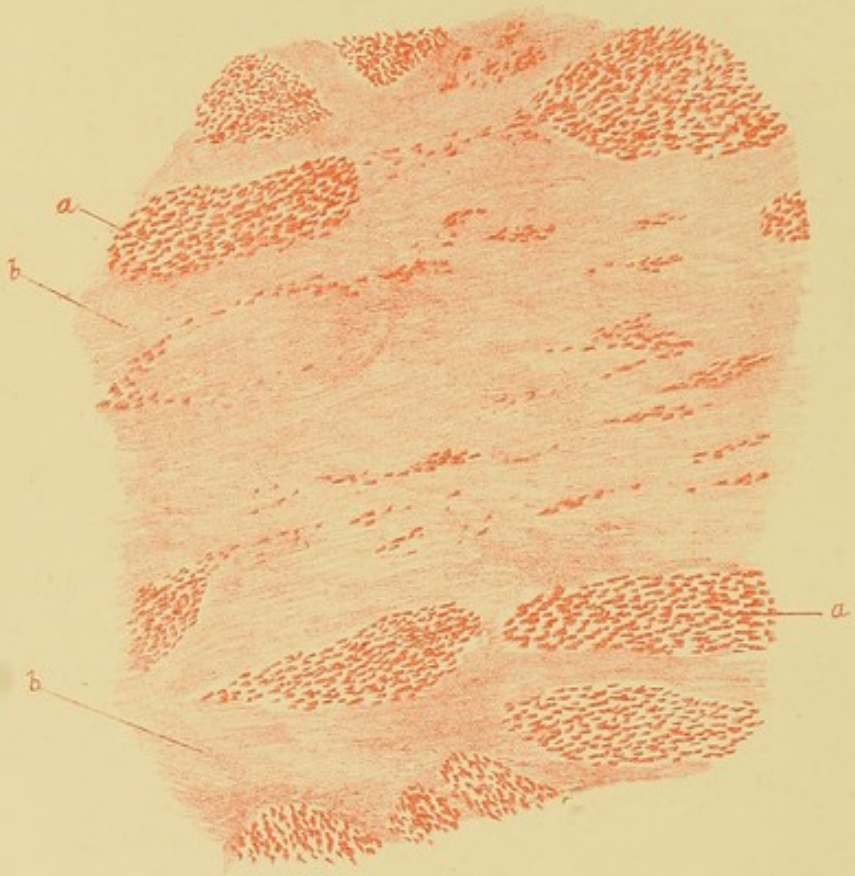


Fig. 127.

Transverse section through the left ventricle of the heart in a case of Pseudo-hypertrophic Paralysis. Stained with picro-carmine, mounted in Farrant's solution, and magnified about 10 *diameters.*

Bundles of muscular fibres, a,a (which in the preparation itself are stained of a dull red colour), are widely separated by masses of connective tissue, b,b, which in the preparation are stained of a bright carmine hue.

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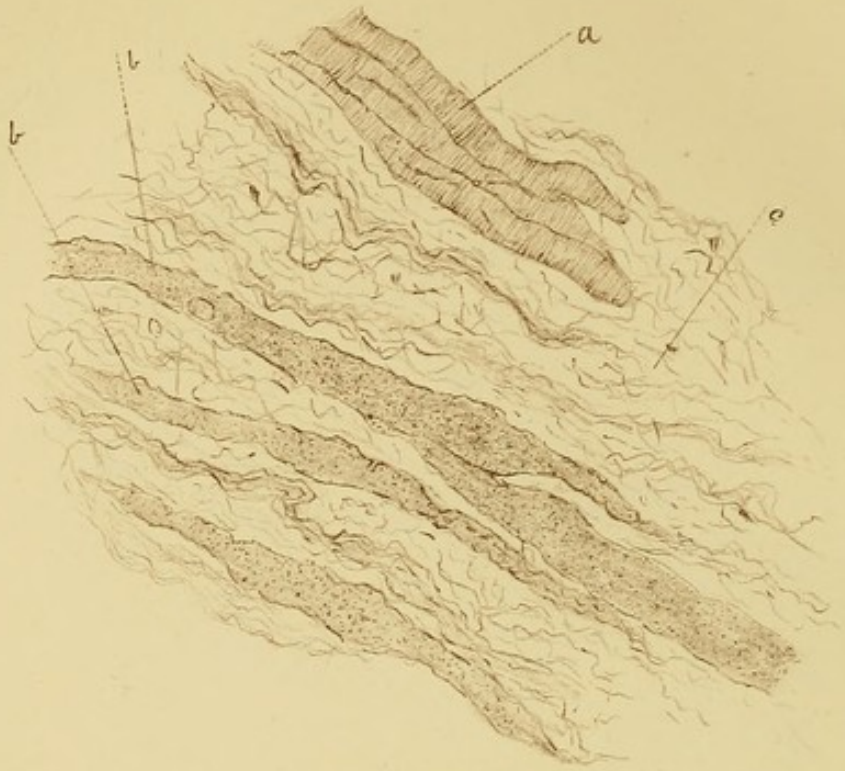


FIG. 128.

Section through the left ventricle of the heart in a case of Pseudo-hypertrophic Paralysis. Magnified about 250 *diameters.*

a, a, Healthy muscular fibres; b, b, degenerated muscular fibres; c, c, bundles of wavy connective tissue.

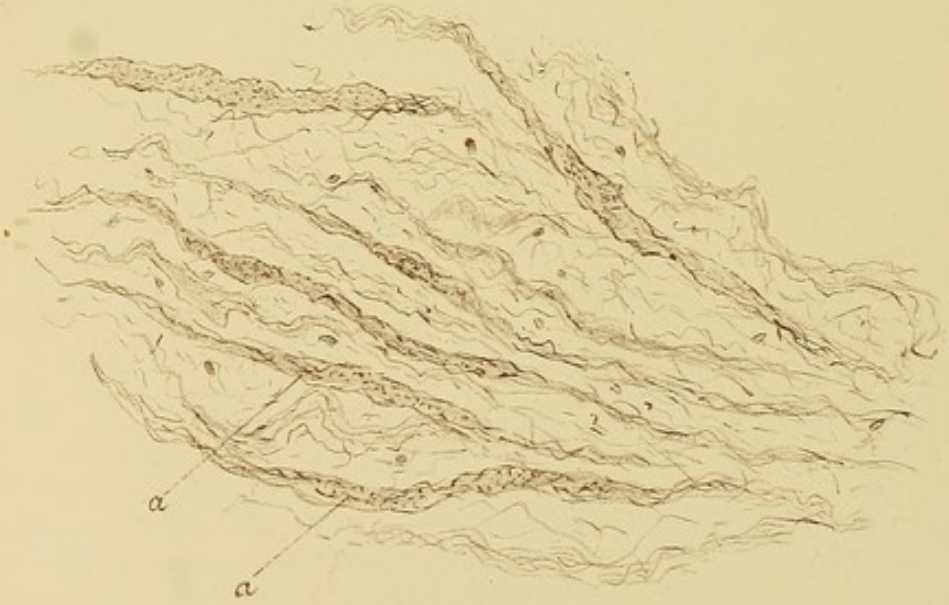


FIG. 129.

Section through the left ventricle of the heart in a case of Pseudo-hypertrophic Paralysis. Magnified about 250 *diameters.*

a, a, Degenerated muscular fibres (?) becoming transformed into connective tissue.

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Mc Lagan & Cumming, Lith. Edin.

3. In the lower part of the cervical enlargement *the grey matter on both sides of the cord was lacerated and fissured.* The examination of numerous sections has convinced me that this appearance was probably produced during the process of hardening. The primary lesion in this case was, I believe, the extravasation of leucocytes and the subsequent occurrence of patches of inflammatory softening around the blood-vessels; during the hardening process these softened patches gave way, and fissures were in this manner produced. The position of the fissures is, I think, explained by the fact that numerous large arteries pass through the central part of the grey matter, and that the extravasations of leucocytes and patches of softening reach their highest degree of development around these large vessels.

The nerve cells were very numerous, and some of them appeared to be hypertrophied.

The *dorsal region* of the cord was normally shaped; on microscopic examination a few small collections of leucocytes were seen in the grey matter. The blood-vessels throughout the dorsal region, and indeed throughout all parts of the cord, which were examined (see fig. 123), were extremely numerous and abnormally dilated.

The examination of the heart.—In the wall of the left ventricle, numerous degenerated patches could be seen with the naked eye; and on microscopical examination they were found to consist of wavy bundles of connective tissue. The atrophy of the muscular fibres does not appear to be a simple one, such as is described by Dr Gowers; but so far as I can at present judge from a somewhat hurried examination, the muscular fibres first become swollen, and filled with minute fatty granules; their nuclei are in places prominent; finally, they atrophy, and are, I believe, transformed into the wavy bundles of connective tissue. These appearances are shown in figs. 127, 128, and 129.

In this case, then, very striking alterations were found in the spinal cord; and the fact that three independent observers have found very similar changes seems to show that the lesion is by no means an accidental one. It is probable, I think, that the same changes will again be found in advanced cases of the disease. These cases do not, however, prove *conclusively* that pseudo-hypertrophic is, *from the first*, a disease of the spinal cord, and not a primary affection of the muscles.

§ 164. Etiology. — Pseudo-hypertrophic paralysis is essentially a disease of early life. The great majority of cases commence before the tenth year.[1] Boys are much more frequently affected than girls. In a considerable number of cases the disease is hereditary, and it presents the remarkable peculiarity that, while the females of a family suffer much less frequently than the males, the disease is almost exclusively transmitted through the female line. The explanation of this circumstance is to be found in the fact that females are rarely affected, and that in males the condition commonly leads to death soon after the time of puberty. The causes of the condition, with the exception of the hereditary tendency, which is present in many cases, are unknown. Dr Gowers thinks the disease less common amongst the poor than amongst those in better circumstances. In some cases, an acute febrile attack, such as measles or scarlet fever, seems to arouse the latent tendency into activity.

§ 165. Onset, Symptoms, and Course.—The commencement is very gradual. The first symptom is weakness and unsteadiness in the lower extremities. The child appears to be clumsy, and frequently stumbles or falls. This motor weakness in the lower extremities gradually becomes greater, and the characteristic attitudes and gait are developed.

The attitude.—The patient stands with his feet wide apart (see fig. 130), so as to enlarge his base of support as much as possible. The heels are usually drawn up as the result of retraction of the tendo Achillis. The back is strongly curved backwards, and in many extreme cases, as Duchenne pointed out, a vertical line from the middle of the shoulders falls behind the sacrum. The hands are extended by the sides, and are used to balance the body. The attitude is a very striking one; and it is remarkable that the upright position can be maintained at all; it shows the extraordinary power of balance and

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[1] 'The date of the earliest symptoms is given,' says Dr Gowers, 'in the records of 139 cases—123 males and 16 females. In one-half of the male cases the disease commenced before the sixth year, and in 102, or about 75 per cent., before the tenth year. On the other hand, of 16 cases in girls, in one only did the first manifestation of the disease coincide with the first walking; in only three cases did it commence before the sixth year, and in only nine cases before the tenth year. Thus the disease begins in or after the tenth year in only 25 per cent. of the males, but in 50 per cent. of the females who suffer from it.'—*Pseudo-hypertrophic Paralysis*, p. 26.

adjustment that can be obtained under very unfavourable circumstances. Although the patient can stand and maintain his equilibrium so long as he is undisturbed by external conditions, he is extremely insecure, the slightest touch in any direction being sufficient to throw him down.

The antero-posterior curvature, which is such a striking feature when the patient stands up, entirely disappears when he assumes the sitting position. Duchenne ascribed the curvature of the spine to weakness of the spinal extensor muscles. Dr Gowers thinks it doubtful whether the lordosis is mainly due to this cause. 'Weakness of these muscles may,' he says, 'as Duchenne showed, cause the shoulders to be carried far back, so as to bring the centre of gravity of the upper part of the trunk as far back as possible; but, according to his description, the inclination backwards from this cause starts from the pelvis, which is more extended on the thigh-bones than normal. But in pseudo-hypertrophic paralysis, in the erect posture, in which posture alone the lordosis is observed, I have found that the pelvis is much less extended, its inclination forwards is much greater than normal. This inclination is probably due to the weakness of the extensors of the hip, and the lordosis is apparently connected with it, since the lowest lumbar vertebræ share the direction of the sacrum, the weight of the abdomen falls unduly forwards, and a compensatory backward inclination of the dorsal spine is necessary to keep the centre of gravity in the normal position.'—*Pseudo-hypertrophic Paralysis*, page 31.

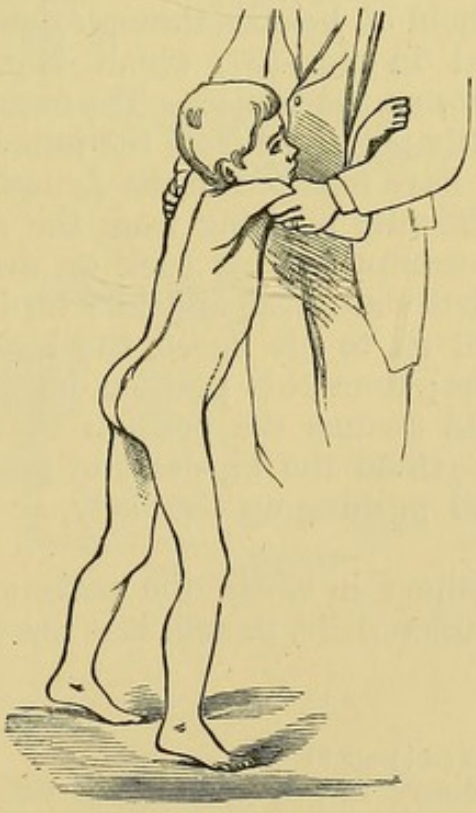


FIG. 130.

An advanced stage of Pseudo-hypertrophic Paralysis, showing the position of the feet in the erect position of the body.

The Gait.—The patient stands in the position described above; the gait is 'waddling,' the body oscillates from side to side; there is an evident difficulty in flexing the thigh on the abdomen, and in projecting the foot forwards. At each step the foot is raised, but the movement in advance is small.

According to Duchenne, the peculiar gait is due to weakness of the gluteus medius muscle. 'This muscle normally,' says Dr Gowers, 'counteracts the tendency of the pelvis at each step to incline towards the leg, which is off the ground; and if the muscle is weak, the weight of the body has to be thrown further over the supporting leg than in health, and hence the oscillating gait.'[1] Dr Ross differs from this opinion, and thinks that the oscillation in walking, instead of being caused by paralysis of the gluteus medius is necessarily effected by contraction of the muscle.[2]

Another remarkable peculiarity, which results from the muscular weakness, is the fact that the patient has great difficulty in rising from the recumbent or sitting position. Indeed in advanced cases he may not be able to raise himself at all. Even in the earlier stages he makes use of his arms, catching hold of chairs or other pieces of furniture, and dragging up his body by means of the upper extremities. When he has nothing to take hold of, he goes through the series of movements represented in fig. 131, which is taken from Dr Gowers' able lecture on the subject. The nature of the movements will be readily perceived from the following description of a case which I have reported in the *Lancet*;[3] 'the patient has the greatest difficulty in rising from the recumbent position. If laid, for example, on his back on the floor, and told to rise, he would first with great difficulty turn on to his face; he would next get on to his knees, the head being almost between the thighs; from this position he would gradually extend himself and assume the position shown in fig. 132. Finally, he would extend the hip-joint by grasping the thigh with the hand, and pushing up the body, as it were, by the arm.'

The peculiar manner in which the patient 'climbs up his thighs' is very characteristic, though it is not pathognomonic.

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[1] *Loc. cit.*, page 31.

[2] *Diseases of the Nervous System*, page 189.

[3] *Lancet*, August 9th, 1879.

I have seen the same action in a case of rickets, and in that of a woman with a diseased (malacosteal) pelvis.

By placing the hands on the knees the patient is able by the force of the arm to aid the extension of the leg on the thigh, *i.e.*, to straighten the knee; and by climbing up the thighs he helps the extension of the hip-joint, as I have already described.

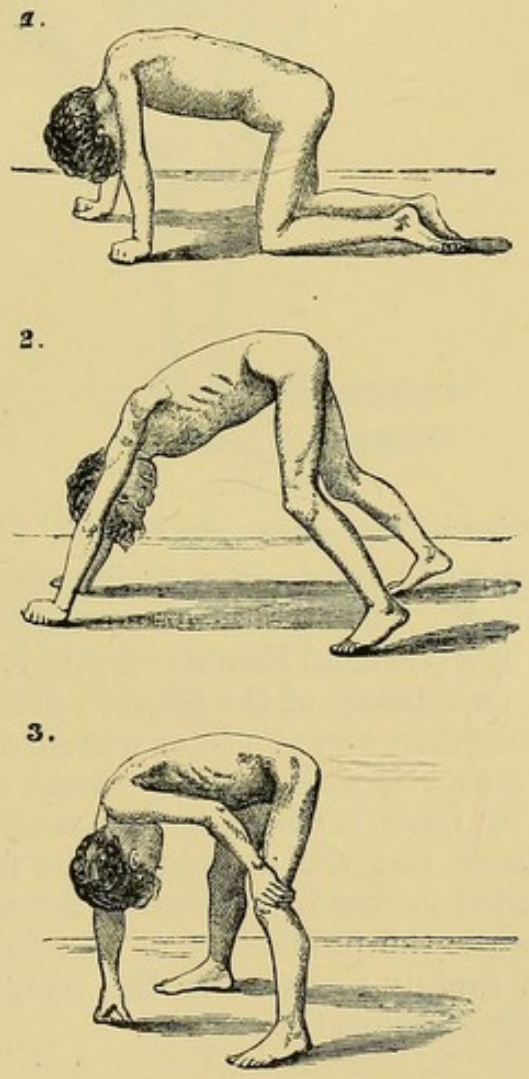


FIG. 131.

The attitudes which patients with Pseudo-hypertrophic Paralysis assume in rising from the recumbent to the erect position. (*After Gowers.*)

Should the patient come under observation at an early period of the disease, the calf muscles will probably be found to be large and firm; they stand out prominently, and appear to be in a permanent condition of contraction. But notwith-

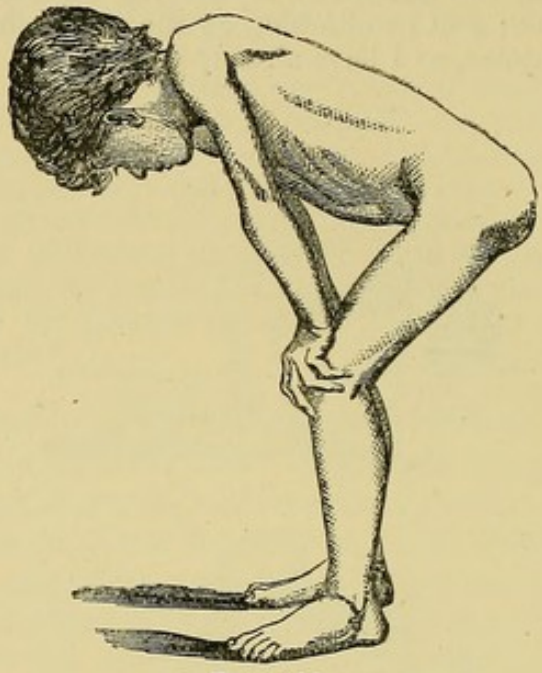


FIG. 132.

Climbing up the thighs in Pseudo-hypertrophic Paralysis. (*After Gowers*).

standing this apparent hypertrophy, their motor force is very distinctly weakened. The amount of contraction, too, which can be produced by a powerful Faradic current is below the normal. The reflex movements of the affected muscles are in the earlier stages diminished, and at a later period, abolished. If a small portion of muscle is withdrawn by means of Duchenne's, or, better still, by Leech's, trocar, the increase of the interstitial fibrous tissue and the atrophy of the muscular fibres can be demonstrated during life. During the active period of the disease, *i.e.*, while the production of the interstitial connective tissue is rapidly progressing, the temperature of the affected parts may be above the normal.[1]

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[1] In the case reported by me in the *Lancet*, the temperature measurements were as follows:—

Right calf, ...	98° F.
Left calf, ...	98.8 F.
Axilla, ...	98.5° F.

Whereas in healthy boys I have never found the surface temperature of the calf as high as that of the axilla.

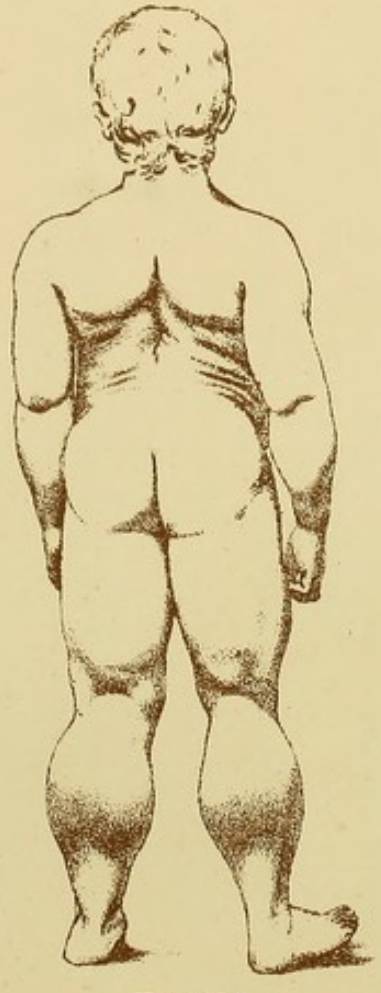


Fig. 134.

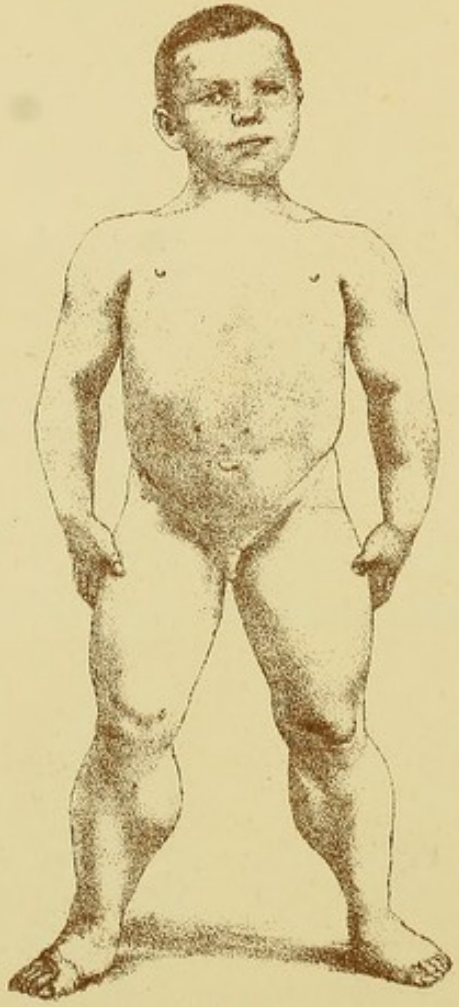


Fig. 133.



Fig. 135.

Fig. 133, *front view; Fig.* 134, *back view; and Fig.* 135, *side view of a case of Pseudo-hypertrophic Paralysis, in which almost all the muscles of the body were hypertrophied.* (*After Duchenne*).

McLagan & Cumming, Lith. Edin.

In the course of time other muscles become involved, and ultimately almost all the striped muscles of the body, including the heart, may become affected. It is seldom, however, that all the muscles become hypertrophied, as represented in figs. 133, 134, 135, which are copied from Duchenne. As a rule, the enlargement is limited to a few muscles, the calves and buttocks being most frequently enlarged (see figs. 136 and 137), while others are atrophied; the latissimus dorsi

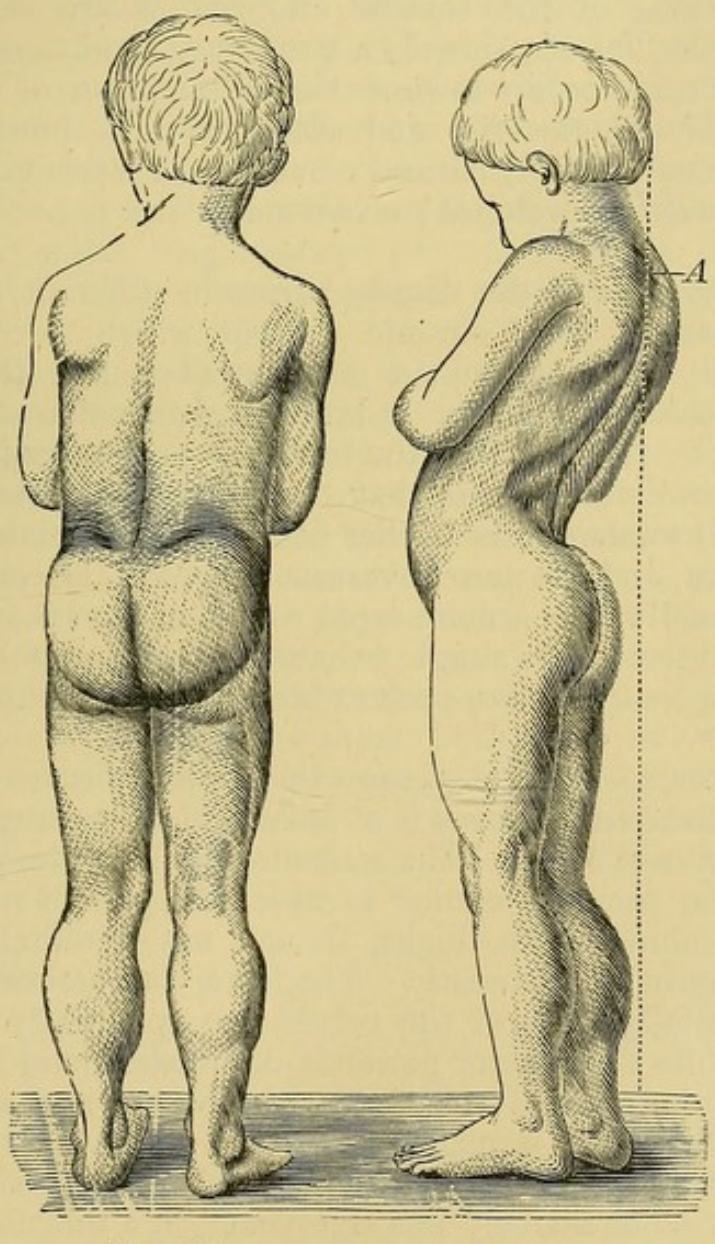


FIG. 136. FIG. 137.

Pseudo-hypertrophic paralysis, showing enlargement of the calves and buttocks. The back is curved, and a line drawn downwards from the scapulæ falls behind the sacrum. (After Duchenne.)

and the sterno-costal portion of the pectoralis major are, according to Dr Gowers, very frequently wasted, a fact which he considers of considerable diagnostic value, since it often occurs in cases which are otherwise untypical. The cirrhosis of the calf muscles usually produces a condition of talipes equinus, and the patient is unable to stand with his heels on the ground (see fig. 130).

The sensory functions of the skin are usually quite normal. The functions of the *bladder* and *rectum* are seldom interfered with, but occasionally a temporary condition of incontinence of urine occurs towards the termination of the case. The *intellectual faculties* and other cerebral functions are in some cases perfectly natural; in others, the mental faculties are imperfectly developed; occasionally the patient is quite idiotic.

The *duration* of the disease varies in different cases, but it is generally very chronic. Cases which develop soon after birth, seem to run a more rapid course than those in which the development is later—a circumstance which is explained by the fact, that the tendency to the disease is born with the individual; and that the stronger the latent (*i.e.*, congenital) tendency, the earlier does the active manifestation of it appear, and the more severe and rapid is the course. The disease usually runs a more rapid course in boys than in girls. Death may be due to simple exhaustion, but generally results from some complication, such as bronchitis or pneumonia.

Diagnosis.—In typical cases the clinical picture is so very striking that the diagnosis is at once evident to any one who has seen a case before. The attitude and gait are pathognomonic; the peculiar manner in which the patient raises himself by climbing up his thighs, though not absolutely distinctive, is highly characteristic. The facts, that certain muscles, more especially those of the calves, are apparently increased in size, while their motor power is diminished, and that other muscles are atrophied; together with the age and sex of the patient, are of the strongest diagnostic value. Dr Gowers[1] thinks the atrophy of the latissimus and lower portion of the pectoralis major scarcely less significant, in a diagnostic point of view, than the condition of the calves.

[1] *Pseudo-hypertrophic paralysis*, page 50.

PROGNOSIS.—The prognosis is very unfavourable. Almost all cases terminate sooner or later, in death. The earlier the development, the more rapid the course. As I have previously mentioned; males are usually attacked more severely than females.

TREATMENT.—The treatment, which has been hitherto employed, has been of little avail. The general health must be carefully attended to; the patient should have plenty of fresh air and good nourishing diet, and general tonics may be given. The nutritive condition of the affected muscles must be maintained in the highest possible state of efficiency, systematic muscular exercise, and the application of the Faradic current to the muscles, are the means to be employed. Duchenne states, that he cured two cases by the interrupted current, but other observers have not been equally fortunate.

Dr Gowers points out, that when, from any cause, the patient ceases to walk, the weakness in the muscles rapidly increases; contractures and deformities must, therefore, be prevented by passive exercise, and, if necessary, by section of the tendons; mechanical appliances and supports sometimes enable the patient to move about longer than he would otherwise do, and should therefore be employed. The patient must be carefully guarded against cold and exposure, for, as in all chronic spinal diseases, the end is often due to some intercurrent pulmonary affection. Arsenic seems to have been of use in some cases (Dr Meryon), and phosphorus is, according to Dr Gowers, sometimes beneficial; but drug treatment is, as a rule, of little avail.

LESIONS OF THE CROSSED PYRAMIDAL TRACT IN THE LATERAL COLUMNS.

A. Primary system lesions { 1. Primary lateral sclerosis. 2. Amyo-trophic lateral sclerosis.

B. Secondary system lesions.—Secondary descending degenerations.

C. Indiscriminate lesions.

PRIMARY LATERAL SCLEROSIS.

SYNONYM.—Spastic or spasmodic paraplegia.

This is an extremely rare affection, which has only recently been recognised as a distinct disease.

PATHOLOGY.—The lesion is a symmetrical sclerosis of the crossed pyramidal tracts. The morbid process seems to commence in the connective tissue. The nerve tubes become compressed, strangled, and ultimately destroyed. The position of the lesion is seen in fig. 139, and in the chromolithograph drawing which faces page 48.

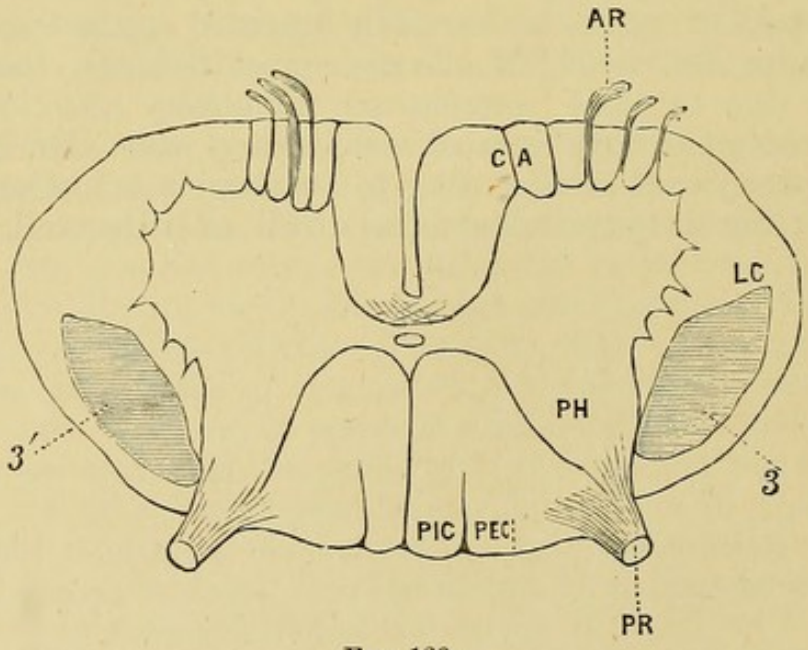


FIG. 139.

Transverse section of the cord showing a symmetrical lesion of the crossed pyramidal tracts. 3, 3′ point to the position of the lesion in each lateral column.

GENERAL CONSIDERATIONS AND ETIOLOGY.—The disease is essentially an affection of adult life, usually commencing between the ages of thirty and fifty. The male sex is more liable to the affection than the female. The causes of the condition are quite unknown; in some cases exposure to wet and cold is blamed, but whether it is actually the cause of the condition is very doubtful. The affection seems usually to attack robust muscular individuals. There is, however, a congenital form, which probably depends upon arrested or abnormal development of the motor tract, or which results from injuries to the motor tract received at the time of birth, *e.g.*, injury to the skull by the forceps, etc.

ONSET, SYMPTOMS, AND COURSE.—The affection, which is an extremely chronic one, may conveniently be divided into three stages:

First. A stage of incomplete spastic paraplegia, which usually lasts for several years, and during which the patient is able to walk about with the help of sticks.

Second. A stage of complete spastic paraplegia, which may also last for years, and during which he is confined to bed, the legs being rigidly extended.

Third. A stage of extension of the morbid process, and of complications, in which muscular atrophy, cystitis, and bed-sores may develop, and in which intercurrent attacks of pneumonia or bronchitis are apt to occur.

First Stage.—The onset is extremely slow and gradual. The first symptom is a feeling of weakness, weight, and stiffness in the lower extremities, in consequence of which the patient experiences some difficulty in walking.

Occasionally the motor weakness and stiffness is preceded by pain in the back and limbs, but these sensory disturbances are not constant or characteristic. They probably depend upon a limited meningitis.

Should the patient present himself for examination at this stage of the disease, the only abnormal condition which can be detected is slight stiffness and rigidity in the lower limbs, and increase of the deep reflexes.

The weakness and stiffness gradually increase, and the *characteristic gait* becomes fully developed; the patient walks with two sticks; each step is attended with evident effort;

the feet appear to be stuck to the ground, and can only be moved forward by raising the pelvis, and with it the limb as a whole. In this process the back is strongly arched, the chest thrown forward, the patient leans forcibly, first on one stick and then on the other, and appears to aid the elevation of the trunk by movements of his arms. The toes are dragged along the ground with an unpleasant scraping noise, the knees are apt to interlock, and the foot which is being brought forward tends to cross in front of its fellow. In some cases, after the foot leaves the ground, a peculiar hopping movement of the whole body is observed. It is due, according to Erb, to spasmodic contraction of the calf muscles.

Rigidity, spasmodic twitchings, and tremors, which may arise spontaneously, but which are generally due to some external (reflex) irritation, or to attempts at voluntary movement, affect the muscles of the lower extremities. Should the patient press upon the balls of the toes, when in the sitting or even in the standing position, a rhythmical tremor (the ankle clonus) is sometimes spontaneously set up.

The condition of the affected muscles.—The affected muscles, which are tense and rigid, especially when manipulated, present no trace of atrophy, and their electrical irritability presents no distinctive alterations; some writers state that there is a 'simple increase' to both forms of current; but in most cases the electrical reactions are normal, or there is a simple decrease.

The *deep* reflexes, are markedly exaggerated, the *knee-jerk* being very marked, and the *ankle clonus* readily obtained; a knee clonus may sometimes be elicited; and not unfrequently a blow upon one tendon produces a jerk of the opposite leg (radiation of the reflex). Any manipulation or external irritation throws the whole limb into a condition of tonic spasm. The *superficial* reflexes are sometimes exaggerated, sometimes normal, sometimes diminished or even abolished.

I formerly supposed that the diminution of the superficial reflexes, which is seen in some cases of spastic paraplegia was only apparent, and that movements of the leg did not result from tickling the sole, because the whole limb was thrown into a state of tonic extension by the reflex irritation. I still think that this explanation applies to some cases, but the examination of a well-marked case of secondary spastic paralysis, which I have recently had under observation, has shown me that the superficial reflexes may actually be absent, while the deep reflexes

are markedly exaggerated. This fact can only be explained in one of two ways, either by supposing that the two sets of reflex impulses enter the cord by different channels, and that there is a break in the superficial, but not in the deep reflex arc; or, that the so-called deep reflexes are not true reflexes. If cases do occur in which the deep reflexes are exaggerated, the superficial reflexes diminished or abolished, and *the sensory functions normal;* if, in other words, it is possible for a lesion *confined to the pyramidal tracts* to produce exaggeration of the deep, without causing exaggeration of the superficial reflex movements, we must, I think, conclude that the two phenomena are essentially different. Such a condition of matters would, to my mind, be a strong argument in favour of Dr Gowers' view that the so-called deep reflexes are compound phenomena, and that the blow upon the tendon produces direct stimulation of the muscular fibres, the irritability of which has previously been *reflexly* increased by passive tension of the muscle.

There are no objective sensory disturbances, though increased susceptibility to cold is commonly observed. The bladder and rectum are usually quite normal; there are no cerebral complications; the digestive and other organs are healthy.

Occasionally, according to Erb, the lesion may affect one leg only; or, it may involve one leg and one arm. It is then apt to be confounded with cerebral hemiplegia.

Second Stage.—This state of matters lasts for a considerable time, it may be for several years, but the stiffness and weakness gradually become greater, and the second stage of the affection is at last reached; the power of locomotion is now destroyed; the patient lies in bed with his legs stiffly extended, the thighs closely approximated as the result of spasm of the adductor muscles, and the feet inverted. Ultimately the upper extremities may become affected in exactly the same manner as the lower ones.

Third Stage.—After the second stage has continued for some time — generally for some years—the morbid process may extend to the anterior cornu, or to the postero-external column. Should the anterior cornu become invaded, *muscular atrophy* gradually invades the affected muscles, the rigidity and stiffness slowly decrease, the reflexes diminish, and ultimately become abolished. When the postero-external column is invaded, *lightning-like pains* and *inco-ordination* are superadded. In some cases cystitis or bed-sores develop, and the patient dies from gradual exhaustion or from pyœmia; in many cases he is carried off during the second or third stage of the affection by some accidental complication.

The duration of the case is extremely long, ten, fifteen, twenty, or even more years.[1] The affection does not of itself seem to destroy life; death generally results from some intercurrent affection, such as pneumonia or bronchitis.

DIAGNOSIS.—The rigidity and increase of the reflexes (positive symptoms), together with the facts, that the muscles are well nourished, and that there is no derangement of sensibility, nor of the bladder and rectum (negative symptoms), show most clearly that the lesion is limited to the pyramidal tracts.

Having arrived at this conclusion, we have next to determine whether the 'lateral sclerosis' is primary or secondary. It is important to remember:—that primary lateral sclerosis is an extremely rare affection; that exactly the same motor symptoms (weakness, spastic paraplegia, and increase of the reflexes) result from the comparatively common condition, secondary descending degeneration; that we are never justified in diagnosing a *primary* lateral sclerosis unless the history shows an extremely slow and chronic onset; and unless we can satisfy ourselves that there is no primary lesion present which could give rise to secondary descending degeneration. The conditions which are most likely to be mistaken for primary lateral sclerosis are: (1) a chronic transverse myelitis, which has produced secondary descending degeneration of the pyramidal tracts: (2) any lesion which produces slow compression of the cord, and which causes secondary descending degeneration. In making the diagnosis, special attention must be directed to the condition of sensibility, and to the state of the vertebral column.

It would of course be impossible to distinguish primary lateral sclerosis from a chronic indiscriminate lesion, such as cerebro-spinal sclerosis, which chanced to be limited to the pyramidal tracts; but in practice such difficulties are not likely to occur. The future course of the case, and the development of fresh and characteristic symptoms, would be the only means of arriving at a correct conclusion in such cases.

The differential diagnosis of primary and secondary sclerosis of the pyramidal tracts in the lateral column is given in the table on page 218.

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[1] In a case which came under my observation in the year 1877, and which I have recorded in the *Medical Times and Gazette*, Dec. 6, 1879, p. 633, the affection had commenced three years previously. It is still *in statu quo*.

PROGNOSIS.—The prognosis as regards life is good; uncomplicated cases run a very chronic course, for the condition does not of itself tend to produce a fatal termination.

The prognosis as regards recovery is unfavourable; but Erb thinks that the prognosis 'as regards recovery is at least more hopeful than in the other forms of chronic myelitis.'[1]

TREATMENT.—Attention must be directed to the state of the general health; the patient should spend as much of his time as possible out of doors; all reflex irritation is to be avoided, and a minimum of active (walking) exercise allowed. The constant current should be perseveringly passed through the spinal cord; but no benefit, in fact rather the reverse, is to be obtained by local electrical treatment of the affected muscles. The spasmodic rigidity is relieved by warm baths, but I doubt whether the ultimate result is beneficial. Erb speaks strongly in favour of *a reasonably-conducted* cold-water cure; gaseous thermal springs may also, he says, be used; but simple thermal waters, unless most carefully managed, are objectionable. Where there is a distinct history of syphilis, a prolonged course of iodide of potassium and mercury should be tried. In other cases, nitrate of silver, ergot, and iodide of potassium should be persevered with; but the results hitherto obtained by drug treatment are not encouraging. In cases which resist the treatment indicated above, I should be disposed to apply the actual cautery as a counter-irritant.

SECONDARY LATERAL SCLEROSIS.

(SECONDARY DEGENERATION OF THE CROSSED PYRAMIDAL TRACT IN THE LATERAL COLUMN).

As I have previously explained (see § 16) secondary degeneration of the crossed pyramidal tract results from any lesion which separates its component fibres from their trophic centres—the multipolar nerve cells of the cerebral cortex. The sclerosis of the crossed pyramidal tracts is attended with spastic symptoms, rigidity of the muscles, and increase of the reflexes; and, when the condition is bilateral, spastic paraplegia will, of course, be present; in cases which depend

[1] *Ziemssen's Cyclopædia*, vol. xiii. p. 645.

upon a chronic lesion, and in which the onset is slow and gradual, the symptoms very closely resemble those of *primary lateral sclerosis.* The points of distinction between the two conditions are shown in the following table:

The differential diagnosis of primary lateral sclerosis, and of spastic paraplegia due to a chronic transverse lesion (chronic transverse myelitis or slow compression of the cord).

The symptoms common to both conditions are (*a*) motor weakness in the lower extremities; (*b*) rigidity and spasms; and (*c*) increase of the deep reflexes.

	Primary Lateral Sclerosis.	Secondary Lateral Sclerosis after a Chronic Transverse Lesion of the Cord.
Condition of Muscles.	Motor weakness and rigidity are developed together. The rigidity is usually more marked than the paralysis.	Motor weakness is the first symptom. The rigidity follows the paraplegia. In the earlier stages the paralysis is more marked than the rigidity.
	There is no muscular atrophy at the upper level of the paralysis.	There may be muscular atrophy at the upper level of the lesion.
Onset.	Very slow and gradual.	Usually more rapid.
Sensory Functions.	No sensory disturbances.	More or less or even complete anæsthesia. In cases of slow compression, subjective sensations (shooting pains, etc.) due to pressure on the posterior nerve roots, are present.
The Condition of the Bladder and Rectum.	Normal.	The bladder and rectum are usually affected.
Trophic Condition of the Skin.	No trophic disturbances of the skin.	Trophic disturbances of the skin sometimes occur.

Secondary degeneration of the crossed pyramidal tract on one side may be due either to a cerebral or spinal lesion; in both cases a condition of spastic hemiplegia may be present, but the differential diagnosis does not present much difficulty. The chief points of distinction are given in the following table:—

The differential diagnosis of unilateral sclerosis of the crossed pyramidal tract due to a spinal, and to a cerebral lesion, respectively.

	Spinal Lesion.	Cerebral Lesion.
The Distribution of the Paralysis, and Rigidity.	In spinal cases the rigidity and loss of motor power are greater in, and are usually confined to, the lower extremity. An exception occurs in amyotrophic lateral sclerosis, but the atrophy in that condition is distinctive.	In cerebral cases the rigidity and loss of motor power are greater in the upper extremity.
	There is no paralysis of the face or tongue.	The face and tongue are usually affected in the earlier stages.
The Condition of the Sensory Functions.	In spinal cases there are no sensory disturbances in those in which the lesion is *confined* to the crossed pyramidal tract. Where the whole half segment is affected, there is anæsthesia on the opposite side to the paralysis.	Sensory disturbances are usually slight. The anæsthesia, if any, is on the same side as the paralysis.
The Condition of the Reflexes.	Both forms of reflex are usually increased.	The superficial reflexes are diminished or abolished, the deep reflexes exaggerated.
The History of the Case.	In an acute unilateral myelitis, the onset of the paralysis is abrupt; but in chronic cases the paralysis and rigidity are developed gradually.	In cerebral cases, the onset of the paralysis is sudden, and usually follows an apoplectic fit (hæmorrhage or embolism); the rigidity occurs later.
The Presence of other Cerebral Symptoms.	No cerebral symptoms.	Other cerebral symptoms usually present.

Treatment.—The treatment of secondary sclerosis of the crossed pyramidal tract is the same as that of the primary condition. (See page 217.)

THE INDISCRIMINATE LESIONS OF THE CROSSED PYRAMIDAL TRACTS.

The indiscriminate lesions of the cord will be afterwards described.

Combined system lesion of the crossed pyramidal tracts in the lateral column, and of the anterior cornua.

AMYOTROPHIC LATERAL SCLEROSIS.

Under the term *sclérose latérale amyotrophique*, Professor Charcot was the first to describe an affection of the spinal cord, the pathological substratum of which is a combined sclerosis of the pyramidal tracts, and of the anterior cornua. The lesion is not, however, confined to the spinal cord, for it can be followed upwards through the medulla oblongata, and sometimes through the foot of the cerebral peduncle; the internal capsule is usually intact. On microscopical examination the lesion of the anterior cornua seems to be identical with that of progressive muscular atrophy. The nuclei of the facial, hypo-glossal, and spinal accessory nerves are

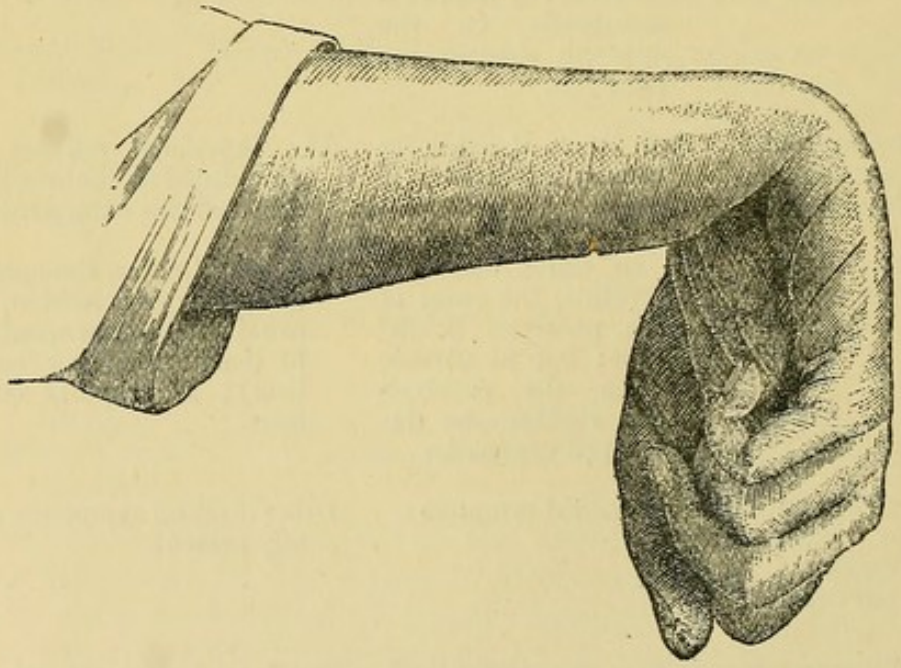


FIG. 140.

Position of the hand in Amyotrophic Lateral Sclerosis. (After Charcot.)

generally involved towards the termination of the case. The cervical enlargement is the part of the cord which is first and most affected.

The *symptoms* which characterise the condition are:—loss of motor power and muscular atrophy, together with rigidity and tension of the muscles. Since the morbid process commences in the cervical enlargement, the muscles of the upper limbs are first attacked. Charcot describes three stages of the affection.

In the *first*, which usually lasts from four to twelve months, the symptoms are confined to the upper extremities, and consist of motor weakness, which is soon followed by *diffuse* atrophy, and by rigidity and contractures. Fibrillary twitchings can usually be observed in the affected muscles. As the result of the muscular tension and contractures, deformities are produced, and the hand often becomes fixed in the position shown in fig. 140.

In the *second* stage the lower extremities are invaded, the muscles become rigid, the tendon reflexes are increased, and for a time the condition of the lower limbs is identical with that which results from primary sclerosis of the lateral columns, viz., spastic paraplegia, together with the absence of any marked sensory disturbances, and of any affection of the bladder or rectum. After a time the morbid process extends to the anterior cornua of the lumbar enlargement, the muscles of the lower extremities atrophy; the increase of the reflexes gradually diminishes, and the rigidity and spasms gradually decrease. During the second stage the atrophy in the upper extremities becomes greater.

In the *third* stage all the symptoms are exaggerated; the morbid process extends to the medulla oblongata; bulbar symptoms are developed, and death results.

In exceptional cases the morbid process commences in the medulla oblongata and extends downwards; occasionally the lower extremities are first affected, and the morbid process extends from below upwards.

The disease, so far as is known, is invariably fatal. The duration varies in different cases, but death usually results in from one to three years. The affection seems more common in females than in males. It attacks persons between the ages of twenty and fifty. The causes of the condition are unknown.

Diagnosis.—The mode of development, character of the symptoms, and course at once distinguish the affection from all other diseases of the spinal cord.

The only affections with which it is at all likely to be confounded are progressive muscular atrophy, primary lateral sclerosis, and pachymeningitis cervicalis hypertrophica; the points of differential distinction between amyotrophic lateral sclerosis and these three affections are given in the table on p. 277.

The *prognosis* is most unfavourable; *treatment* has, up to the present time, been totally inadequate to check the progress of the affection.

DISEASES WHICH RESULT FROM LESIONS OF THE POSTERIOR COLUMNS.

A. Primary system lesion of the postero-external columns.—Locomotor ataxia.

B. Secondary system lesion of the postero-internal columns.—Secondary ascending degeneration.

C. Indiscriminate lesions.

LOCOMOTOR ATAXIA.

Synonyms.—Progressive locomotor ataxia; tabes dorsalis; sclerosis of the posterior columns.

Locomotor ataxia is an extremely chronic spinal affection, which is characterised in its fully developed stage by difficulty in walking, not from loss of motor power, but from incoordination of the muscles of the lower extremities.

ETIOLOGY.—The disease is much more frequent in males than in females, and occurs between the ages of twenty and fifty.[1] Friedreich and others have described

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[1] Of 149 cases collected by Eulenburg, 128 were males and 21 females. The ages at which the disease occurred in these cases were as follows:—

	Males.	Females.
From 0 to 10 years	0	1
„ 10 „ 20 „	2	0
„ 20 „ 30 „	35	12
„ 30 „ 40 „	39	7
„ 40 „ 50 „	47	1
„ 50 „ 60 „	5	0
After 60 „	0	0
	128	21

a congenital variety, which is at least as common in girls as in boys. In the greater number of acquired (non-congenital) cases, there is a history of syphilis; but, as I have previously remarked (see p. 78), it is extremely doubtful whether syphilis is the sole cause of the affection. Anything which exhausts or unduly excites the functional activity of the nerve tubes composing the postero-external columns, in persons predisposed to the affection, will probably act as an exciting cause of the condition. Blows on the spine, and other traumatic injuries; exposure to cold and wet; sexual excesses; and acute febrile attacks; which in some cases seem to be the starting point of the affection, are probably exciting causes. It is extremely doubtful whether any one of them could produce the disease in a perfectly healthy (non-predisposed) individual.

PATHOLOGY.—On naked eye examination the membranes are usually seen to be thickened and adherent over the posterior surface of the cord. The cord itself looks flattened in the antero-posterior direction, while the posterior columns and posterior nerve roots are evidently shrunken and atrophied. On section the posterior columns present a translucent grey appearance, and their consistency is increased. The lesion commences in the great majority of cases in the lower dorsal and lumbar regions, and the postero-external columns are the parts of the transverse section which are first affected. Indeed, as Pierret and Charcot have shown, the only *necessary* and *essential* lesion of locomotor ataxia is the sclerosis of the *postero-external* columns. As the morbid process advances, the whole of the posterior columns (the postero-internal as well as the postero-external) in the lower dorsal and lumbar regions become invaded; and according to Lockhart Clarke, the lesion usually involves the posterior cornu. In the cervical region the lesion is usually limited to the postero-internal columns, and presents the characteristic features of a secondary ascending degeneration; the tract of degeneration can generally be followed through the medulla oblongata. The cranial nerves, especially the optic, and its expansion in the eyeball (*i.e.*, the optic disc), are apt to undergo the same grey atrophic change. Occasionally the postero-external columns in the cervical region are also affected, and the characteristic symptoms (pains, inco-ordination, etc.) are then observed in the upper

extremities. Towards the end of the case the morbid process may extend to the region of the anterior horn, or to the lateral column.

The morbid process is probably an extremely chronic inflammation, which, commencing in the nerve elements, leads to their slow destruction, and is attended with an increase of the neuroglial connective tissue. The microscopical characters of the lesion are more minutely described in page 49, to which the reader is referred.

Mode of Onset, Symptoms, and Course.—The disease is an extremely chronic one, lasting for ten, twenty, or even thirty years. The affection may be conveniently divided into three stages.

First. The stage of invasion, which is usually characterised by lightning pains, slight disturbances of sensibility in the lower extremities, temporary paralysis of some of the ocular muscles, abolition of the patellar tendon reflex, and sometimes of the reflex contraction of the pupil to light. This stage continues for some months, occasionally it extends over several years, and quite exceptionally it lasts indefinitely, and the second stage is not reached.

Second. The stage of full development.—The inco-ordination and characteristic gait are now present; the lightning pains continue; the disturbances of sensibility are greater.

Third. The stage of complications, and of extension of the morbid process.—Complications on the part of the lungs, bladder, kidneys, etc., may now be developed, and are often the cause of death. During this stage the lesion may extend from the postero-external column to the region of the anterior cornu,[1] or to the crossed pyramidal tract in the lateral column; muscular atrophy and spastic paraplegia will then be observed.

Such is a bare enumeration of the most prominent features

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[1] I am anxiously looking for an opportunity of observing a case of locomotor ataxia in which the lesion has extended to the lateral column. Such a case ought, I think, to throw great light upon the exact character of the patellar tendon reflex. In typical cases of locomotor ataxia the deep reflexes are abolished, but in lateral sclerosis they are characteristically increased. The condition of the knee-jerk in a case of locomotor ataxia which has become complicated with lateral sclerosis is the point which I wish particularly to examine.

of the affection. I will now consider the individual symptoms a little more in detail.

Sensory disturbances.—Sclerosis of the postero-external column necessarily gives rise to some disturbance of sensibility. Lightning pains, hyperæsthesia and anæsthesia of the skin, and retarded conduction of sensation, are the sensory derangements which are most frequently met with; and since the lesion of the postero-external columns is usually confined to the dorsal and lumbar regions of the cord, these derangements of sensation are usually confined to the lower extremities or to the lower part of the trunk. When the postero-external columns in the cervical region are affected, the characteristic symptoms are observed in the upper extremities.

The lightning pains are usually the first symptoms to attract attention, and are often mistaken for rheumatism or neuralgia; they occur in paroxysms, and may be extremely severe; they are sharp and shooting in character, and of momentary duration; some patients compare them to forked lightning, others say they resemble a powerful electric shock, or the effect which would be produced by running a knife into the flesh or into a joint. Unlike the shooting pains of ordinary neuralgia, they are not referred to the skin, but to the deeper structures, and they do not radiate in the superficial area of distribution of any individual nerve, as the pains of ordinary neuralgia do; in the great majority of cases they are felt in the lower extremities or in the pelvic organs (bladder and rectum). Where the postero-external column *in the cervical* region is implicated, the lightning pains are referred to the upper extremities or to the head. Occasionally they are experienced in the region of the stomach, and are then usually associated with vomiting and symptoms of dyspepsia. (See gastric crises, page 231). They are almost invariably present in the first and second stages of the disease, and not unfrequently continue throughout its whole course. The lightning pains are sometimes so severe as to make the patient tire of life; and cases have occurred in whieh ataxics, worn out by repeated attacks of lightning pains and despairing of relief, have committed self-destruction.

Hyperæsthesia of the skin is observed in many cases; like the lancinating pains it is paroxysmal and fugitive—here to-day away to-morrow. The girdle sensation, which is, as we

have seen, the result of hyperæsthesia, is present in a considerable number of cases.

Anæsthesia.—Loss of tactile sensibility is a very common symptom. In the earlier stages of the affection it is partial or incomplete; later it may be total. Sensibility to pain and temperature is often also impaired. In consequence of the incomplete anæsthesia, various subjective alterations are experienced by the patient; in many cases, for example, he feels as if he were standing on cotton wool, a bag of feathers, etc.

Retardation of sensory conduction, especially of the conduction of painful impressions, is present in a considerable number of cases; the prick of a pin, for example, is at once felt as a tactile impulse, but a considerable interval elapses before a sensation of pain is experienced.

Derangements of the motor nerve apparatus. — Since the lesion is in the great majority of cases confined to the lower portions of the cord (the lumbar and lower dorsal regions) it follows that the motor derangements which constitute the most striking feature of the disease, are usually observed in the muscles of the lower extremities. The most prominent motor derangement is difficulty in walking. It is due to inco-ordination and not to loss of motor power; it is necessary to emphasise this fact, and to call attention to the circumstance that the muscles are well developed during the second stage of the disease, for the difficulty in walking was formerly thought to be due to paralysis, and the disease was classed with many other affections under the common term '*paraplegia.*' The difficulty in walking and standing is at first slight, and is only noticed in the dark or when the patient shuts his eyes. In many cases the unsteadiness is first observed when the patient washes his face; he finds that when he shuts his eyes to protect them from the soap, he has difficulty in maintaining his equilibrium, and tends to stagger and fall.

Should the patient come under observation at this stage of the disease, the co-ordinating and balancing powers are to be tested in the manner described on pages 132 and 133.

In the course of time the inco-ordination becomes greater, and the characteristic gait is developed.

The gait of locomotor ataxia.—The gait is very characteristic, though, as has been already mentioned, not absolutely

pathognomonic; the patient walks with sticks; he keeps his eyes fixed on his feet or on the ground in front of him; the steps are taken at regular intervals, slowly and with deliberation, but the movements of the legs are irregular; the feet are jerked outwards, the heels brought suddenly to the ground with a stamp. In uncomplicated cases he walks in a straight line. There is no giddiness, the unsteadiness being, as Duchenne long ago pointed out, not due to the head but to the legs.

The unsteadiness in walking and in gait is much aggravated by psychical causes; ataxic patients, knowing their unsteadiness, and dreading accidents, are extremely loth to move in the dark, to walk over a slippery pavement or in a crowded thoroughfare. The extreme unsteadiness which they manifest under such circumstances is partly due to timidity.

When the lesion involves the postero-external columns in the cervical region, inco-ordination of the muscles of the upper extremities will of course be observed.

In exceptional cases the lesion chiefly involves the mid-dorsal region, and the inco-ordination affects the muscles of the spine. (See page 83.)

During the stage of full development the condition of the muscles is well preserved; their electrical reactions are either normal or undergo a simple decrease, in the earlier periods a simple increase is said to occur. In fully developed cases the muscular sense is impaired, and in some cases altogether abolished.

The condition of the reflexes. — The patellar tendon reflex is generally abolished at an early stage of the affection.[1]

The condition of the *superficial reflexes* varies in different cases. In the majority, the plantar reflex is impaired at an early stage and finally abolished; but it sometimes happens that a lively reflex can be obtained by tickling the sole when

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[1] In those exceptional cases in which the knee-jerk exists, the lesion is probably situated above the lumbar enlargement. Two cases of this description have come under my own notice; in both, the inco-ordination involved the muscles of the trunk more than the muscles of the lower extremities, and was of the 'coarse' kind which I have described in speaking of cerebro-spinal sclerosis. (See page 83.)

the patellar tendon reflex is completely obliterated. (See fig. 141.)

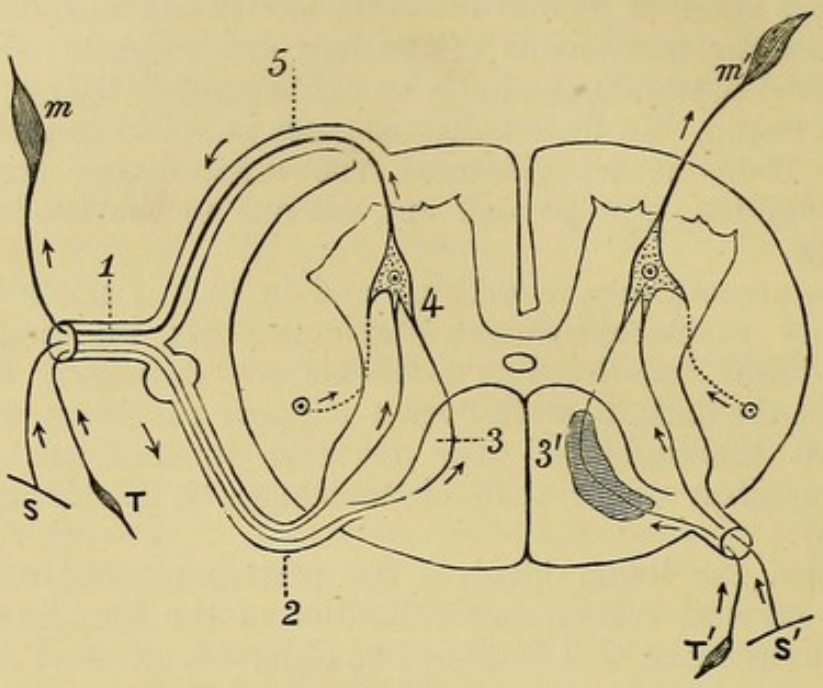


FIG. 141.

Diagrammatic representation of the reflex functions of the Spinal Segment.

The left half segment is normal. The right half represents the early stage of locomotor ataxia, the position of the lesion being shaded dark.

S, Skin from which sensory fibres pass through the common sensory motor nerve trunk, posterior root, and posterior horn of grey matter to the reflex centre (4). T, Tendon, muscle, and fascia, from which the sensory fibres pass which conduct the deep reflex movements. These fibres traverse the postero-external column.

m, Muscle supplied by the anterior root of the left half segment. The dotted line passing from the crossed pyramidal tract in the lateral column represents the inhibitory fibre.

The arrows show the course of the reflex impulse.

On the right side the deep reflex movements are seen to be arrested by a lesion in the postero-external column.

The pupil reflex to light is abolished in a large proportion of cases; and it is important to remember that in these cases the contractility of the iris on efforts of accommodation for near objects, *i.e.* on convergence, is still retained. This condition was first described by Dr Argyll Robertson, and is sometimes called the 'Argyll Robertson phenomenon.'

The abolition of the pupil reflex is, I think, probably due to a lesion in the neighbourhood of the aqueduct of Sylvius, and is independent of the cord lesion. The contraction of the iris, which is produced by a ray of light falling upon the retina, is a reflex act;—the sensory

fibres which carry the impression inwards are the fibres of the optic nerve;—the centre is probably situated in the grey matter around the aqueduct of Sylvius;—the motor fibres which carry the impression outwards are the fibres of the third nerve which supply the circular fibres of the iris. Now, in those cases in which the Argyll Robertson phenomenon is present, there may be no affection of vision. Hence the break in the reflex arc is not situated in the retina; nor in the optic nerve between the retina, and that point at which the fibres of the optic nerve carrying visual impressions to the cerebral cortex diverge from those which pass to the reflex centre for the pupil; while the fact that the pupil responds to efforts of accommodation seems to show that the reflex centre, and the motor nerve, *i.e.*, the third nerve, are healthy. (I am aware that some writers argue from this fact that there are two centres for the iris, one connected with the reflex action to light, the other with the semi-voluntary action of the pupil on accommodation for near objects). Hence I am of opinion that the break in the reflex arc is probably situated either in the corpora geniculata, or at a point between the corpora geniculata and the reflex centre in the grey matter of the aqueduct of Sylvius.

Derangements of the vesical and rectal mechanism are frequently met with in locomotor ataxia. In the earlier stages, symptoms of vesical irritation, such as painful micturition, 'precipitant' urination, stabbing pains in the neck of the bladder, etc., are the most frequent; in the fully developed periods, paresis of the detrusor, with consequent slow micturition and dribbling, or actual paralysis of the bladder may occur. In the third stage, cystitis is a common complication, and complete insensibility of the urethra is occasionally observed.

Constipation occurs in the great majority of cases; in some, it is due to diminished or abolished reflex action; in others, to paralysis of the gut. Stabbing pains and a peculiar, painful feeling of over-distention of the rectum are frequent during the first and second periods of the disease. Anæsthesia of the anus is occasionally associated with the anæsthesia of the urethra, to which I have just referred.

The sexual reflex.—In the earlier stages increased sexual desire (satyriasis) is not uncommon; it is usually combined with a condition of 'irritable weakness.' In fully developed cases the sexual appetite is usually very much impaired, or completely abolished, and in the later periods there may be absolute impotence.

The eye symptoms of locomotor ataxia.—Derangements of the visual apparatus are frequently met with; the more important are:—

1. *Temporary paralysis* of the levator palpebræ superioris, giving rise to ptosis; or, of one or other of the straight muscles of the eyeball, producing diplopia and (sometimes) squint. These conditions are of common occurrence in the earlier periods of the case, and are of considerable diagnostic significance.

2. *Loss of the pupil reflex to light.*

3. *Myosis*—contraction of the pupil. This condition occurs in a considerable number of cases; in some the contraction is extreme, and the pupils are 'pin-pointed.' Moderate contraction of the pupils is probably due to a destructive lesion of the sympathetic fibres in the cilio-spinal region of the cord; but extreme contraction—a condition which is not uncommon in the later stages of the disease—can hardly be due to simple paralysis of the radiating fibres, especially when we remember, that in many of these cases, the reflex contraction of the pupil to light is interfered with. Extreme contraction probably results from an irritation of that portion of the third nerve nucleus from which the fibres which supply the circular fibres of the iris proceed.

4. *Inequality in the size of the two pupils.*—This condition is sometimes met with; in some cases it results from derangement of innervation; in others it is due to iritic adhesions, which have in all probability resulted from previous syphilis.

5. *Optic atrophy—dimness of vision—loss of the perception of colours.*—Grey atrophy of the optic nerve occurs in a considerable number of cases. The perception of colours is interfered with; green is first lost, then red, then yellow, and last of all blue. The visual field is diminished, and finally complete blindness may be established. The characteristic atrophy of the discs can of course be seen with the ophthalmoscope; and it is important from a diagnostic point of view to remember that this symptom may occur at an early stage of the disease, indeed, in some cases it is the first symptom.

Trophic alterations in the skin are occasionally, though not very frequently, observed. Herpetic eruptions are the most common; the vesicles occur in successive crops, and in some cases a distinct relationship can be traced between the crops of herpes, and attack of lightning pains. Towards the terminal period of the disease, bed-sores sometimes form, and tend to hasten the fatal termination.

Trophic alterations in the bones and joints.—Professor Charcot was the first to direct attention to the peculiar disease of the bones and joints which occurs in a small proportion of cases of the disease. The condition seems to be more common in women than in men; the joint tissues become rapidly disorganised; the heads of the bones are absorbed; and marked deformities may be produced. These alterations, which are well seen in figs. 142, 143, and 144 (copied

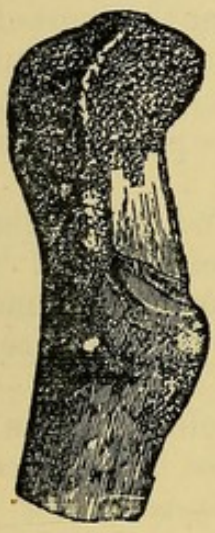


Fig. 142.

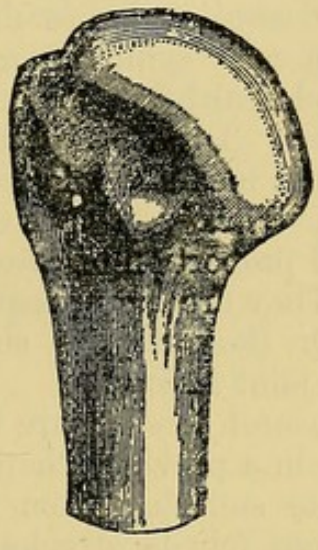


Fig. 143.

Fig. 142.—*The head of a femur from a case of Locomotor Ataxia affected with Charcot's joint lesion.*

Fig. 143.—*Healthy femur for comparison.* (*After Charcot.*)

from Charcot), may give rise to marked external deformities (see fig. 145). A remarkable feature of the joint lesion is the fact, that it is attended by little or no pain,—a circumstance which is probably due to the associated analgesia. The larger joints, such as the knee, shoulder, and elbow, are most liable to be affected. The pathological alterations are very similar to those met with in chronic rheumatic arthritis; but the two conditions differ in the facts; (1.) that one is acute and the other very chronic; (2.) that in chronic rheumatic arthritis the smaller joints and the hip are most frequently affected, whereas in locomotor ataxia the knee and shoulder are most often involved; and (3.) that,

in the joint affection of locomotor ataxia, there is usually a considerable amount of liquid effusion, and dislocations are very apt to occur; but in chronic rheumatic arthritis effusion is seldom seen, and dislocations are very rare. The affected bones become more friable, and are apt to fracture spontaneously, which condition, like the joint affection, is unattended by pain. The exact pathological cause of the joint lesion is not yet decided. Some authorities believe that it is due to an affection of the anterior cornu of the spinal cord, —a view which seems to be contradicted by the facts, that there is no associated muscular atrophy, that the joint affection may be an early symptom, and that the anterior cornua have been found healthy in some cases which have been examined *post-mortem*. Dr Buzzard thinks that it is probably due to a lesion of the medulla oblongata.

Gastric crises.—Attacks of vomiting and gastralgia occur in a small proportion of cases, and have been termed 'gastric crises.' They are more frequent in women than in men, and are, as Dr Buzzard has shown, apt to be associated with Charcot's joint lesion.

The *mental faculties* are usually quite clear, but, as I have remarked in a previous chapter, this disease is not uncommon in patients suffering from *general paralysis of the insane*. Intercurrent febrile attacks occasionally occur, and repeated attacks of bronchitis are sometimes, though rarely, observed.

The affection is very chronic; cases frequently last ten, fifteen, or twenty years; periods of temporary improvement are not uncommon, but the termination is usually in death; the fatal termination is generally due to some intercurrent pulmonary affection; to cystitis and surgical kidney; or the fatal issue is accelerated by the formation of a bed-sore.

DIAGNOSIS.—*In the earlier stages, i.e.*, before the incoordination and characteristic gait are developed, the pains are often put down to *rheumatism* or *neuralgia*. The characters of the pains (see page 225) are, however, quite peculiar, and, to a well-informed observer, at once suggest a lesion of the posterior columns. When optic atrophy is an early symptom, its true character may in such cases be easily overlooked; locomotor ataxia should always be suspected when dimness of vision and optic atrophy are established without any obvious cause; and the other early symptoms, especially lightning

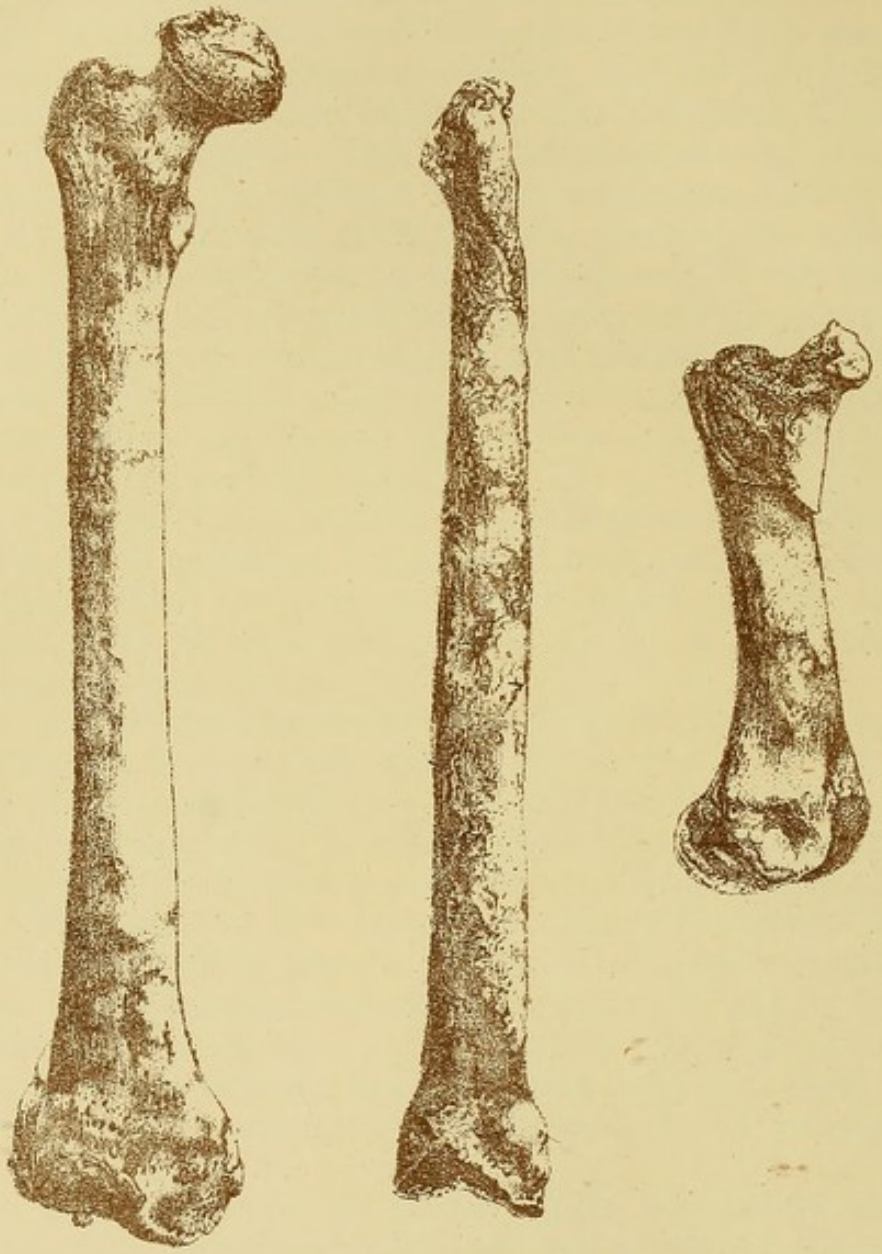


FIG. 144.

Spontaneous fractures of the femur, and the joint lesions of Locomotor Ataxia.
(*After Charcot*).

A normal femur is represented on the left of the figure, for the purposes of comparison.

M'Lagan & Cumming, Lith. Edin'.

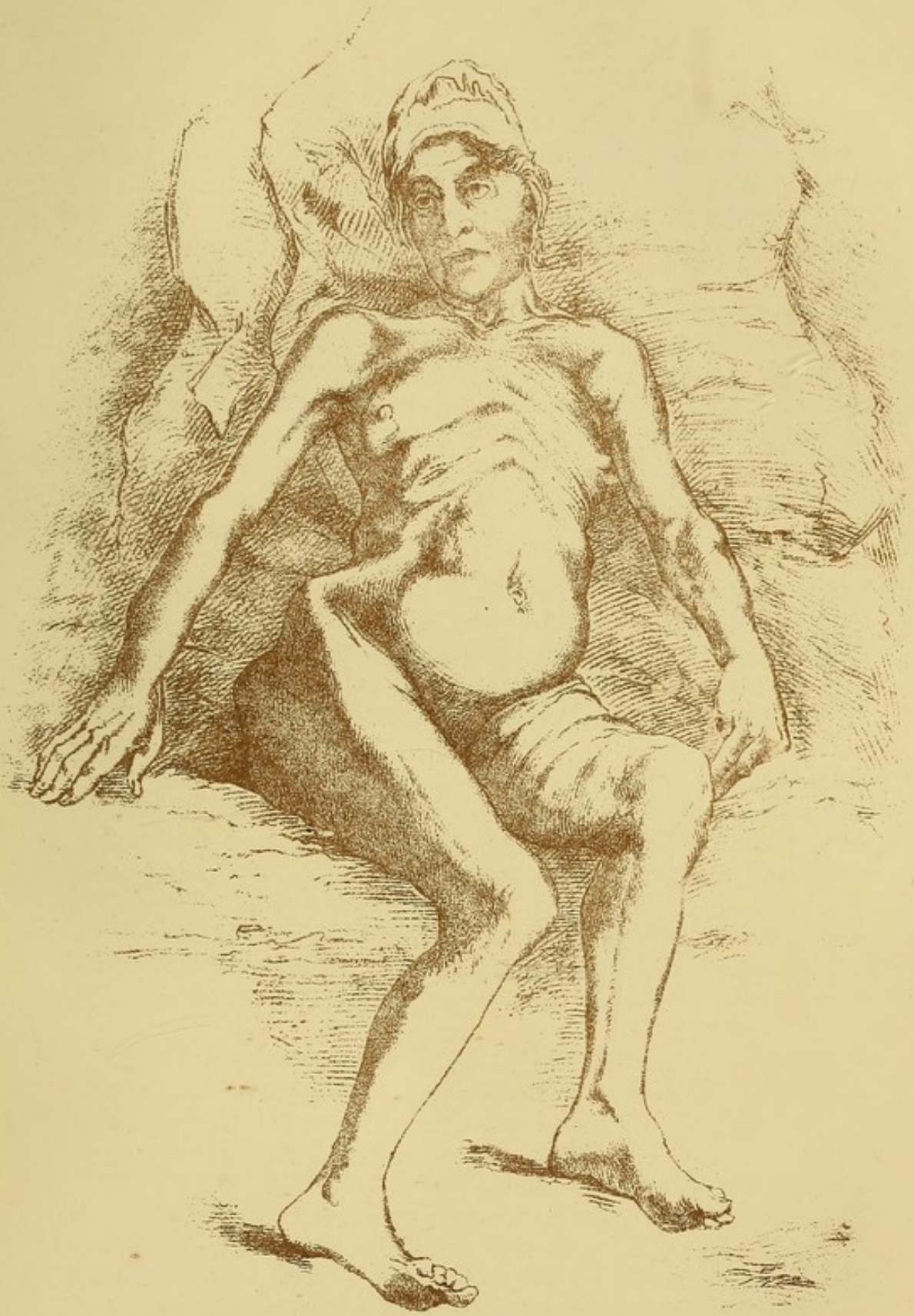


FIG. 145.

Patient affected with the joint lesion of Locomotor Ataxia. (After Charcot).

McLagan & Cumming, Lith. Edin.

pains, abolished patellar tendon reflex, the Argyll Robertson phenomenon, and commencing inco-ordination should be carefully enquired after. The stomach attacks (*crises gastriques*) may also occur before the inco-ordination is developed, and in these cases the condition is often put down to simple dyspepsia.

When the *second stage* is reached the diagnosis should present little difficulty. The facts, that the gross motor power is preserved, and that the difficulty in walking depends upon inco-ordination, at once distinguishes the case from the paralytic affections with which it was at one time confounded. Disease of the cerebellum and cerebro-spinal sclerosis are the two affections which are most liable to be mistaken for locomotor ataxia.

The differential diagnosis of locomotor ataxia and cerebellar disease.—In cases of cerebellar disease:—(1.) The gait is different; the patient reels like a drunken man, and complains of feeling giddy; or, he walks with that form of gait which depends on weakness or inco-ordination of the lumbar muscles.

2. Then there are the positive symptoms of cerebellar disease, such as vomiting, headache, double optic neuritis. Hemiplegia is present in some cases of cerebellar disease; and tonic convulsive attacks, in which the muscles of the spine are chiefly affected, may occur.

3. The characteristic symptoms of locomotor ataxia (lightning pains, abolished knee-jerk, Argyll Robertson phenomenon, and sensory derangements) are wanting.

The differential diagnosis of typical cases of cerebro-spinal sclerosis and of locomotor ataxia presents no difficulty, and is made by attention to the following points:—

1. The gait in the two cases is different.

2. In locomotor ataxia the mental functions are usually quite clear; there is no affection of speech; vertigo and headache are uncommon. In cerebro-spinal sclerosis there is usually some dulling of the mental faculties; vertigo and headache are frequent; and alteration in speech common.

3. The rhythmical tremor on voluntary movement, which is such a characteristic feature of cerebro-spinal sclerosis, does not occur in locomotor ataxia.

4. The lightning pains are not present in cerebro-spinal sclerosis, unless the postero-external columns are invaded.

5. The knee-jerk is (usually) exaggerated in cerebro-spinal sclerosis, though it may of course be abolished when the lesion involves the postero-external column in the lumbar region.

6. The Argyll Robertson phenomenon does (?) not occur in cerebro-spinal sclerosis.[1]

But while there is no great difficulty in making a diagnosis in typical cases, there are other cases in which the distinction is very difficult, or even impossible. In cases of locomotor ataxia, for instance, in which the lesion is chiefly situated in the dorsal region, and in which the lumbar enlargement escapes, the inco-ordination involves the spinal muscles rather than those of the lower extremities. The gait differs from the typical gait, which depends upon sclerosis of the postero-internal columns in the lumbar region. Again, in those cases in which the lesion is confined to the dorsal region, the knee-jerk may be exaggerated. The differential diagnosis in such cases must be determined by the facts placed under 2, 3, 4, and 6 in the previous paragraph, together with the history, general course, and development of the two affections. Again, the lesion in the early stage of cerebro-spinal sclerosis might be limited to the postero-external columns in the lower dorsal and lumbar regions, and the symptoms be identical with those of locomotor ataxia; the differential diagnosis could then only be made by attention to the general features of the case, and by observing the future course of events.

Prognosis.—The great majority of cases of locomotor ataxia ultimately lead to death; remissions in the severity of

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[1] The query in the text shows that a decided opinion on this point cannot as yet be given. M. Vincent, quoted by Robin, states that in cases of cerebro-spinal sclerosis in which there is myosis, the pupil contracts both to light and to efforts of accommodation; while M. Coingt, also quoted by the same writer, says that in two cases which he examined, the pupil acted feebly to light, but energetically on accommodation for near objects.—*Les troubles Oculaires dans les maladies de l'encéphale, page* 213.

In a very typical case of the disease (cerebro-spinal sclerosis) which I have at present under observation, there is marked myosis, but the reaction of the pupil both to light and accommodation is energetic.

the symptoms are not uncommon; complete intermissions occasionally occur; and in a small number of cases a cure is established. Cases with a strong syphilitic history, which come under treatment in an early stage, are probably sometimes cured by anti-syphilitic treatment, but such a happy event is, I think, rarely to be expected.

TREATMENT.—In the earlier stages of all cases a vigorous anti-syphilitic treatment should be adopted; ergot is strongly recommended by some authorities. Where these means fail, the galvanic treatment[1] should be commenced, and nitrate of silver ($\frac{1}{4}$ to $\frac{1}{2}$ a grain, three times a day), administered. The drug must be given for some months, not continuously, but with occasional intermissions, so as to avoid the risk of 'staining.' The actual cautery may also be applied on each side of the spinal column, at a point corresponding to the seat of the lesion; some authorities speak very highly of this form of counter-irritation, others have not found it give relief.

Baths are strongly recommended by some German observers, and Rosenthal[2] speaks very highly of hydrotherapeutics. Exposure and over-fatigue of all kinds must be avoided. The general health is to be carefully attended to, in accordance with the general principles already laid down (see p. 166). Where there is a tendency to emaciation cod-liver oil is often highly beneficial.

The *lightning pains* are best relieved by subcutaneous injections of morphia; bromide of potassium in large doses, and the constant (galvanic) current, in some cases give relief. Althaus speaks highly of the salicylate of soda in twenty grain doses.[3] Stretching the sciatic or other large nerve trunk seems to relieve the pains, but it does not appear to produce any beneficial effect on the general course of the disease, and it is not altogether free from danger. A decided opinion, either for or against this plan of treatment, is hardly, I think, as yet warranted by facts.

The *gastric attacks* are probably best treated by morphia; but bismuth, and saccharated pepsin, in doses of fifteen to

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[1] For the manner of applying the galvanic current (see p. 167).

[2] *Diseases of the Nervous System*, p. 259.

[3] *British Medical Journal*, Nov. 9, 1878, p. 687.

twenty grains with each meal, are strongly recommended by Hammond.[1]

The *optic atrophy* is uninfluenced by treatment; strychnine, which seems useful in some cases of optic atrophy arising from other causes, does not appear to produce any beneficial effect on the grey atrophy of locomotor ataxia, while it is hurtful rather than otherwise so far as the general condition is concerned.

The derangements of the bladder and rectum, bed-sores and other complications, must be treated on general medical principles.

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[1] *Diseases of the Nervous System*, p. 633.

INDISCRIMINATE LESIONS.

Myelitis { Acute. / Chronic.
Landry's Paralysis.
Disseminated Sclerosis.
Intra-medullary Hæmorrhage.
Intra-medullary Tumours.

MYELITIS.

Inflammation of the spinal cord may be either acute or chronic; and it is necessary to consider the two varieties separately.

ACUTE MYELITIS (*Acute inflammation of the spinal cord.*)

Pathology and morbid anatomy.—Under the term acute myelitis are included all the indiscriminate lesions of the cord which are attended with fever, and which present the pathological features which I have described on page 52, and to which the reader is again referred.[1]

The extent of cord which happens to be involved varies greatly in different cases, and several distinct varieties of the disease are accordingly described by systematic writers. The more important of these forms are:

1. *Acute general myelitis,* in which a large extent of the cord is affected by the inflammatory process.
2. *Acute central myelitis,* in which the central grey matter is chiefly affected.
3. *Acute transverse myelitis,* in which the whole transverse section is affected, but in which the vertical extent of the lesion is small. The lumbar, dorsal, and cervical regions are all liable to be invaded in this manner.
4. *Acute unilateral myelitis,* in which half, of one or more segments in the lumbar, dorsal, or cervical regions, is implicated.
5. *Acute disseminated myelitis,* in which numerous distinct foci of inflammation are scattered throughout the cord This condition is very generally syphilitic.

[1] Polio-myelitis anterior acuta is an acute inflammation of the cord, but it is a system lesion; and it is not therefore included under *acute myelitis*, but has been previously considered.

6. *Acute bulbar myelitis,* in which the medulla oblongata is implicated by the lesion.

In addition to this division, which is based upon the position and extent of the inflammatory lesion, another classification which is sometimes useful is the etiological; it attempts to classify the inflammatory process in accordance with its cause. Thus we have an acute *traumatic* myelitis, an acute *syphilitic* myelitis, an acute *idiopathic* myelitis, and so on.

ETIOLOGY.—In many cases the exact cause of the attack is obscure. The condition seems more common in youth and early adult life than in children or old people.[1]

Predisposing causes.—Anything which debilitates the system as a whole, and the cord in particular, seems to act as a predisposing cause. Sexual excess, and excessive muscular exercise, probably act as predisposing causes; though I have known excessive muscular exertion act as an exciting cause, and reproduce a myelitis which had been recently recovered from.

Exciting causes.—The chief exciting causes are:—exposure to cold and wet; traumatic injuries, such as blows on the back, concussions of the spine; extension of the inflammatory process from adjacent and distant parts, especially from the (adjacent) membranes, or from the (distant) urinary organs; acute inflammatory affections, such as diphtheria, small-pox or typhoid;[2] syphilis;[3] excessive bodily exercise, though this is rare; and compression of the cord by diseased bones, tumours, or thickened membranes.

SYMPTOMS.—The symptoms vary very much in different cases, the differences being readily accounted for by the different degrees of acuteness, and more especially by the extreme differences in the position and distribution of the lesion which are met with in different cases. I do not propose to describe in detail each of these different types; provided a good general grasp of the symptoms, which the inflammatory process produces, has been obtained, it will be easy to fill in the special features, which result from inflam-

[1] The system disease (*polio-myelitis anterior acuta*) is, as has been already pointed out, very much more common in children than in adults; but that affection has been expressly excluded from the present section.

[2] In these cases the inflammatory process is usually sub-acute.

[3] In these cases the myelitis is generally sub-acute or chronic.

mation of the different parts of the cord, by referring to the detailed description which has been already given of the symptoms produced by lesions of the different physiological regions and tracts (see Chapter II.).

In studying the symptoms of acute myelitis it is essential to remember:

(1) That the inflammatory process first produces irritation and subsequently destruction of the affected part. (2) That the stage of irritation is short, and that the symptoms, which result from destruction of tissue, are therefore rapidly developed. (3) That as soon as the acute stage passes off, chronic changes are usually developed; and (4) That in many cases (*i.e.*, where the inflammatory process destroys the continuity of the pyramidal tracts), a secondary descending degeneration of the pyramidal tracts, below the lesion, is established.

We may then conveniently divide the process into (1) a premonitory stage; (2) a stage of irritation; (3) a stage of destruction; and (4) a stage of cicatrisation and secondary degenerations.

It is also important to remember, that in some cases the inflammatory process affects the membranes of the cord, and that symptoms of meningitis are present in addition to those which result from inflammation of the cord itself.

The Premonitory Stage.—In some cases, premonitory symptoms, chiefly consisting of derangements of sensation, such as a feeling of numbness, or tingling, or of slight pain in the limbs or back; and of mild febrile disturbance and general malaise, are observed. In others, the attack commences with a chill, which is followed by rapid elevation of temperature. In others again, premonitory symptoms are entirely wanting, and the spinal symptoms appear at the commencement of the case; occasionally the spinal symptoms (paralysis, anæsthesia, etc.) are developed so rapidly as to suggest an intra-medullary hæmorrhage.[1]

The Stage of Irritation.—The symptoms which characterise this stage are both sensory and motor. The sensory symp-

[1] The onset in acute myelitis is never *instantaneous*, as it may be in an intra-medullary hæmorrhage. Again, an intra-medullary hæmorrhage is not preceded by fever as an acute myelitis may be. In distinguishing the two conditions (myelitis and intra-medullary hæmorrhage) *post-mortem*, it is important to remember that in many cases of acute myelitis, blood is extravasated at the seat of the inflammation.

toms consist of *painful sensations*, and sometimes *hyperæsthesia* of the skin, to which the sensory nerves, entering the cord at the seat of the lesion, are distributed; *myalgic pains*, and sometimes *pain in the back*.[1] When the inflammatory process involves the postero-external column, *shooting pains*[2] in the sensitive areas of the root-fibres which are irritated, will probably be observed. In many cases the *girdle sensation* is an early symptom; it may persist throughout the course of the disease, and is probably due to irritation of the posterior root-fibres at the junction of the healthy and diseased tissues. These irritative phenomena are usually, even from the first, associated with symptoms of sensory impairment (numbness, 'pins and needles,' anæsthesia and analgesia). After a few hours the irritative phenomena subside, and the symptoms of sensory impairment become more prominent.

Symptoms of sensory irritation may persist in the parts which are supplied by sensory nerve fibres, which enter the cord at the upper level of the lesion. The *girdle sensation* which remains in many cases throughout the disease, is probably due to this cause.

The *motor symptoms* which characterise the stage of irritation are muscular *twitchings* and *tremors*, and in some cases *cramps* and *spasms*. These symptoms are seldom prominent, and, as in the case of the sensory phenomena, they may be from the first accompanied by symptoms of impaired motor power. The twitchings, spasms, etc., are chiefly observed in the muscles supplied by motor nerves coming off from the cord at the level of and below the lesion. In some cases, particularly in those in which the lumbar region of the cord is implicated, the attack may be ushered in by bladder symptoms, such as increased frequency in micturition, spasm of the detrusor, or (more frequently) loss of expulsive power.

The stage of Destruction.—The duration of the irritative stage is, as a rule, short, and in the course of a few hours symptoms due to destruction and impaired function become prominent; the *paresis* rapidly increases, and a condition of complete *paralysis* may be quickly established; the symptoms of sensory irritation subside, and complete *anæsthesia* and *analgesia* are developed. In other cases, the sensory

[1] The pain in the back is not increased by movement, and is never very prominent unless there is associated meningitis.

[2] This symptom is by no means common. When sharp shooting pains are prominent, they probably depend upon associated meningitis.

impairment is only partial, and the symptoms are:—inability to localise tactile impressions, retardation of sensory conduction, '*anæsthesia dolorosa*,' and a peculiar condition described by Charcot, in which a touch on the skin of the affected part produces a diffuse sensation of vibration and pain in the whole of the extremity. In a large proportion of cases the functions of the *bladder* and *rectum* are seriously interfered with; constipation is present in almost all, and in some, as I shall presently mention, paralysis of the sphincters occurs. Gangrenous inflammation (*the acute bed-sore*), see fig. 146, over the sacrum and trochanters, or the heels, which is probably due to an irritative affection of the posterior horn of grey matter, sometimes develops towards the end of the first week; when the lesion is unilateral, the acute bed-sore is developed on the side of the anæsthesia, *i.e.*, on the opposite side to the cord lesion.

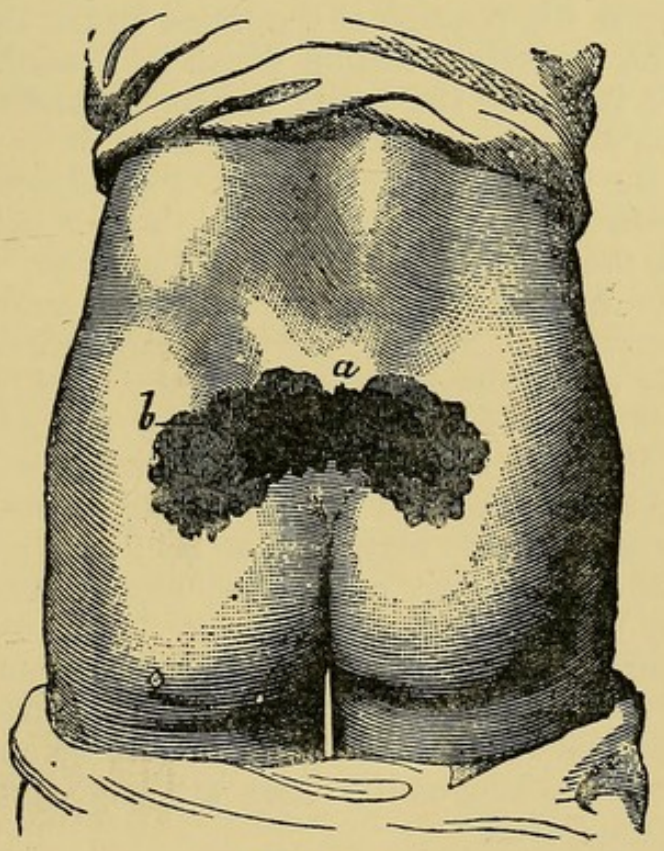


FIG. 146.

The acute bed-sore, from a case of myelitis, which involved the dorsal region of the cord (*After Charcot.*)

a, slough; *b*, erythematous zone.

Cerebral symptoms are rare;[1] but the morbid process does occasionally extend to the nerve centres within the cranial cavity. In extremely rare cases optic neuritis has been observed. The amount of *febrile disturbance* varies greatly in different cases; in some, the temperature rapidly rises and attains a considerable height, 103° or 104° Fahr.; the pulse may reach 120°, 130°, or even 160° in the minute; headache, loss of appetite, thirst, and the general feeling of malaise which accompanies all severe febrile attacks, are then present. In other cases—and they constitute the majority—the elevation of temperature is slight or wanting, and the general constitutional symptoms are not so prominent; in some the fever continues, in others it subsides. When the acute bedsore, and acute cystitis and pyelo-nephritis are established, a fluctuating temperature, hectic symptoms, or pyœmia, may be developed.

The exact extent and distribution of the paralysis depend, of course, upon the position of the lesion in the cord, and the extent of the transverse section which is invaded. In cases, for example, in which the lesion is a total transverse one, all the parts supplied by motor nerves arising from the cord at and below the level of the lesion will be paralysed.

The condition of the affected (paralysed) *muscles* also depends upon the position and extent of the lesion. When the inflammation involves the grey matter of the anterior cornua, the muscles, supplied by motor nerves arising from the affected part of the cord, undergo rapid atrophy, and present the 'reaction of degeneration.' Where the paralysis depends upon a lesion of the motor tract in the lateral column, rapid atrophy and the 'reaction of degeneration' do not occur. The paralysed muscles may, at first, be flaccid; but after a time (*i.e.*, when secondary descending degeneration is established) they become rigid.

The condition of the reflexes varies in accordance with the position of the lesion. The essential facts to remember are:

(1) That the reflex movements of any segment are not interfered with so long as the reflex arc, passing through the segment is uninjured.

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[1] In Polio-myelitis Anterior Acuta, as I have previously mentioned, the onset may be attended with convulsions.

(2.) That lesions (in this case, myelitis) which involve the pyramidal tracts, arrest the cerebral control, give rise to secondary descending degeneration, and are attended with increase of the reflexes passing through inferior segments.

The condition of the paralysed muscles and the state of the reflexes will be more easily understood by referring to the description which I have already given of total and unilateral transverse lesions (see also figs. 71 and 73).

The condition of the bladder and rectum.—The exact nature of the vesical and rectal derangements depends upon the position of the lesion. When the reflex centres are implicated, *i.e.*, when the lesion is situated in the lumbar region of the cord, paralysis of the sphincters will be present;—the urine rapidly becomes ammoniacal, bloody, or purulent, and the trophic form of cystitis may be produced. When the centres for the bladder and rectum are not directly implicated, when, for example, the lesion involves the upper dorsal region, a temporary paralysis of the detrusor may be present, but it usually passes off, and the vesical reflex is re-established. In these cases micturition may be performed unconsciously (see § 86), and the patient may be unable to restrain or influence the process by an effort of the will.

When the cervical region is affected, the upper as well as the lower limbs are paralysed; *priapism* and *hyper-pyrexia* are often present. The action of the heart may be deranged; in some cases the pulse is extremely rapid, in others irregularity, or painful sensations in the region of the heart, are observed.

Lesions in the upper dorsal and cervical regions may be attended with serious derangement of the respiratory functions. Where, for example, the inflammatory process runs an acute ascending course, the different muscles concerned in respiration (viz., those of the abdomen, the intercostals, and the diaphragm) may be all in turn paralysed, intense dyspnœa and death from asphyxia resulting. Paralysis of the abdominal muscles causes difficulty in expiration, in coughing, and in expectoration; the bronchial tubes are apt to become obstructed, and a trivial bronchitis may prove fatal. Paralysis of the intercostal muscles produces difficulty in inspiration, the patient breathes chiefly with the diaphragm, and the extraordinary muscles of respiration are called into play. When the diaphragm itself becomes paralysed, the extraordinary muscles of respiration are thrown into still more vigorous

action, the dyspnœa becomes intense, and death from asphyxia quickly supervenes.

Termination of the acute stage.—The acute stage not unfrequently terminates in death; the fatal result may be due to:—paralysis of the respiratory muscles, cystitis and kidney complications, the acute bed-sore, pneumonia, or other pulmonary affections.

In most cases, the acute stage is tided over, the condition becomes chronic, and the stage of cicatrisation and secondary degenerations is reached. The future course of events varies greatly in different cases.

In many, the constitutional symptoms subside, the general health is gradually re-established, and a chronic paraplegia remains, a considerable amount of motor power being slowly regained, and the sensory derangements being more or less completely recovered from; finally, the patient is able to walk about with the aid of crutches or sticks. In a few cases, the recovery is much more complete; and occasionally, but very rarely, a perfect cure is established. As the process of cicatrisation and secondary descending degeneration advances, the paralysed muscles below the level of the lesion become rigid, their reflexes (especially the deep reflexes) are exaggerated, and the paraplegia assumes the spastic type. In fact, acute and chronic myelitis are the usual causes of this condition.

In other cases little or no improvement occurs. After the acute symptoms have subsided, the patient remains for some time in *statu quo*, and ultimately dies, exhausted by the discharge from extensive bed-sores, or from cystitis, or from complications on the part of the kidney or respiratory organs.

Diagnosis and differential diagnosis.—The conditions with which acute myelitis is most likely to be confounded are, acute meningitis, simple hæmorrhage into the substance of the cord or into the meninges, and hysterical paraplegia.

The differential diagnosis of myelitis and spinal meningitis.—The distinction is sometimes impossible, for the two conditions are not unfrequently combined. Both affections are acute, both may be ushered in by fever, and both are attended by symptoms which are clearly spinal. The main points of difference are, that in myelitis the irritative symptoms are comparatively slight, of brief duration, and soon followed by paralytic symptoms of a marked character; while in menin

gitis the irritative symptoms are very prominent, the irritative stage is long, and the paralytic symptoms are late in appearing and relatively slight. The chief points of differential distinction are given in the following table:—

The differential diagnosis of myelitis and spinal meningitis.

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

The differential diagnosis of myelitis and simple hæmorrhage into the cord.—Here again a distinction is not always possible, for the two conditions are often present in the same case. The points in favour of a simple hæmorrhage are a very rapid (or immediate) onset, which is not preceded or accompanied by fever, and the fact that a high degree of paralysis is at once reached. In myelitis, on the other hand, the onset is, as a rule, more gradual; there is often fever, the paralytic symptoms are gradually developed after a preliminary stage of irritation, the duration of which is usually brief; in some cases, the lesion pursues an ascending course, and the paralysis increases.

The distinction from hæmorrhage into the spinal membranes would probably not be difficult. In that rare condition the symptoms occur abruptly, and are indicative of meningitis and pressure upon the cord rather than of myelitis.

The differential diagnosis of paraplegia, the result of myelitis, and paraplegia due to functional (hysterical) causes, has already been described. (See page 153).

PROGNOSIS.—The prognosis is generally unfavourable; some patients die during the acute stage; in the great majority of which escape the initial dangers, some motor paralysis remains, a perfect cure being extremely rare. The opinion as to the immediate result must be determined by the special features of each individual case. High fever, paralysis of the sphincters, the acute form of cystitis, the acute bed-sore, paralysis of the respiratory muscles, and extension of the morbid process to the medulla oblongata, are highly unfavourable indications.

When the case has become chronic, the opinion as to the future course of the paralysis must be chiefly guided by:—its extent and severity, the effect of treatment, and the pathological character of the lesion. In many cases a fair degree of motor power is regained, and in some syphilitic cases the recovery is complete. Spastic symptoms, which have developed slowly after myelitis, are usually, associated with secondary descending degeneration, and are seldom completely recovered from.

TREATMENT.—The general plan of treatment which I have sketched out in speaking of polio-myelitis anterior acuta is to be adopted. When the case is seen quite at its commencement an attempt may be made to cut short and allay the inflammatory process by the application of ice-bags to the spine, and by the internal administration of ergot and belladonna; these measures are sometimes attended with success, but in most cases the inflammatory process pursues its course, and the treatment must be chiefly directed to tiding the patient over the acute stage, and to the prevention and treatment of bed-sores and bladder complications. (See pages 169 and 170). The diet must be light and nutritious; the patient's room should be kept cool and well ventilated. After the acute stage has passed off, the galvanic treatment may be commenced, iodide of potassium administered, and the usual treatment for a case of chronic myelitis adopted. In syphilitic cases the iodide of potassium and chloride of mercury should be given from the first.

PARALYSIS ASCENDENS ACUTA.

SYNONYM.—Landry's paralysis.

Under the name *paralysis ascendens acuta*, Landry was the first to direct attention to a rare affection, the characteristic feature of which is, motor paralysis commencing in the muscles of the lower extremities, and gradually extending to those of the upper extremities and trunk, and finally to those supplied by motor nerves arising from the medulla oblongata.

PATHOLOGY AND MORBID ANATOMY.—The morbid anatomy of the affection is unknown; in all the cases which have been hitherto examined, with the exception of one doubtful case, which is reported by Dr Ross,[1] the spinal cord, medulla oblongata, brain, peripheral nerves, and muscles, seemed healthy. In consequence of these negative results, and also in consequence of the fact that changes have in some cases been found in the spleen and other glandular organs, similar to the changes found in the infectious fevers, Landry and some other observers think the disease may be due to the introduction into the system of some toxic material.

ETIOLOGY.—The conditions which predispose to and excite the disease are unknown. The affection is more common in men than in women, and seems to occur almost exclusively between the ages of twenty and forty. In some cases, there is a history of exposure to cold and wet; in others of syphilis; in others again the disease follows an acute febrile affection, such as typhoid, pneumonia, etc.

ONSET, SYMPTOMS, AND COURSE.—*Premonitory symptoms* consisting of slight febrile disturbance, symptoms of slight sensory or motor derangement, such as numbness, weakness, and heaviness of the limbs, usually precede the onset of the paralysis. In other cases, the attack commences suddenly.

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[1] Dr Ross found distinct pathological changes in the central group of cells of the anterior horn, but as he did not see the case during life, 'the diagnosis,' he says, 'must perhaps be regarded as somewhat doubtful.'—*Diseases of the Nervous System*, vol. ii. page 270.

The characteristic symptom is muscular weakness, which quickly becomes complete paralysis. The muscles of the toes and feet are first affected; then those of the leg and thigh; the muscles of the hands, arms, and trunk, are next involved in turn, and finally the muscles of respiration and of deglutition are implicated, and the patient dies asphyxiated.

The affected muscles do not become markedly atrophied, and do not present the 'reaction of degeneration.' Their reflexes are for a time retained, but soon become diminished or abolished.

The sensory disturbances are slight; the bladder and rectum are seldom affected; trophic alterations in the skin do not occur, and there are no cerebral symptoms. In most cases fever is absent, and the general state of nutrition is well preserved; in others the temperature is elevated, and the usual constitutional disturbances which attend acute febrile affections, such as quick pulse, thirst, emaciation, etc., are observed.

The *duration* of the disease varies considerably in different cases, the average being from eight to twelve days. Some prove fatal in three or four days; others in two or three weeks. The termination is usually fatal, but a few cases recover.

DIAGNOSIS.—The facts, that there are no marked sensory derangements, no trophic alterations in the skin, and no disturbances of the bladder and rectum, at once distinguish the condition from the ordinary form of acute ascending myelitis; while the absence of sensory disturbances, the ascending course of the disease, and the negative evidence afforded by post-mortem examinations, seem to show that the condition cannot be due to a neuritis of the common sensory motor nerve trunks.

The disease is readily distinguished from *polio-myelitis anterior acuta* by the facts; that the paralysis is progressive, and that the paralysed muscles do not undergo rapid atrophy, and do not present the 'reaction of degeneration.'

Landry's paralysis closely resembles, in some particulars, the subacute inflammation of the anterior cornual region which was described by Duchenne under the name of Paralysie générale spinale antérieure subaiguë. The points of differential distinction are given on page 277.

THE PROGNOSIS is very unfavourable; a rapid, ascending course, the early implication of the respiratory muscles, and the occurrence of bulbar symptoms are very unfavourable indications; it is hopeless in those cases in which the paralysis rapidly ascends, in which the respiratory muscles are implicated, and in which bulbar symptoms occur.

TREATMENT.—The same treatment which has been recommended for acute inflammation of the anterior cornual region (see page 180) is to be adopted.

CHRONIC MYELITIS.

Under the term chronic myelitis, we may include all the indiscriminate lesions, which run a slow and tedious course, which are unattended with fever, and which present the microscopical characters described on page 53.[1]

PATHOLOGY AND MORBID ANATOMY.

The extent and distribution of the lesion varies very much in different cases, and here, as in acute myelitis, several distinct varieties may be described, the most important of which are:—

1. *Chronic transverse Myelitis,* in which the whole transverse section, usually in the lumbar or dorsal region is affected.
2. *Chronic disseminated Myelitis,* in which many separate foci of disease are scattered throughout the cord, and occupy very different positions in the transverse section at different levels. This form is very generally syphilitic.
3. *Chronic peripheral or annular Myelitis,* in which the inflammatory process chiefly affects the surface of the cord. This form is usually secondary to meningitis, or to compression.
4. *Chronic focal Myelitis,* in which a single patch of inflammation occupies a portion only of the transverse section.

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[1] Locomotor ataxia and primary lateral sclerosis are considered by many authorities to be chronic inflammations; but they are system diseases, and have therefore, been previously considered. Again, disseminated sclerosis undoubtedly presents many of the histological characters of a chronic myelitis but it is a cerebro-spinal affection of such a distinct type, that it will be considered separately

5. *Chronic general Myelitis,* which is perhaps the lesion in some cases of chronic ascending paralysis.

Or we may, as in the acute form, make an etiological subdivision, and class the cases as chronic *syphilitic* myelitis, chronic *compression* myelitis, chronic *traumatic* myelitis, chronic *idiopathic* myelitis, and so on.

The affected part of the cord is generally firmer than natural; the greater density being due to an increase of the connective tissue elements; in advanced cases the nerve elements may be entirely replaced by the connective tissue growth.

The microscopical characters of the lesion in chronic myelitis are described on page 53.

Etiology.—The same causes which give rise to acute myelitis may also produce the chronic form of the disease, and an acute attack not unfrequently passes into the chronic condition. Hereditary predisposition to nerve disease seems in some cases to be an important etiological factor; and syphilis is a frequent cause both of the focal and disseminated varieties.

Mode of Onset, Symptoms, and Course.—The onset is in most cases extremely gradual. The first symptoms may consist of sensory derangements, such as numbness, 'pins and needles,' and occasionally, though rarely, of eccentric pains or hyperæsthesia; in other cases motor symptoms, such as weakness in the lower extremities, difficulty in emptying the bladder, or obstinate constipation, are the most marked features at the commencement of the attack.

The clinical picture presented by fully developed cases of chronic myelitis varies considerably in different cases, in accordance with the position and extent of the lesion. The most prominent symptoms are:—gradually increasing motor weakness, which ultimately becomes complete paralysis; loss of sensibility, which after a time may become complete; constipation; difficulty in emptying the bladder; and, in some cases, paralysis of the sphincters.

The paralysis is generally paraplegic in distribution; in fact, most cases of chronic paraplegia depend upon chronic myelitis, or acute myelitis which has become chronic. The trophic condition of the paralysed muscles, the condition of

the reflexes, and indeed the character of all the symptoms, depend upon the exact position and extent of the lesion; in many cases rigidity of the muscles and increase of the deep reflexes are combined with the loss of motor power; in fact, chronic myelitis is by far the most common cause of spastic paraplegia—a circumstance which must always be kept in view in the diagnosis of supposed cases of *primary lateral sclerosis.*

The general health is usually well preserved, and the other organs and systems are, as a rule, healthy.

In the course of time the paralysis becomes complete, the patient is confined to bed; sloughing of the skin, cystitis, and complications on the part of the kidney may develop, or the case may end by an intercurrent attack of pneumonia or phthisis.

In those cases in which the myelitis is due to compression, or in which the membranes become involved in the inflammatory process, the clinical picture is still more complicated; and symptoms characteristic of an extra-medullary lesion (see page 66) are present.

Diagnosis.—In most cases the diagnosis of chronic myelitis is synonymous with the diagnosis of chronic paraplegia. The general plan of diagnosis has been already described (see p. 149), and to that description the reader is again referred; but it will perhaps be necessary to mention the chief conditions which may give rise to difficulty. They are as follows:—

1. The various conditions with which functional paraplegia is associated, viz., hysterical paraplegia (see p. 153); reflex paraplegia (see p. 157); alcoholic paraplegia and anæmic paraplegia (see p. 158).

2. Lesions of the cauda equina (see p. 158).

3. Conditions which produce slow compression of the cord, such as disease of the bones or membranes; in these cases myelitis is often present as a secondary complication, and an exact diagnosis is not always possible, but the presence of an extra-medullary lesion is usually indicated by the symptoms which result from pressure on the nerve roots and membranes (see p. 66).

4. Simple softening. It is impossible, without post-mortem evidence, to distinguish the two conditions (paraplegia due to chronic myelitis, and paraplegia due to simple softening).

5. Primary lateral sclerosis. *The differential diagnosis of the two conditions—spastic paraplegia, due to chronic myelitis, and primary lateral sclerosis*—is shown in the table, p. 218.

The exact anatomical diagnosis of transverse myelitis.—In cases of transverse myelitis we must determine the exact position and vertical extent of the lesion. The following are the steps in the enquiry:—

First, Ascertain the exact distribution of the sensory and motor derangements, and by reference to paragraph and figures 99, 100, 101, and 102, determine the segments of the cord with which the affected sensitive and muscular areas are connected. In this way the upper level of the lesion is readily determined.

Second, Observe the reflex and trophic conditions of the muscular areas of the segments of the cord, beginning at the upper level of the lesion and proceeding downwards. The vertical extent of the lesion can in this manner be ascertained, as will be readily understood by reference to figures 73 and 74. (The reflexes passing through the affected segments are abolished, while the reflexes passing through the segments which are situated below the lesion are exaggerated. The muscles supplied by anterior nerve-roots arising from affected segments are markedly atrophied, but the nutrition of muscles supplied by motor nerves arising from segments below the lesion is not much interfered with. Then again, the presence of an *acute* bed-sore in any 'sensitive area' shows that the grey matter of the segment connected with that sensitive area, or the sensory nerve fibres proceeding to it, are implicated by an acute irritative lesion.)

Prognosis.—The most frequent termination is death, but the course is usually very protracted. Occasionally the morbid process is arrested, and the motor paralysis is to some extent recovered from; very exceptionally the cure is complete. In syphilitic myelitis the prognosis is more favourable; most cases improve, and some completely recover, even after spastic symptoms have been prominent.

Treatment.—The general plan of treatment which is described on p. 166 is to be adopted. The most useful drugs are *iodide of potassium* and *mercury* in syphilitic cases; *ergot* and

nitrate of silver where spastic symptoms are prominent; *arsenic, iron, quinine, strychnine,*[1] and *phosphorus* where there is paraplegia with flaccidity. Erb[2] speaks very favourably of a properly conducted 'water-cure,' and of electricity. Counter-irritation to the spine, especially the actual cautery, is in some cases beneficial.

CEREBRO-SPINAL SCLEROSIS.

Synonyms. — Multiple sclerosis; Disseminated multiple sclerosis; Insular sclerosis; Sclérose en plaques disséminées.

Cerebro-spinal sclerosis is an extremely chronic affection, in which degenerated patches (nodules of interstitial myelitis and encephalitis) are scattered throughout the nerve centres (brain, pons, medulla oblongata, cerebellum, and spinal cord) and are also found on the peripheral nerves (especially the cranial nerves and the spinal nerve-roots).

Pathological Anatomy.—The reader is referred to the description which has been given of the pathological characters of the lesion, on pages 55 and 56.

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[1] I must again caution the reader against using this drug in those cases in which irritative phenomena (*eg.*, spastic symptoms) are present.

[2] 'On the other hand,' says Erb, 'the results of a rationally conducted *cold-water cure* are exceedingly favourable. Out of twenty-nine cases of which I have notes, the results of this treatment were favourable in twenty-one, negative in five, and unfavourable in three. Here, too, of course, everything depends on the manner in which the cold water is used. I cannot insist too much on the danger of forced cures, of all severe and strongly exciting procedures, such as the employment of water at very low temperatures, douches, sharp slappings, etc. These measures are as a rule, absolutely injurious in cases of myelitis; even wet packs of the entire body have, to my surprise, generally proved injurious. Simple rubbing with we cloths, foot-baths and sponging the back, hip-baths, half-baths with affusions to the back, local compresses to the back, left on till they become warm, etc., seem to be the measures which are chiefly applicable. The treatment should always be begun with moderate temperatures (20-25° C.—68-77° F.), and we should never go below 16-20° C. (60½-53½° F.). I believe also, that excessive prolongation of the treatment is injurious. Many over-zealous hydropathists are only too often led, by their great confidence in the water cure, to prolong the treatment to an extent that proves detrimental to the patients.'—*Ziemssen's Cyclopædia*, vol. xiii. p. 461.

Etiology.—In many cases there is a history of traumatic injury, such as a blow on the head or spine; in some the symptoms seem to develop after exposure to cold and wet; occasionally the condition appears as a sequel to acute diseases; pregnancy also has been stated to be a cause; in a few cases there is a distinct hereditary tendency; but in most the exact conditions which excite the affection are obscure.

Cerebro-spinal sclerosis is essentially an affection of early life, generally appearing between the ages of fifteen and thirty; it seems equally common in both sexes; Charcot, it is true, states that women are more liable to be attacked than men, but his experience in this respect differs from that of Erb and many others; of the six cases which have come under my own observation, five were males.

Onset, Symptoms, and Course. — The *onset* is, as a rule, extremely slow and gradual; occasionally the symptoms develop somewhat abruptly. In one of my own cases, for example, the affection commenced suddenly with deafness and 'noise in the head,' after exposure to a hot sun.

As we might expect from the extremely irregular distribution of the lesion, the symptoms are multifarious, in fact, as Charcot states, the affection is polymorphous one.[1] Both cerebral and spinal symptoms are usually present; but in some cases the lesion is, for a time at least, chiefly limited to the intra-cranial nerve centres, and the symptoms are for the most part cerebral; in others the spinal cord is chiefly or (?) exclusively involved, and spinal symptoms are most prominent. In the following account I shall not limit myself entirely to the spinal symptoms, but shall describe the common (cerebro-spinal) variety of the disease.

In many instances, the first symptoms are spinal, and consist of some derangement of the motor nerve apparatus, which is manifested externally in the form of motor impairment (paresis) or inco-ordination (ataxia). The loss of motor power very generally commences in one leg, and subse-

[1] In some cases the lesion may for a time be chiefly confined to the posterior columns of the cord, and symptoms very closely resembling or identical with those of locomotor ataxia may be present; in others, the lateral columns are chiefly invaded, and spastic paraplegia is developed.

quently extends to the other, or to the arms. In other cases, the first symptoms are cerebral, such as headache, vertigo, deafness, or some psychical derangement.

These symptoms slowly increase, and a characteristic symptom is soon developed, viz., a rhythmical, jerking tremor, which only occurs on voluntary effort, and which ceases when the parts are at perfect rest. In consequence of this jerking tremor, movements are rendered extremely unsteady, though their main direction is still preserved. The tremor may occur in any of the muscles, but it is generally more marked in some parts than in others. Charcot supposes that it is due to irregular conduction through axis cylinders, which are lying naked in the midst of the sclerosed tissue, but other authorities think that it results from the presence of sclerotic nodules in some special parts of the brain (the pons, and the parts of the brain anterior to it). When the patient is quietly seated there may be nothing noticeable, or the only peculiarity which close observation can detect is a slight rhythmical jerking of the head. In well-marked cases any voluntary effort at once develops the characteristic tremor, and the more intense the effort the greater does the tremor become; it may be exquisitely demonstrated by making the patient raise a glass of water to his lips; the main direction of the movement is preserved, and the glass reaches the lips, but it is forcibly jerked against the teeth, and the contents are thrown over the patient's face and neck. Some patients attempt to 'dodge' the tremor (*i.e.*, to reduce the amplitude of the jerking movements of the head and arm) by throwing the head back and fixing it as it were between the shoulders, and by holding the arm and forearm firmly to the side, but even then their efforts are unsuccessful, and the water is spilt.

The jerking tremor in the muscles of the legs and trunk produces marked unsteadiness in the gait. In the earliest periods of the case the unsteadiness may be extremely trifling, and the only alterations which can be detected in many cases are a certain stiffness in the way in which the neck is carried, and a slight jerking of the head. When the disease is more advanced, the gait is extremely unsteady; in some cases it resembles that of locomotor ataxia; in others the inco-ordination chiefly affects the muscles of the trunk, and the patient does not walk deliberately in a straight line (as the subject of locomotor ataxia does) but is apt to shoot forcibly from side

to side; in other cases again the feet seem to cling to the ground, and the spastic gait is conjoined with a rhythmical, shaking tremor of the whole body. A jerking movement of the eyeballs (nystagmus) occurs in many cases, and is a very characteristic symptom.

The affected muscles are of normal bulk, and their electrical reactions are healthy. The reflexes are, as a rule, normal or exaggerated.[1] The bowels are generally constipated, but the functions of the bladder are seldom interfered with, till a much later stage. The sensory functions are not much affected at this period of the case; as a rule, subjective derangements of sensation, such as 'pins and needles,' numbness, etc., are experienced by the patient, but distinct objective anæsthesia cannot be demonstrated. The general state of nutrition is well preserved, and complications on the part of the other systems and organs are rarely present.

The symptoms gradually increase; but periods of temporary improvement, or even complete remission, are not uncommon, and may lead an inexperienced observer to expect a favourable termination of the case. After the condition has become well established, the *facial expression* becomes vacant, stolid, and stupid-looking; and a peculiar alteration in the voice is very generally observed, the *speech* is slow and drawling, every syllable being pronounced separately (*scanning speech*); the tone of voice, too, is singularly monotonous; towards the end of the case it may be weak and whispering. The cerebral symptoms become more prominent, the headache and vertigo may continue; the mental faculties are decidedly blunted; in many cases the patient becomes irritable, and loses his self-control; actual mental derangement is occasionally observed. Dimness of vision is very frequent; in some cases it is only apparent, depending upon nystagmus; in others, it is real, and is associated with white atrophy of the optic discs or alteration of the central parts of the visual nerve apparatus. The pupils may be irregular, in some cases they are 'pin pointed,' but the Argyll Robertson phenomenon (see p. 230) is probably rare. In a small proportion of cases, apoplectiform attacks, characterised by complete insensibility, quick

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[1] Exceptions to all of these statements may occur, for the lesion may invade any part of the transverse section of any spinal segment.

pulse and elevation of temperature, occur; the patient may die in the attack, but as a rule, the coma disappears in the course of a day or two, leaving behind it hemiplegia, which in its turn is quickly recovered from. These symptoms, which characterise the *first stage* of the disease, slowly but steadily increase, and in the course of time (two to six years) the *second stage* is reached.

The patient can no longer stand or walk, but is confined to bed or to his chair. In some cases the paralysis is complete; in many the legs are rigidly extended, the deep reflexes markedly increased, and the condition exactly resembles an advanced stage of spastic paraplegia. The tremor, too, is markedly intensified, and attempts at voluntary movement not only cause an increase of the rigidity and spasms, but may produce a violent clonic movement in the muscles, causing a forcible shaking of the whole body. The mental deterioration becomes much greater, the speech still more scanning, monotonous, and weak. The general state of nutrition for a time continues good.

In the course of a few years the *third* stage is reached. The general state of nutrition is now affected, and the patient becomes emaciated; the mental deterioration becomes greater; the paralysed muscles may become profoundly atrophied; bulbar symptoms, paralysis of the bladder, cystitis and bed-sores may develop. Ultimately he dies either from simple exhaustion, from pyœmia, from paralysis of the respiratory muscles, or from some intercurrent complication.

The average duration of the whole case is from eight to ten years; some cases linger on for fifteen or twenty years; occasionally the course is rapid, and death occurs within three years of the onset of the disease.

Diagnosis.—In fully developed and typical cases the diagnosis presents no difficulty; the gradual onset, and slow progress of the symptoms with perhaps periods of improvement; the presence of both spinal and cerebral symptoms, *especially the peculiar tremor*, the marked character of the motor derangements, the comparative insignificance of the sensory disturbances, the vertigo, the alterations in speech, and in the mental condition, constitute a striking picture

which it is impossible to mistake. But in the early stages and in those rare cases in which the symptoms are exclusively spinal or exclusively cerebral, it may be difficult or impossible to diagnose the condition.

The affections which are most liable to be mistaken for cerebro-spinal sclerosis are: — *cerebellar tumour, locomotor ataxia, and paralysis agitans.*

The differential diagnosis of cerebro-spinal sclerosis and cerebellar tumour.—In both conditions there may be headache, giddiness, and disordered gait from weakness or incoordination of the muscles of the trunk; and in those cases of cerebro-spinal sclerosis in which the characteristic tremor is absent or slightly marked, the diagnosis from cerebellar tumour is extremely difficult. The chief points of distinction are:—

1. *The condition of the optic discs.*—Optic neuritis or optic atrophy is present in most cases of cerebellar tumour; they are rare and only late symptoms in cerebro-spinal sclerosis.

2. *Vomiting.*—This is a prominent symptom of cerebellar tumour, but not of cerebro-spinal sclerosis.

3. *Headache.*—This is usually much more severe in cerebellar tumour than in cerebro-spinal sclerosis.

4. *The presence of the characteristic tremor.*—This is a very strong point in favour of cerebro-spinal sclerosis; a similar tremor is occasionally seen in tumours of the cerebellum, and is, I believe, due to interrupted conduction through the motor strands of the medulla oblongata which are pressed upon by the growth.

5. *Convulsive seizures.*—In many cases of cerebellar tumours, tonic spasms chiefly affecting the muscles of the back, or epileptiform convulsions are observed; they are quite exceptional in cerebro-spinal sclerosis.

6. *The age of the patient.*—Cerebro-spinal sclerosis is rare in children, and is most common between the ages of fifteen and thirty; cerebellar tumour often occurs between the ages of twenty and thirty, but is very common in children.

7. *The course of the disease.*—Cerebellar tumours, as a rule, run a much more rapid and continuously progressive course than cases of cerebro-spinal sclerosis.

When the scanning speech, the monotony of the voice, the heavy stolid expression, and the volitional tremor have become prominent, the diagnosis of the two conditions no longer presents any difficulties.

The differential diagnosis of cerebro-spinal sclerosis and locomotor ataxia.—In some cases of cerebro-spinal sclerosis, the lesion is chiefly confined to the posterior columns of the cord, and many of the symptoms of locomotor ataxia, including the lightning pains and characteristic gait are present; the diagnosis may then be difficult or impossible. But such a distribution of the lesion is rare, and the distinction can generally be made by attention to the following points:—

1. *Age.*—Locomotor ataxia is seldom seen before thirty; cerebro-spinal sclerosis is most frequently met with, between twenty and thirty.

2. *Sex.*—Locomotor ataxia is much more frequent in males than in females; cerebro-spinal sclerosis occurs with equal frequency in the two sexes.

3. *The condition of the sensory nerve apparatus.*—Lightning pains, and anæsthesia, are prominent features in locomotor ataxia, but are seldom noticeable in cerebro-spinal sclerosis.[1]

4. *The presence of motor paralysis.*—Paresis and paralysis do not occur until the terminal stage of locomotor ataxia, but are early symptoms in cerebro-spinal sclerosis.

5. *The characteristic volitional tremor.*—This symptom is distinctive of cerebro-spinal sclerosis, for it does not occur in locomotor ataxia.

6. *The condition of the reflexes.*—The knee-jerk and the pupil reflex to light are absent in the great majority of cases of locomotor ataxia, but are usually present in cerebro-spinal sclerosis.

7. *Nystagmus.*—This symptom is common in cerebro-spinal sclerosis.

8. *The condition of the mental faculties and of speech.*—The mental condition and speech are not interfered with in locomotor ataxia, and in that condition, vertigo, which is almost always present in cerebro-spinal sclerosis, does not occur.

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[1] When a patch of sclerosis invades the postero-external column, these symptoms may, of course, be present.

The differential diagnosis of cerebro-spinal sclerosis, and paralysis agitans, in typical cases presents no difficulty, but there are cases in which the two conditions seem combined. The points of distinction are:—

1. *The character of the tremor.*—(*a*) The tremor of cerebro-spinal sclerosis only occurs on voluntary movement, and ceases when the muscles are at rest; the tremor of paralysis agitans occurs while the muscles are at rest, and is diminished or arrested by volitional efforts. (*b*) The tremor of paralysis agitans is a finer tremor than that of cerebro-spinal sclerosis. (*c*) The tremor of paralysis agitans rarely, if ever, affects the head; in cerebro-spinal sclerosis the head is invariably involved.

2. *Age.*—Paralysis agitans is a disease of advanced age; while cerebro-spinal sclerosis is a disease of youth.

3. *The associated symptoms.*—In cerebro-spinal sclerosis other symptoms (cerebral and spinal) are present, and confirm the diagnosis.

PROGNOSIS.—The prognosis is extremely unfavourable, the condition sooner or later terminates fatally.

TREATMENT.—The treatment must be mainly directed to the relief of symptoms, for all remedies which have been administered, up to the present time, have failed to arrest the progress of the disease. Nitrate of silver, arsenic, iodide of potassium, the constant (galvanic) current and hydropathy should be tried in turn, even though the results are discouraging.

INTRA-MEDULLARY HÆMORRHAGE, AND INTRA-MEDULLARY TUMOURS.

These subjects will be more conveniently described in connection with extra-medullary hæmorrhage and extra-medullary tumours (see pages 279 and 283).

TABULAR CLASSIFICATION OF EXTRA-MEDULLARY LESIONS.

EXTRA-MEDULLARY LESIONS.

- *Meningitis.*
  - *Leptomeningitis.*
    - Acute.
    - Chronic.
  - *Pachymeningitis.*
    - Externa.
    - Interna.
      - Hæmorrhagica.
      - Hypertrophica.
- *Extra-medullary Hæmorrhage.*
- *Extra-medullary Tumours.*

SPINAL MENINGITIS.

Inflammation of the spinal membranes may be either *acute* or *chronic*, *general* or *local*. In some cases the pia mater and arachnoid are chiefly affected; in others the inflammatory process is for the most part limited to the dura. Inflammation of the pia and arachnoid is technically termed *leptomeningitis spinalis;* inflammation of the dura is called *pachymeningitis spinalis;* leptomeningitis spinalis may be either acute or chronic; pachymeningitis spinalis is almost invariably chronic and local. It will be necessary, therefore, to consider these varieties separately.

LEPTOMENINGITIS SPINALIS.

Inflammation of the pia and arachnoid is much more frequent than inflammation of the dura, and it is the condition which is usually meant by the term spinal meningitis.

ACUTE LEPTOMENINGITIS SPINALIS.

Acute inflammation of the pia and arachnoid is almost always *general*, and is usually associated with a similar condition (inflammation) of the cerebral membranes; while in many cases the periphery of the cord is affected by the inflammatory process. The clinical picture is, therefore, in most cases a complicated one.

Morbid Anatomy.—The inflammatory process commences with a *stage of congestion* and hyperæmia, which is seldom seen *post-mortem*, for the disease is rarely fatal until a later period.

During the *first stage* of the inflammatory process the vascularity of the membranes is very much increased; the vessels are gorged with blood; small extravasations of blood sometimes occur; the arachnoid looses its normal, smooth appearance, and becomes velvety and swollen; the spinal fluid is slightly turbid.

The duration of this stage is probably very short, and the *second stage*, that of *exudation*, is next developed. The pia mater and arachnoid are more swollen, and are covered with exudation matters, consisting of fibrinous lymph, leucocytes, or pus; the spinal fluid is more turbid, and contains flakes of lymph and other exudation products; in some cases it consists chiefly of pus. The exudation matters are generally more abundant on the posterior surface of the cord, for the patient usually lies on his back, and the exudation products gravitate to the most dependent point. The peripheral layer of the cord, and the nerve roots, are very frequently implicated by the inflammatory process,—a fact which is not to be wondered at, when we remember that the pia is the nutritive membrane of the cord, and that from it numerous processes of connective tissue and blood-vessels pass into the interior of the organ. In tubercular cases miliary nodules can usually be seen in large numbers on the surface of the inflamed membranes, more particularly on the surface of the arachnoid. In severe cases death generally takes place during the second stage of the disease.

Should the *third stage* be reached, the exudation matters are absorbed, the inflammatory products tend to organise, and adhesions are formed between the opposed surfaces of the inflamed parts.

Etiology.—Acute inflammation of the pia and arachnoid is much more common during childhood and youth than during the later period of life. Its most frequent cause is (probably) exposure to cold and wet; in many cases it is tubercular; and it not unfrequently results from extension to the membranes of the cord, of an inflammation which has commenced in the cerebral meninges. In some cases it is due to traumatic injuries (blows on the spine, etc.); and in fact all the conditions which have previously been mentioned as causes of myelitis (see p. 238), may give rise to it. Violent cerebro-spinal meningitis sometimes results from the bursting of an abscess into

the cavity of the cranium, or into the spinal canal; and a very acute form of the disease which is seldom met with in this country, is probably due to the introduction into the system of a 'particulate,' organic poison, and is termed epidemic cerebro-spinal meningitis.

ONSET, SYMPTOMS, AND COURSE.—*Premonitory symptoms*, such as headache, irritability of temper, vomiting, slight febrile disturbance, etc., are sometimes observed, but in many cases the disease is suddenly ushered in by a rigor or convulsions.

The *pyrexia* is, as a rule, considerable, and the general symptoms which are associated with rapid elevation of temperature, such as quick pulse, heat of skin, thirst, etc., are present. When the inflammatory process affects the membranes of the brain as well as those of the spinal cord, cerebral symptoms, consisting of headache, vomiting, intolerance of light and noise, contracted pupils, epileptiform convulsions, etc., are the prominent symptoms at the commencement of the case, and it may be some hours or days before the characteristic spinal symptoms are developed.

The great feature of the spinal symptoms is, that they are indicative of an irritating rather than a destroying lesion.

Pain in the back, which is increased by the slightest movement of the patient, but not aggravated by percussion or pressure on the spine, and which depends upon irritation of the sensory nerve filaments in the pia and dura, is one of the most marked symptoms.

Shooting pains and *hyperæsthesia* in the sensitive areas of the posterior nerve roots, together with *spasms* and *rigidity of the muscles* supplied by anterior nerve roots, which are irritated by the inflammatory process, are also highly characteristic. In consequence of the muscular spasm the spine is stiff and rigid; in some cases the head is drawn back so that the occiput is thrust, as it were, between the shoulders; in others, the spine is so strongly arched that a condition of opisthotonos is produced; in others again, the thighs are firmly flexed on the abdomen, and the legs on the thighs—a position which is, however, much more frequent in chronic than in acute cases. Temporary exacerbations of the spasms every now and again occur, and the retraction of the head

and arching of the spine are thereby aggravated. In consequence of the retraction of the head and neck, and the extreme tension of the anterior muscles of the neck (sterno-thyroid, sterno-hyoid, and thyro-hyoid), the larynx may be firmly pressed against the spinal column, and difficulty in breathing, attended by a marked stridor, and difficulty in swallowing, may be observed.

In the earlier periods of the case there is no paralysis, but the patient instinctively lies as quiet as possible, for he knows by painful experience that any effort on his part, or any external irritation will be attended by pain and an increase in the spasms.

During the irritative period, retention of urine is occasionally observed; in some cases it results from spasm of the sphincter; in others it is due to paralysis of the detrusor. The bowels are obstinately constipated.

As the case progresses, symptoms indicative of sensory and motor impairment are gradually established, and should the case continue sufficiently long, complete paralysis and anæsthesia, paralysis of the sphincters, and bed-sores, may be developed.

The reflex irritability is increased during the stage of irritation, but afterwards becomes diminished or abolished. When the inflammatory process affects the cervical region, severe dyspnœa, the result of spasm or paralysis of the respiratory muscles, may be a prominent symptom.

The fever is of an irregular type; the temperature may reach 104° F., but is generally below that point. Emaciation rapidly takes place, and in severe cases the patient is soon worn out by sleeplessness and the intensity of his sufferings.

When the inflammation involves the cerebral meninges, the characteristic symptoms of cerebral meningitis are, of course, present. In those cases in which the morbid process is limited to the membranes of the spinal cord, cerebral symptoms are wanting, and the mind may be quite clear until the end. Where the inflammation of the membranes is complicated with inflammation of the cord, the paralytic symptoms are more prominent, and the other symptoms which characterise acute myelitis are present.

The duration varies considerably in different cases; in purulent meningitis, — the result of the bursting of an abscess into the brain or spinal canal,—and in severe cases of

the epidemic variety of the disease, death may take place within two or three days, or even within a few hours from the commencement of the attack. In tubercular and traumatic cases the progress is, as a rule, more tardy, and not unfrequently the condition becomes chronic. In acute cases death may result from spasm or paralysis of the respiratory muscles; or, it may be due to cerebral complications. In chronic cases the patient dies from gradual exhaustion, from bed-sores or cystitis, or from respiratory or other complications.

DIAGNOSIS.—An acute onset, fever, pain in the back, shooting pains in the limbs, hyperæsthesia, motor spasms, rigidity of the spinal column, and increased reflex excitability, are characteristic of acute spinal meningitis. The only conditions which are likely to be mistaken for the affection are myelitis and tetanus.

The *differential diagnosis of spinal meningitis and myelitis* has already been considered. (See page 245.)

The points of *differential distinction between acute spinal meningitis and tetanus* are given in the following table:—

	Acute Spinal Meningitis.	Tetanus.
CAUSE.	Often indistinct.	In traumatic tetanus there is a history or presence of a wound.
DERANGEMENTS OF SENSIBILITY.	Pain in the back, shooting pains in the limbs, and hyperæsthesia, are prominent symptoms.	There are no sensory derangements.
SPASMS.	Opisthotonos may be present, but the tonic spasms are never so prominent as in tetanus. Trismus and the *risus sardonicus* are not observed.	Tonic *convulsions* occuring in paroxysms, and affecting almost all the muscles of the body, are characteristic. Trismus occurs at the commencement of the attack; the *risus sardonicus* is a marked feature.
REFLEX IRRITABILITY.	The reflexes are increased, but not to an extreme degree.	An extraordinary increase of the reflexes occurs.
CEREBRAL COMPLICATIONS.	Often present.	Are not observed.

PROGNOSIS.—Acute spinal meningitis is an extremely serious affection, and in many cases it proves fatal. In rheumatic and syphilitic cases the prognosis is more favourable than in the other varieties of the disease. During the progress of the case periods of amelioration are not uncommon, but as a rule the improvement is only temporary. In those cases in which the inflammatory process is limited to the membranes of the cord, the prognosis is more favourable than in cases in which the cerebral membranes are also affected. Dyspnœa, the result of spasm or paralysis of the respiratory muscles, is an extremely unfavourable symptom.

TREATMENT.—In the earlier stages of the case, the antiphlogistic treatment which has been recommended for acute myelitis (see page 246) is to be adopted; and the pain and spasm are to be allayed by morphia, chloral, and bromide of potassium. Great attention must be paid to the feeding and nursing of the patient, and the condition of the bladder and rectum must be carefully attended to.

In those cases in which the acute symptoms subside, and in which the chronic stage is reached, mild counter-irritants, such as iodine and small blisters, may be applied to the spinal column, and the iodide of potassium should be given internally. At this stage of the disease an important part of the treatment consists in the prevention of bed-sores and other complications, and in the treatment of the paralysis which is often present. The same means which have been previously recommended for the treatment of the paralysis in chronic myelitis are to be employed (see page 252); but it is important to remember that electricity should not be used until all signs of meningeal irritation have disappeared.

CHRONIC LEPTOMENINGITIS SPINALIS.

In some cases inflammation of the pia and arachnoid is chronic from the outset, but in others a chronic leptomeningitis results from a previous acute attack.

ETIOLOGY.—The same causes which produce acute leptomeningitis spinalis may give rise to the chronic form of the disease; but exposure to cold and wet, traumatic injuries, syphilis, extra-medullary lesions, such as extra-medullary

tumours, and intra-medullary lesions which extend to the surface of the cord and then implicate the membranes, are the most common causes of the condition.

Pathological Anatomy.—Where the condition has resulted from a previous acute attack, an extensive area of the membranes may be involved, but in the majority of cases the inflammatory process is limited in its distribution. The intense hyperæmia, which characterises acute inflammation of the membranes, is not observed, but the blood-vessels are dilated and their walls thickened; the spinal fluid is generally turbid and increased in quantity. The most characteristic post-mortem appearances consist in opacities and thickenings of the membranes, and in the presence of adhesions which bind together the opposed surfaces of the inflamed parts; the pia is thickened and firmly adherent to the cord; the connective tissue septa passing into the cord are thickened, and in many cases a peripheral myelitis or peripheral sclerosis of the spinal cord can be demonstrated; in some cases the cord lesions are still more extensive. The nerve roots may be compressed by the thickened membranes, and in some cases they are distinctly softened or atrophied.

Onset, Symptoms, and Course.—The onset is extremely gradual, and unattended by fever; an exception of course occurs in those cases in which an acute inflammation has become chronic. Pain in the back, shooting pains and hyperæsthesia in the sensitive areas of the posterior roots passing through the affected portion of the membranes, are usually well-marked, but in consequence of the more limited distribution of the inflammation, these symptoms are more localised than in the acute form of the disease. Muscular spasms and rigidity are much less prominent than in the acute variety; while motor weakness, which gradually increases and ultimately may become complete paralysis, is usually an early symptom; the paresis and muscular spasms are, like the sensory phenomena, generally limited in distribution. In the earlier stages the muscular nutrition and reflex activity are not much interfered with, but towards the later periods of the case the muscles which are supplied by compressed and atrophied anterior nerve roots may be markedly wasted, while their reflexes are diminished or completely

abolished. The functions of the bladder and rectum are not interfered with, except in those cases in which the nerves, arising from the lower end of the cord, are implicated by the lesion.

The description, which has just been given of the symptoms of chronic meningitis, applies to the cases in which the inflammatory process is chiefly limited to the membranes; when, as so frequently happens, the cord itself is also affected, symptoms of chronic myelitis will also be present.

DIAGNOSIS. — Locomotor ataxia, chronic myelitis, and 'spinal irritation,' are the conditions which are most likely to be mistaken for chronic spinal meningitis.

The differential diagnosis of chronic meningitis and locomotor ataxia.—Some of the symptoms of locomotor ataxia are probably due to the chronic inflammation of the membranes, which is usually found over the posterior surface of the cord; and in the earlier stages the affection (locomotor ataxia) may be confounded with chronic meningitis. In locomotor ataxia the meningitis is practically limited to the posterior surface of the cord, the anterior roots are not involved, and symptoms of motor irritation and paralysis are not observed; the lightning pains are, as a rule, much more severe than in chronic myelitis, while eye-symptoms (see page 240) are usually present. When the characteristic inco-ordination is developed, the diagnosis no longer presents any difficulty.

The differential diagnosis of chronic meningitis and chronic myelitis.—An uncomplicated case of chronic meningitis is distinguished from an uncomplicated case of chronic myelitis by attention to the same points which have been already referred to, in speaking of the differential diagnosis of acute meningitis and acute myelitis (see page 245). In some cases a sharp distinction is impossible, for the two conditions are not unfrequently combined.

The differential diagnosis of chronic meningitis and 'spinal irritation.'—In some cases the distinction is difficult. 'Spinal irritation' occurs chiefly in young women, and is very frequently accompanied by symptoms of hysteria; its characteristic features are (*a*) *pain in the back*, usually between the

scapulæ, but not unfrequently in the lower dorsal or lumbar regions; (*b*) *marked hyperæsthesia*, the slightest touch on the skin producing loud complaints of pain and suffering; (*c*) shooting pains in different parts of the body. Marked anæsthesia, motor weakness or even complete loss of motor power are sometimes present, but there is no evidence of organic disease in the cord or the affected muscles; the disproportion between the subjective and objective symptoms, and the fact that marked fluctuations in the intensity of the symptoms occur, are highly characteristic features. The points of differential distinction between the two conditions are shown in the following table:—

The differential diagnosis of chronic meningitis and spinal irritation.

	Chronic Meningitis.	Spinal Irritation.
Age and Sex.	May occur in either sex, but most common in young males.	Occurs almost exclusively in young females.
Mode of Onset.	Usually very gradual; may follow an acute attack, the onset is then ushered in by fever	May be gradual, but often sudden, the onset is not then attended by fever.
Hyperæsthesia over the Spine.	Seldom very prominent.	A very marked and characteristic feature.
Stiffness of the Spine.	Usually present.	Does not occur.
Character and Course of the Symptoms.	The pains, spasms, etc., are localised; and do not vary much in their distribution and intensity. Symptoms indicative of organic disease, such as local muscular atrophy, may be present.	Marked fluctuations in the character and intensity of the symptoms occur; and there is a striking contrast between the severity of the subjective symptoms and the objective appearances. There are no signs of organic disease of the cord.
Other Signs of Hysteria.	Very seldom observed, and then only accidental complications. The mental condition is natural.	Common; psychical alterations, such as occur in hysteria, are frequent.
Uterine and Ovarian Functions.	Usually quite normal.	Often deranged.

PROGNOSIS.—The prognosis is uncertain; in most cases the condition is tedious and difficult to cure; the opinion must be chiefly guided by the cause of the attack, its severity, the presence or absence of organic changes in the spinal cord, and the general condition of the patient.

TREATMENT.—The same measures which have been recommended for the treatment of the acute myelitis after the acute symptoms have subsided (see page 246), are to be adopted.

PACHYMENINGITIS SPINALIS.

Inflammation, commencing in and chiefly confined to the dura mater, is usually limited in distribution, and is very generally chronic. In some cases, the osseous surface of the membrane and the connective tissue network, which lies between the dura and the bones of the vertebral canal, are chiefly involved; in others the inner surface of the membrane is most affected; the terms pachymeningitis *externa* and pachymeningitis *interna* have been given to these two conditions respectively.

PACHYMENINGITIS EXTERNA.

Inflammation, commencing in the outer surface of the dura mater, and in the connective tissue which lies between the dura and the bones of the spinal column, is a secondary condition which results from disease of the vertebræ or some other external source of irritation, such as an abscess or deep bed-sore. The inflammatory process, which is very generally chronic, and limited in distribution, extends to the inner surface of the dura, and in some cases invades the arachnoid and pia. The most characteristic pathological feature is the extensive exudation; the affected portion of the dura is enormously thickened; the nerve roots passing through it, and the cord itself, are compressed by the exudation products. Localised pain in the back is generally present, but the most characteristic symptoms are those which result from irritation and compression of the anterior and posterior nerve roots (hyperæsthesia and muscular spasms, anæsthesia, paralysis, muscular atrophy, and absence of the reflexes in limited areas of the body); and from slow compression of the

cord (impairment of motion and sensation below the level of the lesion),[1] together with the symptoms of the primary lesion (Pott's disease of the vertebræ, etc.).

DIAGNOSIS.—The condition has to be distinguished from other forms of meningitis, and from the other conditions which may cause slow compression of the cord; in many cases the diagnosis resolves itself into the diagnosis of Pott's disease of the vertebræ, for that is by far the most frequent cause of the condition which we are now considering.

PROGNOSIS.—The prognosis depends upon the nature of the primary affection, and upon the condition of the cord itself when the patient comes under observation.

TREATMENT.—*The objects of treatment are:—*

1. To remove and cure the primary affection. Where there is Pott's disease of the vertebræ, the appropriate treatment for that condition must be adopted.

2. To promote absorption of the inflammatory products, and to relieve the cord from the compression to which it is subjected. To carry out this indication, counter irritation by blisters, iodine, or the actual cautery; the internal administration of iodide of potassium; and the application of a weak constant current to the spine at the seat of the lesion, are the chief means which we employ. Where the inflammation of the membranes is secondary to injuries or disease of the bones, and where there is any reason to suppose that the compression of the cord is due to displacement of the vertebræ, extension by means of weights or Sawyer's jacket, should be had recourse to.

3. To allay the chronic myelitis which follows the compression of the cord. To carry out this indication, the measures which have been recommended for the treatment of chronic myelitis are to be adopted. (See page 252.)

PACHYMENINGITIS INTERNA HÆMORRHAGICA.

PATHOLOGY AND ETIOLOGY.—This condition is almost invariably associated with a similar affection of the cerebral

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[1] See also Chapter II., 43, 44, 45, and 46.

dura mater. In fatal cases, the internal surface of the membrane is seen to be covered by a thick layer of soft, exudation matters which are partly organised, and in which numerous thin walled vessels are found; the exudation which may be limited in distribution, or scattered over an extensive surface of the dura, is usually of a rusty red or brown colour, owing to the presence of extravasated blood; fresh blood may be present in the meshes of the exudation, or on the surface of the cord; and large cysts containing clots of blood in various stages of absorption, are not uncommon. The condition, like hæmatoma of the dura mater is usually seen in persons suffering from *general paralysis of the insane.* Excess in alcohol is thought by some to produce it; and a traumatic form has also been described.

Symptoms.—The symptoms are usually complicated with those of hæmatoma of the cerebral dura. The spinal symptoms are those of a chronic and ill-defined meningitis, such as pains in the back, some stiffness in the spine, slight indications of motor and sensory irritation, and of motor and sensory impairment. It is important to remember that sudden extravasation of blood from the thin walled vessels in the inflammatory exudation may occur; and that symptoms characteristic of meningeal hæmorrhage may be developed in consequence.

Diagnosis.—The condition should be suspected when symptoms of chronic meningitis are associated with the symptoms of cerebral hæmatoma; if, in addition, sudden exacerbations of the spinal symptoms every now and again occur, a diagnosis of pachymeningitis interna hæmorrhagica may be ventured upon.

PACHYMENINGITIS INTERNA HYPERTROPHICA.

This condition is a chronic inflammation of the inner surface of the dura; the great characteristic of which is the enormous thickening and production of cicatricial fibrous tissue which take place. The affection has been described by Professor Charcot under the name of *pachyméningite cervicale hypertrophique.* The morbid process affects the cervical region of the membranes, and usually extends in a ring

round the cord; the pia and arachnoid become involved, as shown in figure 147; the nerve roots arising from the cord at the seat of the brain are first irritated and then destroyed; and the cord itself is subjected to slow compression.

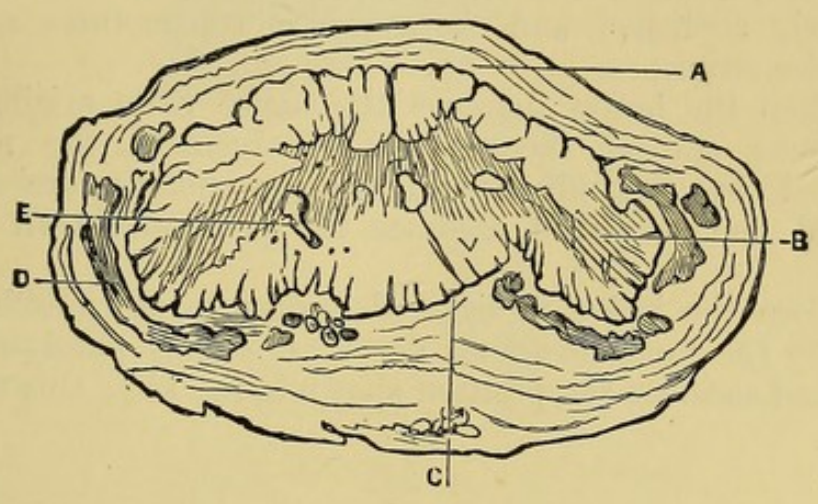


FIG. 147.

Transverse section of the cervical enlargement of the spinal cord in a case of Hypertrophic Cervical Pachy-meninigitis. (After Joffroy.)

A, Hypertrophied dura mater. B, Nerve roots traversing the thickened meninges. C, Pia mater confounded with the dura mater. E, Section of two canals newly excavated in the grey substance.

Charcot divides the affection into—(1) a stage of irritation, which lasts for two or three months; (2) a stage of paralysis and atrophy.

The symptoms of the *first stage* are due to irritation of the anterior and posterior nerve roots which pass through the affected portion of the membranes; they consist of:—shooting pains, severe in character, and referred to the sensitive areas of the affected roots (back of the neck, shoulders, arms, or upper part of the thorax); hyperæsthesia; muscular twitchings and spasms in the muscles supplied by the irritated anterior nerve roots (rigidity of the neck, upper parts of the spine, or muscles of the upper extremities); together with symptoms of slight sensory and motor impairment. Trophic

disturbances in the skin (herpetic eruptions, etc.) are also sometimes seen.

These symptoms slowly increase, and *the second stage* is reached; it is characterised by anæsthesia, paralysis, and atrophy in the sensitive and muscular areas of the compressed nerve roots; the Faradic excitability of the affected muscles is slowly abolished, and characteristic contractures and deformities occur.

When the lesion involves the nerve roots arising from the lower end of the cervical enlargement, the muscles supplied by the ulnar and median nerves are chiefly affected, and the hand assumes the position shown in fig. 148.

When the lesion is situated higher up, the muscles supplied by the musculo-spinal nerve are chiefly paralysed, and the hand assumes the position shown in fig. 149; this was the

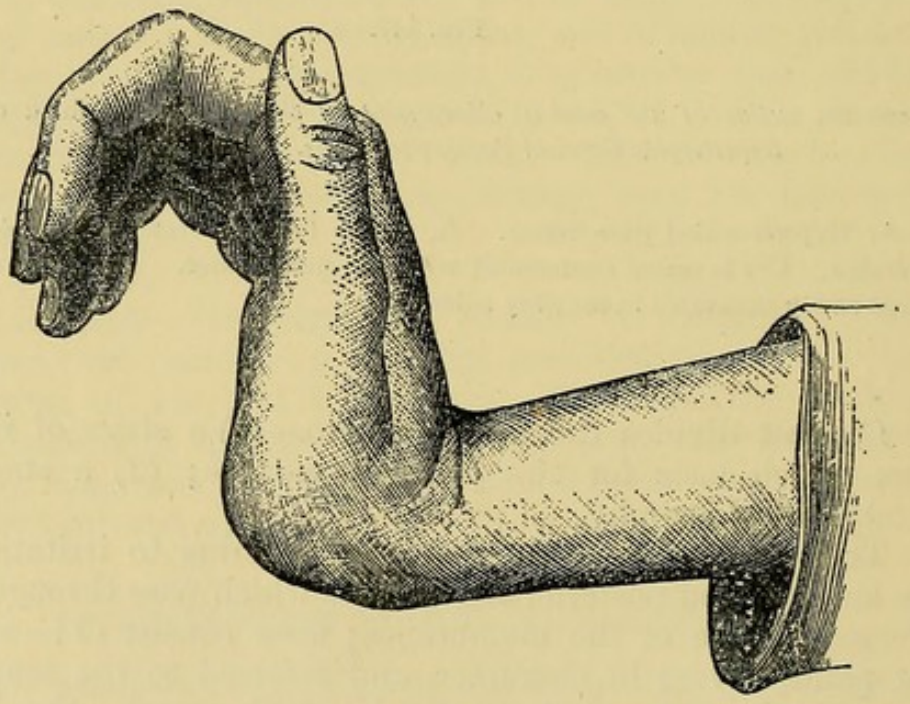


FIG. 148.

The position of the hand in Pachymeningitis Cervicalis Hypertrophica, affecting the lower part of the cervical enlargement. (*After Charcot.*)

condition in a case quoted by Dr Ross,[1] which was under the care of Dr Leech.

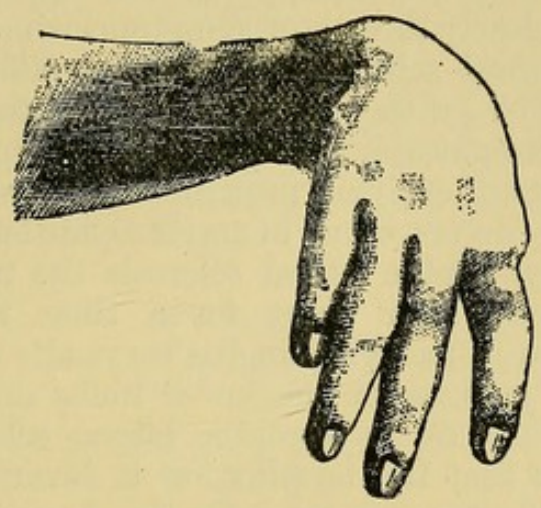


FIG. 149.

The position of the hand in Pachymeningitis Cervicalis Hypertrophica, affecting the upper part of the cervical enlargement. (*After Ross.*)

Transverse myelitis at the seat of the compression, and secondary descending degeneration below the lesion, gradually develop, and a condition of spastic paraplegia, together with the sensory disturbances and other symptoms which have been described as characteristic of a transverse myelitis (see p. 58), slowly supervenes.

The duration of the case is always long; in some cases, the termination is in death, which may result from some intercurrent complication, or from cystitis, bed-sores, etc.; in others the morbid process is arrested; the paralysis and rigidity in the lower extremities may become less, but the contractures and deformities of the upper limbs, in the great majority of cases, remain.

DIAGNOSIS.—The condition may at first sight be mistaken for amyotrophic lateral sclerosis or progressive muscular atrophy.

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[1] *Diseases of the Nervous System*, vol. ii. p. 388.

The differential diagnosis of pachymeningitis cervicalis hypertrophica and amyotrophic lateral sclerosis.—The motor symptoms (rigidity and atrophy in the upper extremities) resemble very closely the motor symptoms which characterise amyotrophic lateral sclerosis, but the two affections are at once distinguished by the facts, (1) that in pachymeningitis cervicalis hypertrophica, symptoms of sensory irritation (severe pains and hyperæsthesia) are prominent features in the first stage, while they do not occur in amyotrophic lateral sclerosis; (2) that in amyotrophic lateral sclerosis the muscles of the lower extremities, after being for a time rigid, undergo atrophy, whereas in pachymeningitis cervicalis hypertrophica, atrophy in the muscles of the lower limbs does not occur; (3) that the course of amyotrophic lateral sclerosis is much more acute, and that the termination is invariably in death, the fatal result being very generally due to extension of the lesion to the medulla; whereas in pachymeningitis cervicalis hypertrophica the progress is always slow, an incomplete recovery is not uncommon, and the morbid process rarely, if ever, invades the medulla oblongata. (See also table, p. 277.)

The differential diagnosis of pachymeningitis cervicalis hypertrophica and progressive muscular atrophy.—In the second stage, the condition may, at first sight, be mistaken for progressive muscular atrophy; but it is at once distinguished; (1) by the pronounced sensory disturbances; (2) by the fact that the atrophy was preceded by a stage of rigidity and tension; and (3) by the spastic paraplegia of the lower extremities.

The well-marked symptoms of sensory and motor irritation in the sensitive and muscular areas of certain nerve roots, together with the symptoms and signs of transverse myelitis, show most conclusively, that the lesion is extra-medullary.

It is not always easy to make the next step in the diagnosis, *i.e.*, to determine the pathological character of the lesion, for it may be difficult or impossible to decide whether the symptoms are due to—(*a*) Pott's disease of the vertebræ and associated pachymeningitis externa; (*b*) extra-medullary tumours arising from the bones or membranes; or (*c*) pachymeningitis cervicalis hypertrophica. In attempting to decide this question, attention must be directed to the following

Table showing the points of differential distinction between Amyotrophic Lateral Sclerosis, Primary Lateral Sclerosis, Progressive Muscular Atrophy, and Pachymeningitis Cervicalis Hypertrophica.

	Amyotrophic Lateral Sclerosis.	Primary Lateral Sclerosis.	Progressive Muscular Atrophy.	Pachymeningitis Cervicalis Hypertrophica.
Mode of onset.	Usually commences with loss of power in the muscles of the upper extremities.	Usually commences with motor weakness and rigidity of muscles of lower extremities.	Usually commences with muscular wasting of interossei and thenar muscles of one hand.	Commences with severe pains and hyperæsthesia in the upper extremities, or in the head and neck. Muscular spasms, rigidity and contractures follow; paralysis, muscular atrophy, and anæsthesia occur later.
Character of the motor symptoms.	Loss of motor power (paresis), followed by atrophy *en masse*, fibrillary twitchings, and rigidity.	Paresis and rigidity, with increase of the reflexes. No muscular atrophy.	The atrophy is the first event, the loss of power follows, and is in direct proportion to the muscular wasting. There is never rigidity.	Symptoms of motor irritation first occur; symptoms of motor destruction (paralysis, rapid atrophy, etc.), follow.
Condition of sensibility.	Normal.	Normal.	Normal.	Profound sensory disturbances due to pressure on the posterior nerve roots.
Mode of extension.	After 4 to 12 months it extends to the lower extremities; spastic paraplegia, and finally muscular atrophy are developed. In the third stage it extends to the medulla.	Occasionally involves the upper extremities. Never extends to medulla.	Extends to the homologous muscles on the opposite side, which slowly atrophy, and ultimately become paralysed. Other muscles and groups of muscles are next affected; may finally extend to the medulla.	The lower extremities may become affected by spastic paraplegia, but do not atrophy. Never extends to the medulla.
Duration.	Course comparatively rapid (1 to 3 years.)	Very chronic (10, 20, 30 years or more).	Very chronic (8, 10, 15 years).	Chronic.
Termination.	Death.	Not fatal *per se;* death usually results from complications.	Death usually results: it may be due to complications, or to extension of the morbid process to the medulla.	Often ends in recovery, which may be complete; but, as a rule, some atrophy and loss of motor power remain.

points:—1. The condition of the spinal column. 2. The age of the patient. 3. The condition of the temperature. 4. The presence of lesions in other organs, indicative of scrofula, cancer, etc.

The points of differential distinction are given in the following table:—

The differential diagnosis of Pachymeningitis Cervicalis Hypertrophica, Pott's Disease of the Vertebræ, and Extra-medullary Tumours.

	Pachymeningitis Cervicalis Hypertrophica.	Pott's Disease of the Vertebræ.	Extra-Medullary Tumours.
Age.	(?) Middle-aged.	Generally young.	Any age, but often past middle age.
The condition of the spinal column.	Neck may be stiff from rigidity of muscles, but no tenderness on pressure or percussion of the vertebral spines; no curvature.	Great tenderness on pressure, percussion, or movement of the vertebræ; often curvature of the spine; there may be a superficial abscess at the seat of the lesion.	In some cases of cancer there is swelling of the bone, but no great tenderness on percussion or movement. In the case of tumours springing from the membranes, the spinal column is quite normal.
The temperature.	Normal.	There may be irregular fever of a suppurative type.	Normal or subnormal.
General condition.	Usually impaired.	Profoundly affected; emaciation, rigors, and sweatings often prominent.	May be a cancerous cachexia; in many cases nothing distinctive.
Distinctive associated lesions.	None.	Scrofulous disease of the glands, bones, or lungs common.	May be cancerous, sarcomatous, or hydatid tumours in some other part of the body

TREATMENT.—The same treatment, which has been recommended for pachymeningitis externa (see p. 271), particularly counter-irritation at the seat of the lesion, and the internal administration of iodide of potassium, is to be adopted.

INTRA- AND EXTRA-MEDULLARY HÆMORRHAGE.

Blood is occasionally, though very rarely, extravasated into the substance of the cord, or into the spinal membranes; the former condition may be termed *intra-medullary*, the latter *extra-medullary* hæmorrhage.

INTRA-MEDULLARY HÆMORRHAGE.

Synonym.—SPINAL APOPLEXY.

PATHOLOGY AND ETIOLOGY.—The great causes of intra-medullary hæmorrhage are (1) alterations in the vascular walls, which weaken their resistence and (2) increased blood pressure. In some cases the hæmorrhage occurs independently of any previous cord lesion; but in many the extravasation is, as it were, an accidental complication occurring in the course of some other morbid condition. Extravasations of the latter description are not uncommon in acute myelitis (hæmorrhagic myelitis), and blood is occasionally poured out from the thin walled and dilated vessels of a soft glioma. Hæmorrhage into the substance of the cord is probably sometimes due to traumatic violence, such as a direct blow on the spine, or a fall on the feet. The condition may occur at any period of life, but is most frequent between the ages of ten and twenty. In this respect spinal apoplexy differs very markedly from cerebral hæmorrhage, which is much more frequent in old people; the earlier occurrence of spinal apoplexy is probably explained by the fact, that in many cases the bleeding is not the primary condition, but is an accidental event occurring in the course of some other morbid process. The amount of blood which is extravasated is usually small, the clot seldom being larger than an almond; and the bleeding is almost always confined to the central grey matter. The blood ploughs up and destroys the nerve structures, and the function of the affected part is immediately interrupted; should the patient survive for a sufficiently

long period, inflammatory changes occur in the neighbourhood of the clot, and secondary degenerations are established.

ONSET, SYMPTOMS, AND COURSE.—In some cases, the extravasation takes place rapidly, and the patient is struck down with immediate paralysis of a paraplegic type; severe pains in the back may be experienced at the time of onset, but there is no loss of consciousness. In other cases, the blood is poured out gradually, and it may be some hours before the paralysis becomes fully developed. The motor, sensory, reflex, vesical, rectal, and other derangements are identical with those which result from an acute transverse myelitis, in fact a large hæmorrhage into the substance of the cord is a typical example of an acute, total, transverse lesion. (See § 37, fig. 71).

DIAGNOSIS.—Hæmorrhage into the substance of the cord is readily distinguished from cerebral hæmorrhage by the fact that there is no disturbance of the cerebral functions, and by the paraplegic distribution of the paralysis. The only conditions with which it is at all likely to be confounded are polio-myelitis anterior acuta, acute myelitis, and extra-medullary hæmorrhage.

The differential diagnosis of intra-medullary hæmorrhage, and polio-myelitis anterior acuta presents no difficulty; the points of distinction are given in the following table:—

	Intra-Medullary Hæmorrhage.	**Polio-Myelitis Anterior Acuta.**
ONSET.	Always very rapid, may be instantaneous; it is not accompanied by fever.	It is accompanied by fever. Never instantaneous.
CONDITION OF SENSATION.	Often acute pains in the back at the time of onset; anæsthesia which may be total, below the lesion, follows.	No sensory derangement.
CONDITION OF THE BLADDER AND RECTUM.	Often paralysed; when the hæmorrhage involves the reflex centres, paralysis of the sphincters, ammoniacal urine, and cystitis are developed	Normal.
TROPHIC ALTERATIONS IN THE SKIN.	Large bed-sores frequent.	No trophic alterations in the skin.

The differential diagnosis of intra-medullary hæmorrhage and acute myelitis has already been considered (see page 245).

The differential diagnosis of intra- and extra-medullary hæmorrhage should not present any great difficulty. The important fact to remember in distinguishing the two conditions is, that in intra-medullary hæmorrhage, as in acute myelitis, symptoms of motor and sensory *impairment* predominate; whereas in *extra-medullary* hæmorrhage, as in acute meningitis, symptoms of motor and sensory *irritation* are relatively much more marked. The chief points of distinction between intra- and extra-medullary hæmorrhage are given in the following table:—

Intra-medullary Hæmorrhage.	Extra-medullary Hæmorrhage.
Pain in the back may occur at the onset, but soon disappears; shooting pains in the limbs and hyperæsthesia are not prominent, or do not occur.	Pains in the back, shooting-pains in the limbs, and hyperæsthesia are strongly marked, and generally continue for some time.
Muscular cramps and spasms may occur at the very commencement. Profound paralysis and anæsthesia are very rapidly developed.	Muscular cramps, spasms, and rigidity are very prominent features; paralysis and anæsthesia are seldom very marked, and appear late.
Paralysis of the sphincters, ammoniacal urine, and cystitis; or obstinate retention and constipation are very frequent.	The functions of the bladder and rectum are not seriously interfered with; ammoniacal urine does not occur.
Trophic derangements of the skin producing large bed-sores are common.	Bed-sores do not occur.
The affection is very serious; it often proves fatal; and recovery is always incomplete.	The affection is much less serious; it is often recovered from; the cure may be complete.

Prognosis.—The prognosis depends upon the size and position of the clot; other things being equal, a large clot is much more serious than a small one; a hæmorrhage into the upper cervical region is rapidly fatal owing to the respiratory paralysis which it produces; a hæmorrhage into the lumbar region, which causes paralysis of the sphincters, is more serious than a hæmorrhage into the dorsal region; cases in which large bed-sores are quickly developed invariably prove fatal.

TREATMENT.—In the earlier stages we must endeavour to arrest the bleeding in the manner described on page 165. The subsequent treatment of the case is the same as the treatment of acute myelitis (see page 246).

EXTRA-MEDULLARY HÆMORRHAGE.

Synonym.—SPINAL MENINGEAL HÆMORRHAGE.

PATHOLOGY AND ETIOLOGY.—Effusion of blood into the spinal membranes is, like intra-medullary hæmorrhage, extremely rare. The conditions which give rise to it are:—(*a*) traumatic injuries, such as blows on the spine or violent concussions; (*b*) pachymeningitis hæmorrhagica; (*c*) the rupture of aneurisms of the thoracic or abdominal aorta into the spinal canal; (*d*) purpura, scurvy, and other conditions in which a strong tendency to hæmorrhage exists; and finally, blood, which has been poured out into the cranial cavity, may find its way into the spinal canal.

In some cases the blood is poured out between the dura and the bones of the vertebral canal; in others between the dura and the arachnoid. The amount of blood varies considerably in different cases; it is sometimes sufficiently copious to ensheath the whole cord or dura; the blood may be partly fluid, but is generally clotted and dark coloured.

ONSET, SYMPTOMS, AND COURSE.—The extravasation may occur with instantaneous rapidity, and the symptoms are then suddenly developed; in some cases the effusion is more gradual. The initial symptoms are characteristic, and are those which attend any severe meningeal irritation, viz., pain in the back, shooting-pains in the limbs or trunk, muscular cramps, twitchings, spasms, and rigidity. Symptoms of motor and sensory impairment are present, but the paralysis and anæsthesia are seldom great. The stage of reaction may be attended with some febrile disturbance.

PROGNOSIS.—The Prognosis is much more favourable than in the intra-medullary variety; in many cases the symptoms gradually abate, and the patient may eventually get quite well. A large hæmorrhage into the upper part of the spinal canal may prove immediately fatal. Where the hæmorrhage

is sufficiently copious to compress the cord, myelitis may be developed, and death may result from exhaustion, bed-sores, or cystitis.

DIAGNOSIS.—The diagnosis of a well-marked case should present no difficulty. The characteristic features are the sudden occurrence of symptoms of meningeal irritation and the absence of fever at the commencement of the attack.

A spinal meningeal hæmorrhage is at once distinguished from an *intra-cranial hæmorrhage* by the fact that the symptoms are entirely spinal.

Acute meningitis resembles meningeal hæmorrhage, inasmuch as symptoms of spinal irritation are rapidly developed in both conditions; the distinctive points are: (*a*) that in meningeal hæmorrhage the onset may be instantaneous, and is usually much more rapid than in acute meningitis; and (*b*) that meningeal hæmorrhage is not ushered in by fever.

The points of differential importance between meningeal hæmorrhage and *hæmorrhage into the substance of the cord*, have already been considered. (See page 281.)

TREATMENT.—In the earlier stages the treatment recommended for the arrest of an intra-medullary hæmorrhage (see page 165) is to be adopted. Subsequently the case is to be treated as a case of acute meningitis. (See page 266).

INTRA- AND EXTRA-MEDULLARY TUMOURS.

New formations rarely originate in the cord itself, but they not unfrequently spring from the bones and membranes.

INTRA-MEDULLARY TUMOURS.

MORBID ANATOMY.—Gliomata, tubercular nodules, gliosarcomata, and syphilitic gummata have been found in the substance of the cord; they are usually of small size, but 'elongated tumours which have stretched, in various degrees of thickness, throughout the whole cord, from the conus medullaris to the medulla oblongata' have been met with.—(Erb.)

Intra-medullary tumours are usually solitary. They may invade any part of the transverse section, and may originate

either in the white or in the grey matter. The gliomatous tumour is said to occur most frequently in the cervical, while the tubercular tumour is more apt to invade the lumbar enlargement.—(Hayem.)

ETIOLOGY.—A blow on the spine or other injury seems occasionally to be the exciting cause, but in most cases the etiology is obscure.

ONSET, SYMPTOMS AND COURSE.—In some cases the symptoms develop very gradually, in others paraplegia is somewhat suddenly produced. These differences in the mode of onset depend upon the rapidity of growth of the tumour and the effects it produces upon the nervous structures. A tumour which is growing slowly may for a time simply displace the nerve elements, and cause atrophy of the parts in its immediate neighbourhood; in such cases the condition is either latent, or the symptoms are ill-defined, and consist of slight and localised derangements of motion or sensation, localised muscular atrophy, etc. Sooner or later in the case of tumours of slow growth, and at an early period in the history of tumours which develop rapidly, inflammatory changes are established in the neighbourhood of the tumour, and the condition of matters then practically corresponds to a focal or transverse myelitis, the symptoms of which are described on page 58. In some cases, blood is extravasated from the vessels of a soft glioma, and the symptoms of spinal apoplexy (see p. 280) are developed.

The exact character of the symptoms depends therefore upon the position of the tumour, its size, the rapidity of its growth, and especially upon the nature of the secondary changes in the substance of the cord (compression, atrophy, myelitis) which it produces.

DIAGNOSIS.—The diagnosis is attended with extreme uncertainty. A long premonitory stage of ill-defined symptoms, followed by indications of compression of the cord, transverse myelitis, or spinal apoplexy, should suggest the presence of an intra-medullary tumour, but a positive opinion can seldom be given.

PROGNOSIS.—The Prognosis depends upon the pathological

nature of the tumour; in syphilitic cases recovery, which is usually incomplete (some paralysis remaining), is not uncommon; tubercular tumours are perhaps sometimes recovered from; but in the great majority of (non-syphilitic) cases the termination is fatal.

TREATMENT.—Iodide of potassium should first be given; if this fails, arsenic may be prescribed; in tubercular cases cod-liver oil, lacto-phosphate of lime, and general tonics should be administered.

EXTRA-MEDULLARY TUMOURS.

MORBID ANATOMY.—Tumours springing from the bones, membranes, or nerve roots, are more common than new formations of intra-medullary origin. The following are the chief varieties which have been met with:—Inflammatory formations, cancers, and sarcomatous growths, arising from the bones: sarcomatous, scrofulous, fibrous, myxomatous, syphilitic, fatty, cartilaginous growths, and hydatid cysts, springing from the membranes; sarcomatous, myxomatous, gliomatous, and fibrous tumours, springing from the nerve roots. Extra-medullary tumours are usually localised, and in most cases solitary, though occasionally, as in a case figured by Lancereaux,[1] numerous separate nodules have been found.

ETIOLOGY.—It is seldom that a distinct cause for the condition can be ascertained; in some cases, there is a history of traumatic injury to the spine; and, in the case of cancerous sarcomatous, and scrofulous tumours, the extra-medullary growth may be secondary to similar disease in some other part of the body.

ONSET, SYMPTOMS, AND COURSE.—The characteristic symptoms of extra-medullary tumours result from irritation of the membranes and pressure upon the nerve roots; and in contrasting extra- and intra-medullary growths, the same general principle which was insisted upon in treating of intra- and extra-medullary inflammations (myelitis and meningitis) must be kept in view, viz., that *intra*-medullary growths give rise

[1] *Atlas of Pathology*, plate 45, fig. 3.

chiefly to symptoms of motor and sensory *impairment*, while the characteristic symptoms of *extra*-medullary growths are *irritative* in character.

The *premonitory stage*, which is usually long, not unfrequently lasting for years, is characterised by localised pain in the back, and shooting pains, hyperæsthesia, slight sensory and motor impairment, in the districts of the nerve roots which are compressed by the tumour. The exact distribution of these symptoms depends upon the position of the tumour and the number of nerve roots which are affected by it; in some cases the nerve roots of one side only are involved, and the symptoms are unilateral in their distribution.

After the primary stage has lasted for a longer or a shorter period, the cord itself becomes compressed, and other symptoms, which I have described on page 67, are added. Here again the character and distribution of the symptoms depend upon the position of the tumour and its relationship to the different columns of the cord; where the compression affects the cervical enlargement, as is seen to be the case in the lithograph (fig. 150), all four extremities may be paralysed; where the tumour is situated below the cervical enlargement, the upper extremities of course escape; a tumour which compresses one side of the cord produces the symptoms of a unilateral lesion (see page 61); in some cases the pressure chiefly affects the anterior surface of the cord, and the chief symptoms are motor; in others the posterior columns are most implicated, and sensory derangements are prominent. Compression of the cord ultimately produces inflammatory softening, which in its turn is followed by secondary degenerations (see fig. 151). The myelitis is sometimes developed with extreme rapidity, and sudden paraplegia may thus be produced. The exact nature of the symptoms in the second stage will be more easily understood by referring to the description which has been previously given of slow compression of the cord, transverse myelitis, and secondary descending degeneration.

After the second stage is reached the case usually progresses more rapidly, and the patient may die from bed-sores, cystitis, paralysis of the muscles of respiration, lung complications, or any of the other conditions which give rise to death in cases of myelitis (see p. 244). Where the tumour involves the dorsal region of the cord, cystitis and bed-sores

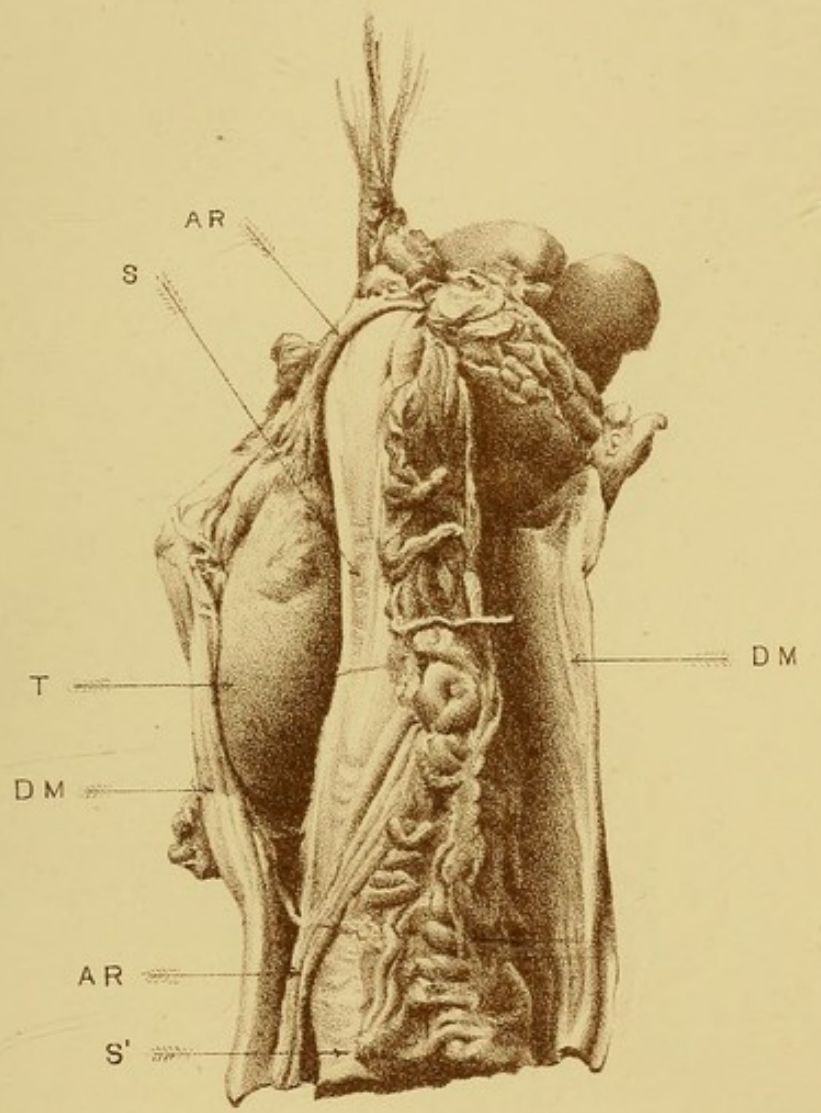


FIG. 150.

Compression of the cervical portion of the spinal cord by a tumour springing from an anterior nerve root. One and a half times the natural size.

S Compressed portion of the spinal cord; S′ Cervical enlargement below the lesion, it is of natural size; T. The tumour; A.R. Anterior nerve root from which the tumour springs; A.R.′ healthy anterior nerve root arising from the cord below the lesion; DM. dura mater.

The cord is suspended by a string attached to it above the tumour.

I am indebted to Dr Banham of Sheffield, and Dr Goyder of Newcastle, for the preparation from which the drawing was made.

McLagan & Cumming, Lith. Edin.

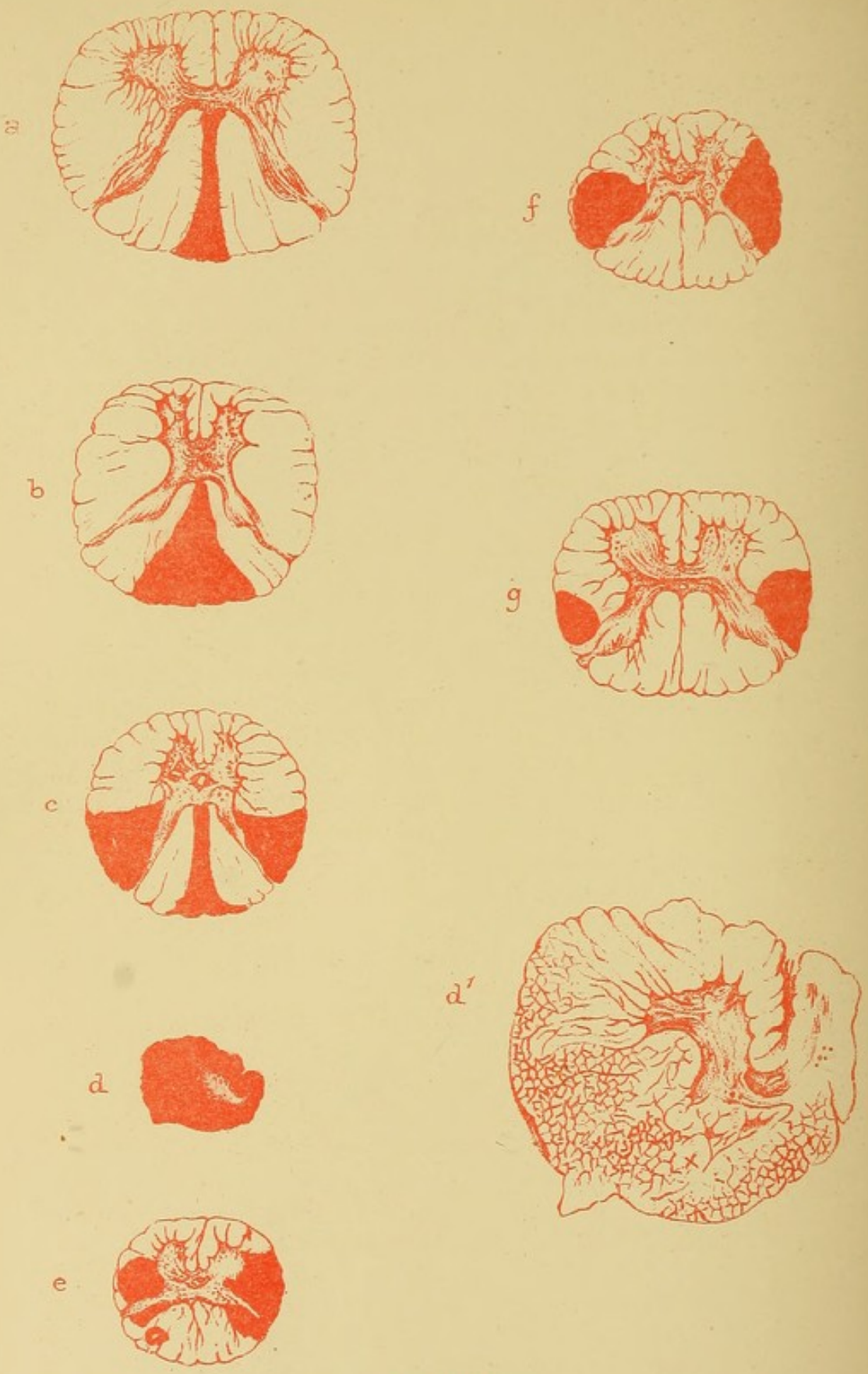


FIG. 151.

A series of sections through the Spinal Cord in a case of slow Compression, from Pott's disease of the vertebræ, showing secondary ascending and descending degenerations. (After Charcot.)

a, Cervical enlargement; b, upper dorsal region; c, mid-dorsal region; d, compressed part; d′, the same more highly magnified; e, lower dorsal region; f, upper lumbar region; g, lumbar enlargement.

The degenerated parts are deeply stained.

McLagan & Cumming, Lith. Edin.

are seldom present, and the patient may live for a considerable time after the paraplegia has been developed; but in the vast majority of cases the termination is fatal.

DIAGNOSIS—The diagnosis is sometimes easy, but often uncertain or impossible. Symptoms of meningeal and nerve-root irritation (viz., pain in the back, and irritative sensory and motor phenomena in certain nerve districts), together with symptoms of compression of the cord, demonstrate the presence of an extra-medullary lesion; and we may conclude with considerable probability that the lesion is a tumour, when:

(1.) The symptoms have developed slowly, and are not attended with fever.

(2.) The lesion appears to be of limited vertical extent.

(3.) There is no evidence of Pott's disease of the spine, and no history of a dislocation or fracture of the vertebræ.

When in addition to this evidence the presence of a cancerous, scrofulous, hydatid, or syphilitic growth can be demonstrated in some other part of the body, the diagnosis of extra-medullary tumour may be made with great certainty.

The position of the tumour and its vertical extent are to be ascertained by observing—

1. The exact distribution of the irritative phenomena which result from pressure upon the anterior and posterior nerve roots. The particular nerve roots which are implicated can in this manner be ascertained.

2. The condition of the reflexes and the trophic condition of the muscles.

3. (In the case of a unilateral lesion of the cord *e.g.*, a tumour pressing upon one side of the cord), the position and vertical extent of the anæsthetic band on the same side as the lesion. (See fig. 74.)

4. The exact position of the pain in the back. The pain which results from pressure on the membranes, or from cancer of the vertebræ, may be strictly localised, and so may afford corroborative evidence as to the exact position of the tumour. Again a local alteration of the spinal columns, such as an external tumour or thickening of the bones, may indicate the position of the internal tumour. It is important to remember that the spinal cord terminates at the lower border of the first lumbar vertebra; and that the greater number of the

spinal nerves leave the spinal canal considerably below their attachment to the cord. Hence a tumour springing from the bones or membranes, say a tumour of the twelfth dorsal vertebra, will not compress the segment of the cord from which the twelfth dorsal nerve arises, but the segments corresponding to the second, third, and fourth lumbar nerves. So again, a tumour springing from the third lumbar vertebra will not press upon the cord at all, but will only involve the nerve roots of the cauda equina.

The pathological character of the tumour is to be determined by observing—

1. The presence of associated diseased conditions which are likely to give a clew to the nature of the lesion. The presence, for example, of malignant disease in the liver or some other part of the body would be a sufficient reason for supposing the intra-spinal tumour to be malignant too.

2. The constitutional peculiarities and tendencies of the individual.

3. The family history—a strong hereditary history of cancer in the absence of any direct evidence would suggest a malignant growth.

4. The effect of treatment. In a supposed syphilitic case, rapid improvement under iodide of potassium and mercury would confirm the diagnosis.

TREATMENT.—The same measures which have been recommended for the treatment of *intra-medullary* tumours are to be adopted; and the possibility of removing the growth considered. Where the conditions mentioned on page 165 are present, an operation would, I think, be justifiable.

Dr Byrom Bramwell will be obliged to any of his readers who will send him spinal cords of typical, interesting, or rare cases. Microscopical sections will be sent back in return. The preparation (suspended in bichromate of ammonium in the manner described below), should be despatched *as soon as possible* after removal.

Directions for the Removal and Preservation of the Spinal Cord.

The body having been laid on its face, and a large block inserted under the thorax, an incision is to be made, in the middle line behind, from the occiput to the sacrum. The skin and muscles are then to be reflected from each side of the vertebræ.

The laminæ of the two or three dorsal vertebræ, corresponding to the point of greatest convexity of the spinal column, are next sawn across. With a little manipulation (the spinous processes being grasped in the left hand), and by the help of a scalpel, the vertebral arches, which have been sawn across, can now be removed, and the posterior surface of the dura mater exposed.

The laminæ of the remaining vertebræ, both above and below those which have been removed, are then to be *cut across with a powerful pair of bone pliers*, and the entire length of the spinal canal exposed. *The dura mater is to be left undivided.* The dura and cord are to be cut across close to the occiput, the dura laid hold of by forceps, and the cord gently raised out of the spinal canal. The spinal nerves are then to be divided, one by one, outside the dura, and the entire cord, *including the filum terminale*, removed.

The dura is now slit up by means of probe-pointed scissors (the greatest care being taken to avoid any injury to the cord), and the naked eye appearances noted.

The cord should then be divided, by means of transverse incisions, into three or four equal portions, and *suspended* by means of threads in a wide-necked bottle, containing a four per cent. solution of bichromate of ammonium. (When bichromate of ammonium cannot be obtained, spirit may be used, but it is to be avoided *if possible.*) The preservative fluid should be changed on the second, and again on the sixth day. The preparation should be kept in a cool place.

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