Diseases of the nervous system / by J.A. Ormerod.

Ormerod's Diseases of the Nervous System

THE DISEASES

OF THE

NERVOUS SYSTEM

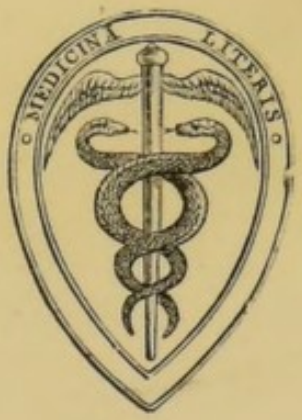
TUDENT'S GUIDE SERIES

THE DISEASES OF THE NERVOUS SYSTEM

BY

J. A. ORMEROD, M.A., M.D. Oxon.

Fellow of the Royal College of Physicians, London; Medical Registrar and Demonstrator of Morbid Anatomy at St. Bartholomew's Hospital; Physician to the National Hospital for the Paralysed and Epileptic, Queen Square, and to the City of London Hospital for Diseases of the Chest, Victoria Park, formerly Fellow of Jesus College, Oxford



LONDON
J. & A. CHURCHILL
11 NEW BURLINGTON STREET
1892

PREFACE.

The subject of nervous disease, attractive though it be, is apt to inspire the beginner with a feeling of dismay. It grows so fast, it has so many roots and branches, it presupposes so much knowledge and demands so much expenditure of time. Thoroughly to grapple with it, he will indeed require much patient clinical work, as well as study of those excellent and elaborate treatises which happily exist. This little volume is offered to him as no substitute for those, but only as an introduction to his work and outline map of territory to be acquired; and should it thus prove to him, perhaps by its very smallness, an encouragement and aid, then it will have served its end.

CONTENTS.

CHAPTER IV.

SYMPTOMS REFERABLE TO THE ORGANS OF SPECIAL SENSE.

CHAPTER V.

SYMPTOMS REFERABLE TO SPECIAL DISTRICTS.

CHAPTER VI.

NEURITIS : ORGANIC SPINAL DISEASES.

CHAPTER VII.

ORGANIC CEREBRAL DISEASE.

CHAPTER VIII.

DISEASES OF WHICH THE ORGANIC BASIS IS NOT KNOWN.

LIST OF ILLUSTRATIONS.

NERVOUS DISEASES.

CHAPTER I.

ANATOMICAL AND PHYSIOLOGICAL INTRODUCTION.

Nerve-cells.—In spite of the complicated functions performed by the nervous system, the histological elements of which it consists are comparatively simple. If we except certain complicated terminal organs, such as the retina, organ of Corti, &c., we shall find that the bulk of it is made up of cells and fibres. The nerve-cells or ganglion-cells vary in size and appearance, some are small and rounded, others larger and bipolar, as in the "vesicular" columns of the cord, others arrow-headed as in the cortex cerebri, others with several processes, as in the anterior grey columns of the cord. The large cells have a distinct nucleus and nucleolus, and often one of the processes is continuous with the "axis cylinder" of a nerve-fibre. Collections of such cells are found in the ganglia of the peripheral nervous system; and cells united by a network of small fibres form the grey matter of the spinal cord, the cerebral ganglia, and the cerebral cortex.

Nerve-fibres.—The essential part of a nerve-fibre is the central filament called the "axis cylinder." There are, however, two varieties of nerve-fibres. In the one the axis cylinder is embedded in a layer of

soft white material, akin to fat in its chemical reactions, which is called the myeline or medullary sheath. Around this again is a fine envelope, bearing on its surface nuclei and called the nucleated sheath of Schwann. At certain intervals the medullary sheath is interrupted, and so the sheath of Schwann approaches the axis cylinder; these intervals are called the nodes of Ranvier. Such is the structure of a white or "medullated" nerve-fibre. The other variety is the non-medullated or grey fibre; it consists simply of an axis cylinder and sheath of Schwann without any intervening myeline. The medullated nerve-fibre is particularly associated with the cerebro-spinal system of nerves, the non-medullated with the sympathetic; there is nevertheless a kind of medullated fibre, distinguished principally by its very small calibre, which probably ranks with the sympathetic nerves.

The function of nerve-fibres is simply to transmit nervous impulses; while cells not only transmit them, but combine, re-arrange, modify, and perhaps produce them. Again, there is this important difference as to the life of cells and fibres: nerve-fibres (at any rate peripheral nerve-fibres) when destroyed can be regenerated; nerve-cells, so far as we know, cannot: and, further, the life of the fibre is in most cases dependent upon the cell with which it is connected, for if the connection be broken or the cell destroyed, then the fibre dies.

Nerve-trunks.—The nervous elements are held together in the peripheral nervous system by envelopes of connective tissue. Not only has each fibre its sheath of Schwann, but the bundles and nerve-trunks have additional connective-tissue sheaths, endo-, peri-, and epi-neurium, which protect and unite them, and convey to them their nutrient vessels. In the nerve-centres, the white fibres lack the sheath of Schwann; the nervous elements lie embedded in a substance called neuroglia, a granular-looking material containing angular cells (cells of Deiters).

General connection of spinal and sympathetic systems.—The system of peripheral nerves consists of two parts, the cerebro-spinal nerves and the sympathetic; of which the former are distributed mainly to the skin and skeletal muscles, the latter to the viscera and blood-vessels. The sympathetic is disposed on either side of the vertebral column in the form of a chain of ganglia, from which nerves run forward to form the large plexuses, cardiac, solar, pelvic, &c., which supply the adjacent viscera and blood-vessels. But it is closely connected with the cerebro-spinal system by the nerve-trunks called the "rami communicantes." This will be seen, if we proceed to consider the general arrangement of a cerebro-spinal nerve. Such arrangement is seen at its simplest in a nerve from the dorsal region.

The nerve commences at its origin from the spinal cord in two roots, anterior and posterior. These are similar in structure except for the fact that upon the posterior root is placed a ganglion. But their function is different, the anterior root is efferent, carrying impulses from the cord; the posterior is afferent, carrying them to the cord. The roots join to form a mixed nerve, in which afferent and efferent fibres are united in a common envelope. This nerve divides into branches, a posterior and anterior, still of mixed function, and going to the skin and muscles of the back, and of the intercostal space respectively. But this is not all, for the "ramus communicans" opens a path by which fibres both come and go between the sympathetic and the spinal cord. Efferent fibres from the spinal cord pass out *viâ* the anterior root down the "ramus communicans" to the sympathetic ganglia. In this part of their course they are medullated fibres, though distinguished by being of a very fine calibre; but after issuing from the sympathetic ganglion for distribution they are found to have lost their myeline sheath, and have become typical grey sympathetic fibres. In a similar manner afferent

fibres ascend from the sympathetic, to join the posterior spinal root and thus enter the spinal cord.

There are certain levels in the cord at which this connection with the sympathetic is best marked. Thus, at the upper cervical region, a large outflow of fibres takes place from the cord to the sympathetic cervical ganglia, and thence to the cardiac plexus; and again in the upper thoracic region to the ganglia whence the splanchnic nerves arise.

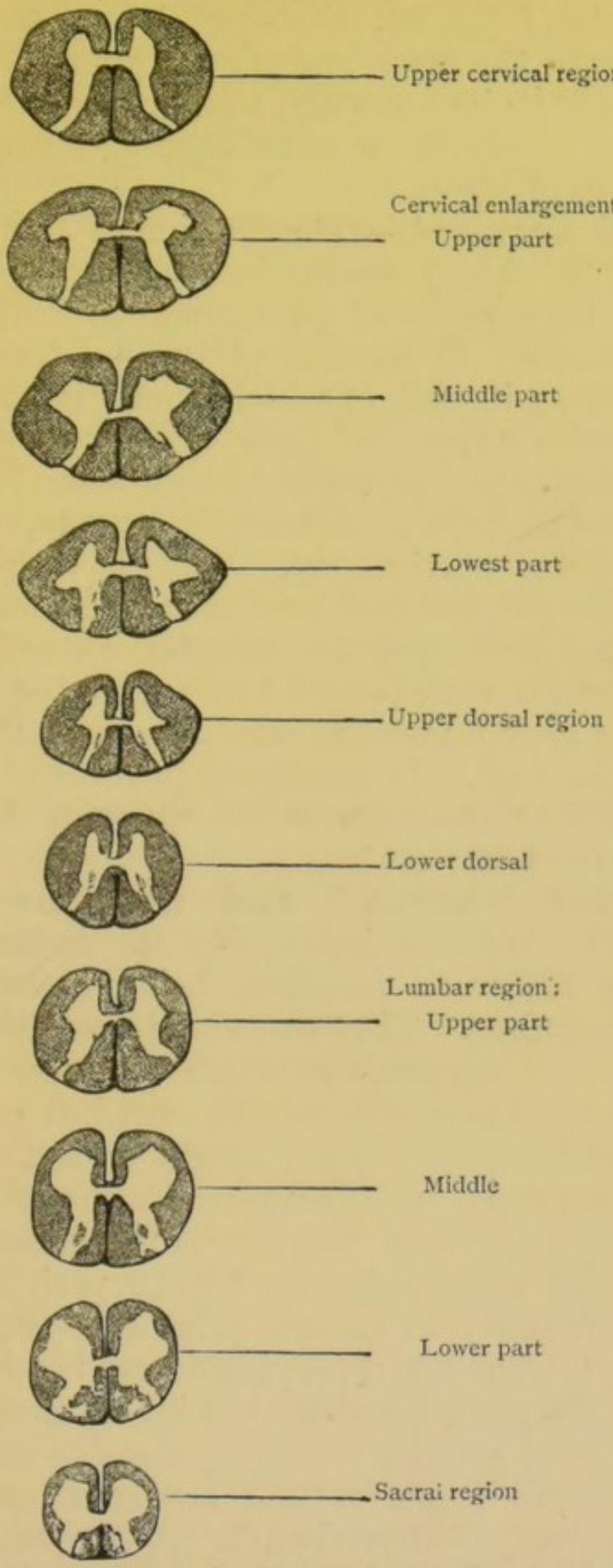
At other levels the sympathetic connection is less important, and the spinal system reaches a higher and more complex development. This takes place at the cervical and lumbar enlargements, where the nerves for the limbs are given off. Here the roots are combined into the well-known plexuses, from which again the main nerve-trunks originate: by this means each nerve-trunk is brought into connection with more than one nerve-root, and more than one segment of the cord.

Spinal cord and membranes.—The spinal cord reaches from the occipital foramen to the lower border of the first lumbar vertebra. It is here continued by the leash of nerves known as the cauda equina. In the cauda equina are comprised all the nerves from the second lumbar downwards. Both these nerves, and also those immediately above them, have to run some way downward before they make their exit from the spinal canal.

The envelopes of the cord are, the dura mater which forms a tube within, but quite detached from, the bony canal; the arachnoid, which, by its denticulate processes, serves to suspend the cord within the dura; and the pia mater, which invests the cord closely and sends into its substance septa which carry its nutrient vessels.

Physiologically, the cord may be considered either as a *centre* (or rather a series of centres) for reflex actions, for the co-ordination and distribution of impulses proceeding from the brain, and for nutrition,

FIG. 1.



Transverse sections of spinal cord showing the shapes of the grey matter at different levels. The white matter has been stained black (according to Pal and Weigert's method) while the grey matter remains unstained.

particularly of the muscles—or else as a *connecting path* between the brain and the periphery. Similarly, in dealing with its anatomy, we may treat it either as a series of segments or as an aggregation of longitudinal tracts of fibres.

Spinal segment.—The division of the cord into segments is chiefly useful in considering the grey matter. Each segment corresponds in level to a pair of nerve-roots. It consists of two bilateral symmetrical halves; in each of which there is an anterior and posterior "horn" of grey matter, corresponding to the anterior and posterior nerve-roots. The shapes of these horns differ in different segments, that of the anterior horn particularly, which is larger at the levels of the cervical and lumbar enlargements. Here also are best developed its characteristic cells.

The horns of each lateral half are united by a commissure of grey matter, in which is seen the central canal of the cord. Immediately in front of this grey commissure at the base of the anterior fissure is a commissure of white fibres.

Anterior "horns" and anterior cornual cells.—The anterior horns, like the anterior nerve-roots, are motor in function, being concerned with efferent impulses. They contain, in addition to the fine plexus of nerve-fibres which pervades the grey matter, large nerve-cells, multipolar, but having one process which gives direct origin to a fibre of the anterior nerve-roots. From the cornu these fibres can be seen, passing forwards in separate bundles through the white columns to the periphery. The cells are arranged in groups (Fig. 4, A and C), more or less well defined, and varying somewhat in different levels of the cord, (α) along the inner margin of the horn (internal group), (β) at its anterior and antero-lateral margin (anterior and antero-lateral groups), (γ) at its postero-external angle (postero-lateral or external group). In some parts of the cord there is (δ) a central group.

As the cells form the starting-point for the fibres of the anterior nerve-roots, so they are the terminal point of the fibres which convey motor impulses from the brain (fibres of the pyramidal tract). Such impulses are probably grouped and re-arranged in the grey matter of the anterior horns.

They are connected also by the grey matter with the nerves that enter the cord by the posterior roots, forming thus part of the spinal centre for reflex action. In addition they maintain the nutrition of their efferent nerve-fibres and of the muscles in which these end. Destructive disease of the cells therefore causes paralysis of the muscles which they supply, loss of the reflex actions subserved by them, and, in addition, wasting of the muscles, usually accompanied by changes in their electrical reactions. Blueness and coldness of the limb (vaso-motor paralysis) usually occurs. Disease of the anterior nerve-roots produces practically the same effects.

Posterior horns and cells.—The posterior grey horns contain, in most segments of the cord, but few and small cells. At certain levels, however, there are some important groups. One of these is known as the posterior vesicular column of Clarke, or, more briefly, as Clarke's column (Fig. 4, B). It extends from the third lumbar segment upwards through the dorsal region. It consists of largish bipolar cells with axis cylinder processes; these are set in the midst of tracts of fine white fibres. Its position (as seen in transverse section) is at the inner part of the posterior horn near the junction of it with the grey commissure. By some authorities, these cells are regarded as the point of origin for the fibres of the cerebellar tract; by others, as the cells which preside over the fine medullated efferent nerves to the sympathetic, viscera, and vessels, just as the anterior cornual cells preside over the motor nerves and skeletal muscles.

Another column of cells is known as the "intermedio-lateral tract." It is situated at the outer side

of the grey matter at the junction of the anterior and posterior horn. It is most distinct in the dorsal region, in the upper dorsal and lower cervical region forming a lateral "process" which projects outwards from the grey matter.

Posterior nerve-roots.—The posterior nerve-roots enter the cord in one large bundle; but this splits into two divisions. One of these, the lateral or external division, goes for the most part directly into the posterior horn, through the "substantia gelatinosa" (*i.e.*, a tract of grey granular material which forms a cap, as it were, to the posterior horn); but a small number of fine fibres turn upwards and run longitudinally in the cord at the tip of the posterior grey horns, forming here Lissauer's tract.

The second division (median or internal) curves inwards into the postero-lateral column, and, after running upwards therein for a short way, partly curves outwards again into the base of the posterior horn, partly runs inwards to join the postero-median column.

The posterior nerve-roots convey to the nervous centres all afferent impulses from the various organs

FIG. 2.

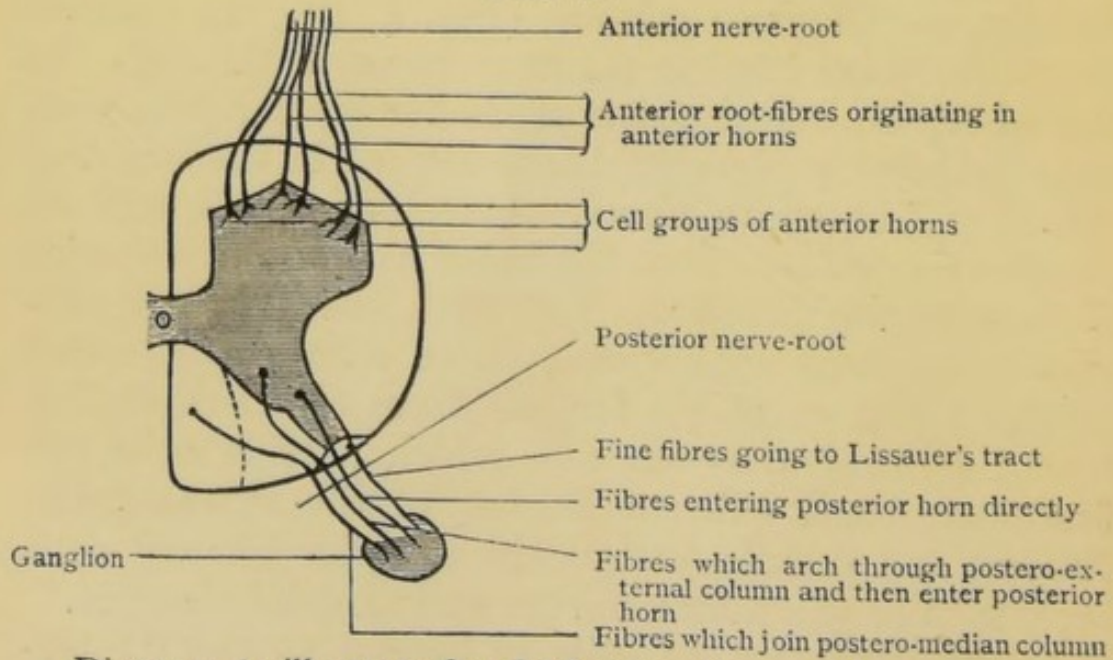


Diagram to illustrate the chief connections of the anterior and posterior nerve-roots.

of the body, whether these be such as, ascending to the cortex cerebri, give rise to actual sensation, or such as call into action lower cerebral centres, or such as, entering the grey matter of the cord, give rise to the simpler spinal reflexes. Of the course pursued by sensory impulses after they reach the cord we know, anatomically, nothing; but it would appear from physiological experiment and some facts of disease that soon after entering the cord they cross to the other side.* The course of sundry upward nerve-tracts to the cerebellum and elsewhere we know, but we do not know their function.

Reflex arc.—The path for reflex action within the

FIG. 3.

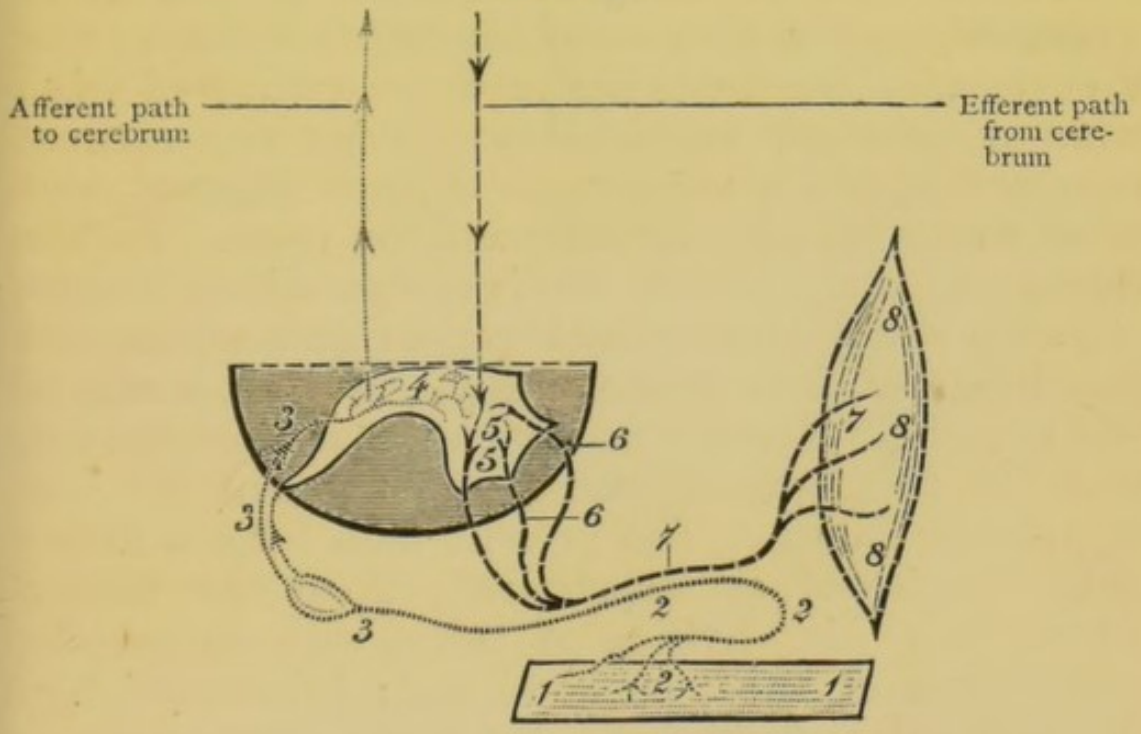


Diagram of a reflex arc (the numbers correspond to those in the text).

cord is provided by the grey matter which joins the posterior nerve-roots to the anterior cornua. Thus, so far as a single spinal segment is concerned, the "reflex arc," as it has been termed, consists of the following parts:

* But *vide* foot-note to p. 82.

(1) Peripheral (sensory) organ.
(2) Afferent nerve (fibrils of origin and nerve-trunk).
(3) Posterior nerve-root (extra- and intra-spinal).
(4) Fibres of grey matter.
(5) Anterior cornual cells.
(6) Anterior nerve-roots (intra- and extra-spinal).
(7) Efferent nerve (nerve-trunk and terminal fibres).
(8) Terminal (motor) organ.

Destruction of any of these parts will annul the reflex; but where 5, 6, 7, or 8 is destroyed, there will be motor paralysis as well.

The more detailed consideration of the various spinal reflexes we shall defer for the present.

Motor and sensory functions of different segments.—The motor and sensory function of the various spinal segments has yet to be considered—*i.e.*, what muscles are supplied by the anterior cornual cells and anterior nerve-roots of each segment, and what areas of skin by its posterior roots. In the dorsal region of the cord this is comparatively simple, since the distribution of each nerve-root is simple and can be ascertained anatomically. At the levels of the large limb-plexuses matters are more complicated, and the functions of each spinal segment are less completely known. The present data for our knowledge consist of (α) experiments upon the several nerve-roots—viz., division of them, with subsequent stimulation of the peripheral end; (β) dissection of nerve-bundles downwards, from the roots through the plexuses and nerve-trunks to their ultimate distribution; (γ) observation of the effects resulting from limited or gradually progressive cord lesions, the locality of the lesion being determined post-mortem. It is usually assumed that the function of each nerve-root corresponds to that of its spinal segment; and there is a certain agreement (though not altogether complete) between the result of the three methods we have mentioned.

The following tables (taken from Thorburn *) are based on the third or clinical method:

Cervical enlargement.	*Motor distribution.*	*Sensory distribution.*
4th cervical segment supplies	Supra- and infra-spinatus	Skin over deltoid and outer aspect of arm and forearm.
	Teres minor ?	
	Biceps	Central parts of arm and forearm on anterior and posterior aspects.
5th do. do.	Brachialis anticus	
	Deltoid	
	Supinator longus brevis	
6th do. do.	Subscapularis	
	Pronators	
	Teres major	
	Latissimus dorsi	
	Pectoralis major	
	Triceps	
	Serratus magnus	
7th do. do.	Extensors of wrist	
8th do. do.	Flexors	
1st dorsal	Intrinsic muscles of hand	Little finger and inner side of hand, forearm and arm.
1st lumbar	None	Ilio-hypogastric and ilio-inguinal.
2nd do.	None	Outer (?) and upper part of thigh.
3rd do.	Sartorius	Anterior aspect of thigh below 2nd lumbar.
	Adductors of thigh	
	Flexors of thigh	
th do.	Extensors of knee	Anterior and inner part of leg.
	Abductors of thigh	
th do.	Hamstring muscles	Back of thigh except in distribution of 1st, 2nd and 3rd sacral.
1st sacral	Calf-muscles	Nervi erigentes. A narrow strip on back of thigh, back of leg, and ankle; sole; part of dorsum of foot.
	Glutei	
	Peronei	
2nd sacral	Extensors of ankle†	
	Intrinsic muscles of foot	
3rd sacral	Perineal muscles, viz., erector penis, transversalis perinei, accelerator urinæ, &c.	Perineum, external genitals, "saddle-shaped" area on back of thigh.
4th sacral	Bladder and rectum.	

* "Surgery of Spinal Cord," pp. 4, 42, 111.

† *I.e.*, dorsi flexors.

Longitudinal conducting paths or "tracts" in the cord.—Viewed as a conducting path to and from the brain, the spinal cord may be mapped out into various longitudinal paths or "tracts." These are formed from the white matter of the cord. Most of them are not distinguishable, in the healthy cord of an adult, anatomically, but their existence is determined partly by the definite courses taken by degenerative processes, primary or secondary, in the cord; partly by the differences in the date of their

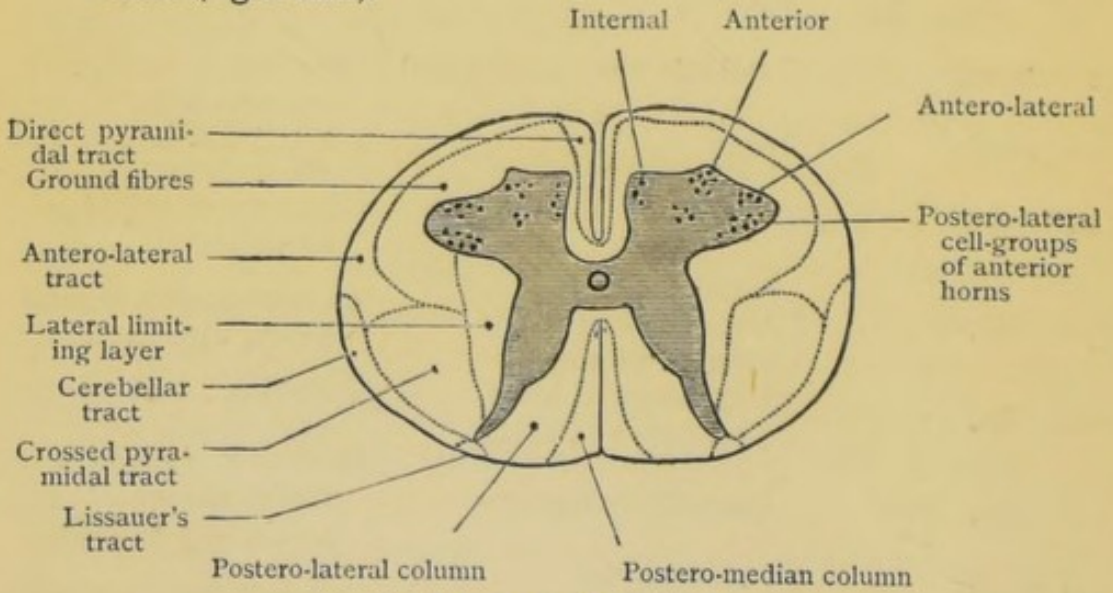
FIG. 4.

Diagrammatic sections of spinal cord in—

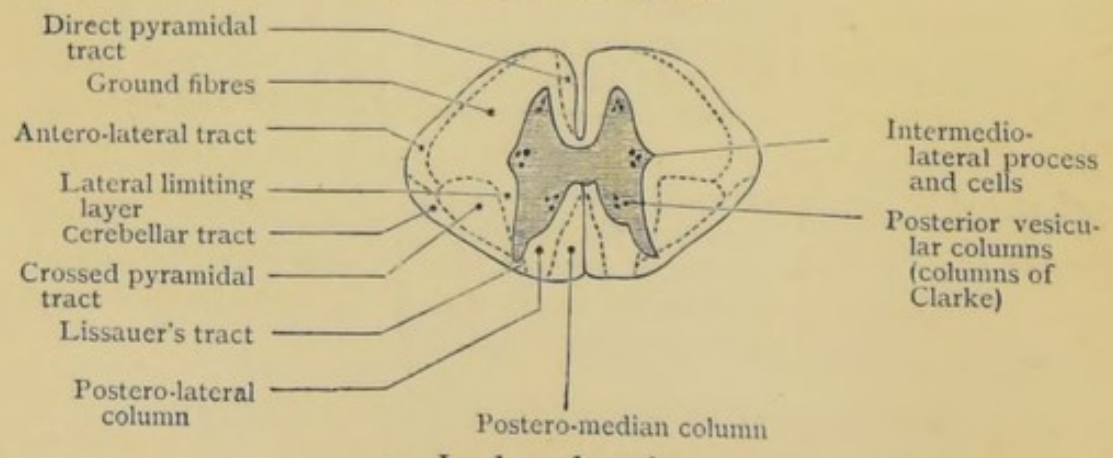
(A) Cervical,

(B) Upper dorsal,

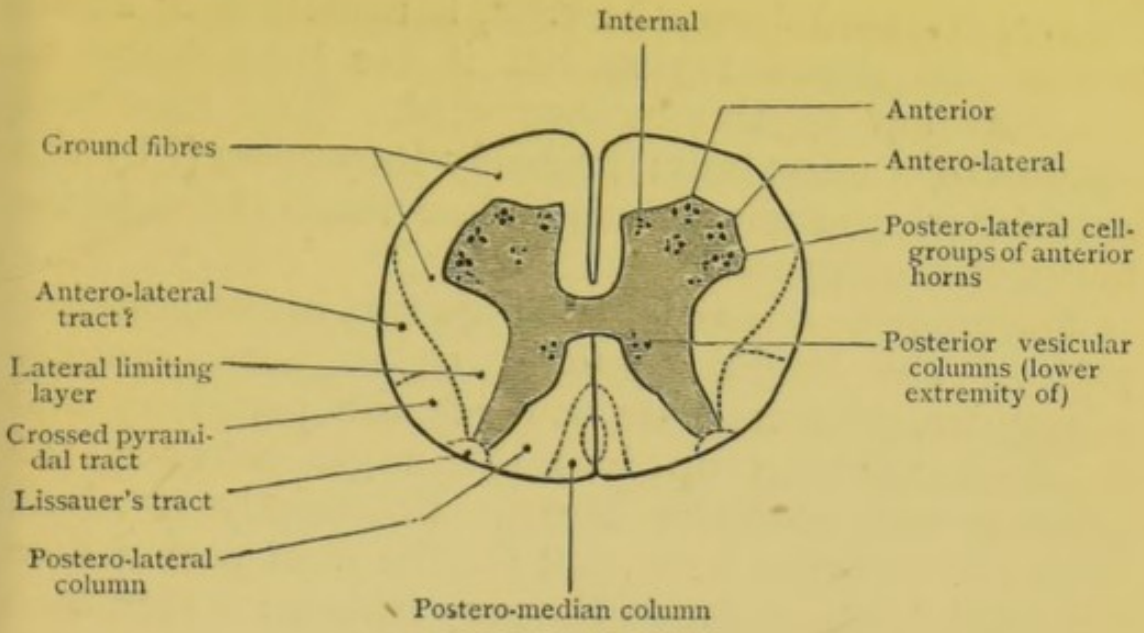
(C) Lumbar regions: to show the tracts in the white matter (left half), and the grouping of the cells in the grey matter (right half).



A. In cervical region.



B. In dorsal region.



C. In lumbar region.

development, the date, that is, at which the nerve-fibres of the various tracts become clothed with myeline. Some certainly, and probably all, of these tracts have distinct functions.

Motor or "pyramidal" tract.—The motor tract is the best known, and we will begin with this. Taking a transverse section, say at the cervical enlargement, there are present in the antero-lateral columns (*i.e.*, the parts between the anterior fissure and the posterior horns):

(*a*) The crossed, or lateral, pyramidal tract.

(*β*) The direct, or anterior, pyramidal tract.

These two are the continuation downwards of the anterior pyramid of the medulla (whence the name pyramidal), and thereby of the fibres which carry motor impulses from the brain.

The crossed, or lateral pyramidal tract represents that (larger) portion of the motor fibres which have decussated at the lower part of the medulla; the direct or anterior pyramidal tract, sometimes also called the column of Türck, is the (smaller) non-decussated portion.

The direct pyramidal tract is seen in transverse section as a narrow strip along the edge of the anterior

fissure. Traced down the cord, it usually ends about the middle dorsal region, but it has been found to reach as low as the lumbar cord. How its fibres terminate is uncertain; probably they cross (? by the anterior white commissure) and so reach the anterior horns of the opposite side.

The crossed pyramidal tract appears in the posterior part of the lateral columns, as an area roughly triangular, with its base towards the periphery and its apex running up into the angle between the anterior and posterior horns. Posteriorly it abuts upon the posterior horn, but for the most part it does not reach quite so far as the grey matter; a thin zone of white matter, called the lateral limiting layer, separates it therefrom. Neither does it, at least in the cervical* and dorsal regions, quite reach the surface of the cord, since the cerebellar tract, presently to be mentioned, here occupies the periphery. But in the lower dorsal and lumbar regions, where the cerebellar tract does not exist, the crossed pyramidal tract extends to the surface. This tract reaches, longitudinally, down to the upper sacral region. It becomes smaller as it passes downwards, because fibres are continually leaving it to terminate in the anterior cornual cells. The actual path of the fibres has not been determined; but it is certain, since this tract constitutes the main motor path from the brain, that its fibres must eventually be connected with these cells.

Cerebellar tract.—The direct cerebellar tract occupies, in transverse section, a narrow zone at the periphery of the lateral columns of the cord, exterior to the crossed pyramidal tract. At most levels this zone extends from the tip of the posterior horn forwards as far as a line drawn horizontally

* Except in the upper cervical region, where, owing to a forward movement of the cerebellar tract, the posterior part of the crossed pyramidal tract comes to the surface of the cord.

through the central canal. In the lower dorsal region where the tract is small, and also about the third cervical segment where it is shifted a little forwards, it does not extend quite so far backwards as the posterior horns. The tract contains afferent fibres passing to the cerebellum. They originate, according to some authorities, in the cells of the posterior vesicular column of Clarke.

The tract begins to be visible in the lower dorsal regions and passes upwards, in the position described above, to the medulla. It then enters the restiform body, and ends in the middle lobe of the cerebellum.

Antero-lateral ascending tract.—The "antero-lateral ascending tract" constitutes another tract of ascending fibres. In transverse section, it occupies the periphery of the cord in front of the cerebellar tract, reaching forwards nearly to the margin of the anterior fissure. But it also dips into the cord, sending a process inwards in front of the crossed pyramidal tract towards the grey matter, and filling up any place left between the cerebellar and the crossed pyramidal tract. Its fibres are believed to originate in the grey matter through the whole length of the cord, commencing at any rate at a lower level than those of the cerebellar tract. Possibly it bears some definite relation to the posterior nerve-roots. When traced upwards into the medulla it maintains, according to Tooth, a position at the periphery, just posterior to the olivary body. It thus becomes separated from the cerebellar tract, which here passes backwards towards the restiform body. It is by some authors thought to end in the lateral nucleus of the medulla. Others maintain that it is really part of the cerebellar tract, and ascends like it *viâ* the restiform body to the cerebellum.

The remainder of the antero-lateral columns of the cord—viz., all that part of the white matter, from the anterior fissure backwards to the posterior grey horn—which has not been included in the various tracts we have described, constitutes what has been called the

anterior and lateral "ground fibres," and the "mixed lateral zone." We shall not here enter into a description of these parts.

Structure of posterior columns. — Each posterior column is divisible into a postero-lateral and a postero-median portion. A rough anatomical distinction between the two is afforded by a septum which passes inwards from the pia mater. The postero-lateral column, called also the cuneate fasciculus or the column of Burdach, constitutes the external division.

Postero-lateral column or column of Burdach.—This is the stripe of white matter which lies next to the posterior grey horn, its extent varying at different levels in the cord. It has a close relation to the posterior nerve-roots; some of these traverse the column horizontally, passing through it into the neck of the posterior grey horn; while others after entering the column run upwards in it a little way, and then join—some the grey matter, some the postero-median column. Their place in the postero-lateral column is taken by a fresh supply from other nerve-roots. The fibres are afferent and degenerate upwards. But owing to the arrangement just stated—viz., the continuous replacement of fibres from the nerve-roots—it is found that these columns do not (in the case of a transverse lesion) degenerate for any great distance from the original lesion. That part of the column which is traversed by the fibres entering from the posterior roots (viz., the part adjacent to the grey horn as it nears the periphery) is of importance, because disease here cuts the posterior root-fibres and causes disappearance of tendon reflex.* The postero-lateral columns are continued into the lower part of

* Westphal defines the area in the lower dorsal and upper lumbar regions thus: It forms a rough triangle whose anterior side is the posterior edge of the grey matter, its posterior the periphery of the cord, its inner side a line drawn backwards from the re-entrant angle formed by the grey matter

the medulla, where they terminate in the post-pyramidal nuclei.

Postero-median, or column of Goll.—The postero-median columns, otherwise called the columns of Goll, are placed on each side of the posterior fissure; the two tracts together form, in the lower parts of the cord, a triangle with the base at the periphery, in the upper parts a narrow strip bordering the fissure on each side. These tracts are to a great extent formed from fibres which come from the posterior nerve-roots and have run a short course (as described above) in the postero-lateral column. After entering the postero-median column, the fibres run in it upwards without interruption to the medulla (fasciculus gracilis) where they terminate in the postero-median nucleus. The fibres that enter from the lower nerve-roots place themselves, as they ascend the cord, behind those that enter from the upper nerve-roots. Therefore, in a lesion of the lower part of the cord, degeneration, although traceable through the whole length of the columns, is limited in the cervical region to their posterior part.

The two postero-median columns do not decussate: the posterior nerve-roots of each side are continued upwards in the column of the same side.

The postero-median, like the postero-lateral columns, are composed of afferent fibres. Yet neither of them, so far as we know, in spite of their intimate connection with the posterior nerve-roots, forms the path for common cutaneous sensation.

To the account of these, the principal known tracts, we may add a few minor points.

1. Between the postero-median and postero-lateral tracts, both of which are, as we have said, afferent or ascending, is a small tract of fibres, which, in a trans-

of the posterior horn, and parallel with the posterior fissure. This he calls the zone of entry of the posterior nerve-roots (*Würzel-eintritt-zone*), and disease here causes loss of patellar tendon reflex (*vide* Fig. 59).

verse lesion of the cord, degenerates *downwards* for a short distance. This is called, from its shape, the comma-shaped degeneration.

2. Low in the lumbar region, on either side of the posterior fissures, and enclosed between the two postero-median tracts, is a small area, shaped like a bi-convex lens, which is sometimes found intact, while the postero-median tracts are degenerated.

3. A small ascending tract has been described at the tip of each posterior cornu between this and the periphery of the cord. It is reckoned by some authors into the posterior, by others into the lateral, columns. It runs the whole length of the cord and conducts and degenerates upwards. It consists of fine medullated fibres derived from the posterior roots (? coming from the sympathetic), and probably terminates at various levels in the posterior grey horns. It is called Lissauer's tract, and it is said to degenerate early in cases of locomotor ataxy.

4. A few fibres in the antero-lateral columns of the cord are found to degenerate in a downward direction.

Medulla oblongata. — Numerous alterations take place in the cord during its passage upwards into the medulla. The most striking of these alterations are—

I. The decussation of the pyramids.

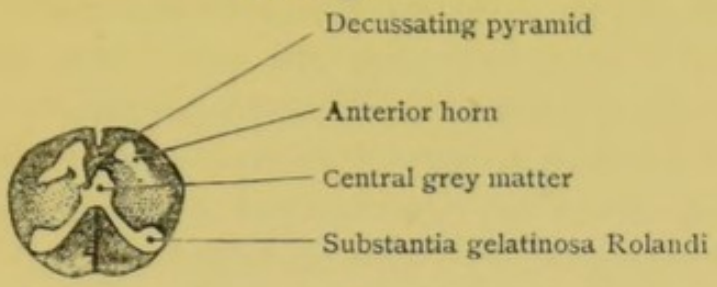
II. The development of sundry grey nuclei, some of which are not represented in the cord.

III. The opening out of the central canal to form, on the dorsal aspect of the medulla, the fourth ventricle.

I. The motor fibres in each lateral column (crossed pyramidal tract) as we trace them upwards to the medulla, advance to the front, and cross each to the opposite side of the middle line (decussation of the pyramids), and there combine with the motor fibres of the opposite anterior columns (direct pyramidal tract) so as to form the anterior pyramids of the medulla. In

thus crossing they cut off and throw to the sides a part of the anterior grey cornua.

FIG. 5.



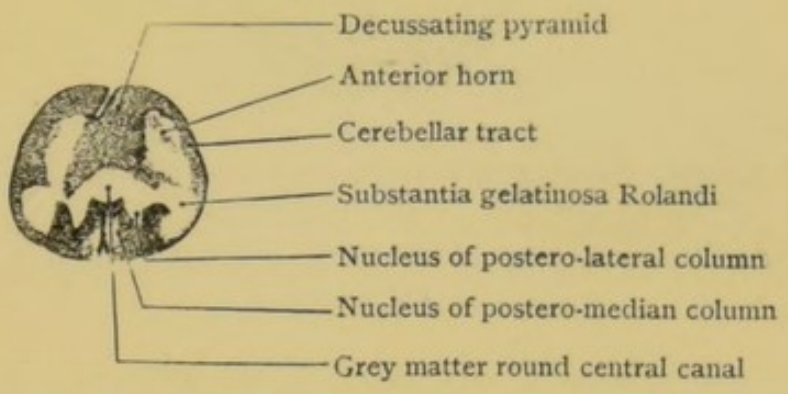
Transverse section through lower part of decussation of the pyramids (just above first cervical nerve).

II. Nuclei of grey matter are developed in the following parts:—

1. In the posterior columns of each side appear—

a. The nucleus of the posterior median column, wherein the fibres of the posterior median column terminate.

FIG. 6.



Transverse section through upper part of decussation of pyramids.

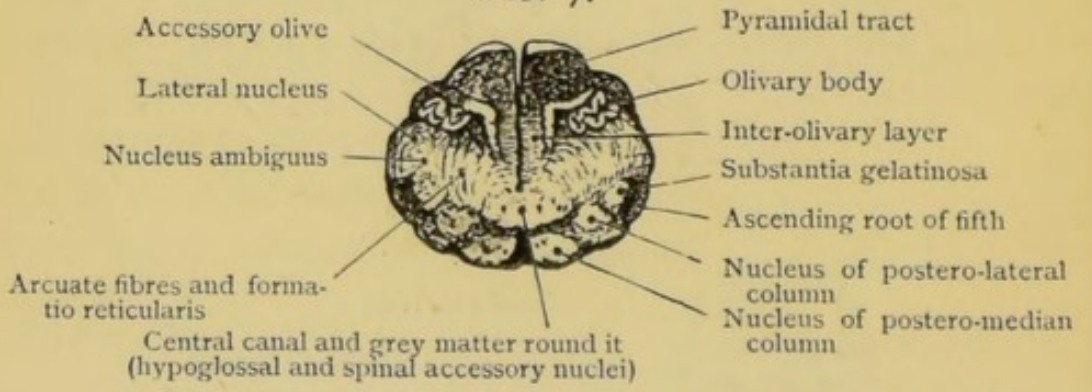
β. The nucleus of the posterior lateral column, wherein the fibres of this column terminate.

[These nuclei are sometimes called the post-pyramidal nuclei: besides receiving the fibres of the posterior column they give origin to fibres which pass brainwards. These are called arcuate fibres, from their peculiar curved course. They sweep forward through the deeper parts of the medulla beneath the grey matter which surrounds its central canal, and thus reaching its ventral aspect they decussate with

each other, at a higher level than the decussation of the anterior pyramids. This is sometimes called the "superior pyramidal decussation." A large proportion of them then passes brainwards on either side of the middle line, between the olivary bodies, forming the "inter-olivary layer," and eventually the "fillet." Others continue a curved course round the periphery of the medulla on its ventral aspect, encircling the anterior pyramid and the olivary body, and eventually join the restiform body.]

2. Between the posterior and lateral column—the grey matter at the tip of the posterior cornu (caput cornu posterioris) enlarges to form the substantia gelatinosa of Rolando. This corresponds to a swelling

FIG. 7.



Transverse section of medulla, a little below the calamus scriptorius.

on the surface of the medulla, which constitutes, as it were, an extra pyramid, known as the funiculus or tubercle of Rolando.

[From the neighbourhood of the tubercle of Rolando rises a nerve bundle, which passes brainwards through the medulla up to the origin of the fifth nerve, this is the ascending root of the fifth nerve.]

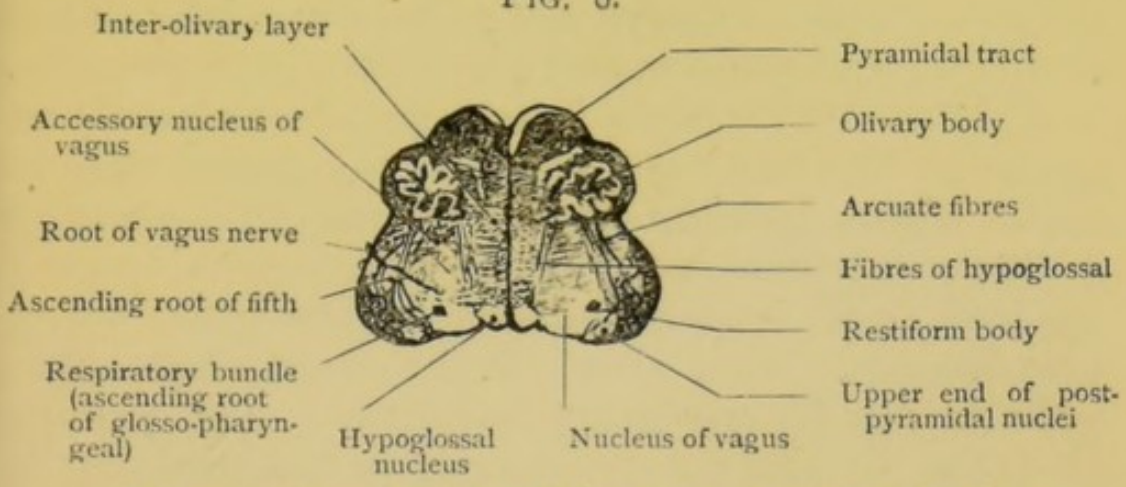
3. In the central and lateral parts of the medulla—

a. That part of the anterior grey cornu which has been cut off and pushed to the side by the decussation of the pyramids, partly remains in the form of distinct nuclei (motor nucleus of the vagus or nucleus ambiguus and antero-lateral nucleus), partly becomes

mixed with white fibres derived from the anterior and lateral columns of the cord, and thus forms a network of grey and white matter known as the reticular formation of the medulla. This reticular formation is continued upwards through the whole length of the medulla and pons, forming a sort of basis for the floor of the fourth ventricle, which it separates from the white strands which lie in the ventral parts of the medulla and pons.

β. The grey matter round the central canal, reinforced by that part of the anterior cornu which has not been decapitated, forms the nuclei of the hypoglossal and spinal accessory nerves.

FIG. 8.



Transverse section of medulla at lower part of fourth ventricle.

4. At a higher level (viz., as the fourth ventricle begins to open out) there appear on each of the anterior pyramids the large wavy capsules of grey matter known as the olivary bodies. These cause a well-defined egg-shaped prominence on the outside of the medulla.

III. The central canal of the cord opens out posteriorly, and forms the fourth ventricle. In the floor of this ventricle are found numerous important nuclei which we shall mention presently: on either side of it (at the level, that is, of the medulla) is the inferior cerebellar peduncle, or restiform body, containing the direct cerebellar tract, the fibres which connect the

olivary body and cerebellum, and other fibres of connection between the cerebellum and medulla and cord.

Thus, when the level of the fourth ventricle has been reached, the arrangement of the constituents of the spinal cord has undergone a tolerably complete modification.

Region of pons.—The parts of the nervous axis above this level—viz., the upper part of the medulla, the pons, and the crura cerebri—are very complicated. Yet a few general remarks may be applied to this region also.

1. Like the cord, it consists of two symmetrical lateral halves: these (in the pons and medulla) are divided from each other by a median "raphé," a structure which conveys many fibres in a longitudinal direction, and receives and gives off others laterally.

2. It is also divisible into a posterior (dorsal) and an anterior (ventral) part. In the latter run the motor tracts, forming distinct columns in the medulla (anterior pyramids) and in the cura cerebri, but split up into small isolated bundles in the pons, by the interlacement with them of the cross fibres of the pons or middle cerebellar peduncles. The posterior or dorsal division consists largely in the medulla and pons of the reticular formation, and of the nuclei of various cranial nerves, while between this and the ventral division runs the flat layer of longitudinal fibres known as the "fillet." * In the region of the

* The fillet originates below in the inter-olivary layer (as above described) principally in connection with fibres from the post-pyramidal nuclei, passes upward between the ventral and dorsal divisions of the pons, and at the level of the crura cerebri divides; part of it (mesial fillet) retains for a time its former position—viz., between the crusta and tegmentum—and then ascends to the superior corpus quadrigeminum and optic thalamus (whence it is also called the superior fillet); part of it passes outwards to the superficial part of the crus, round which it winds to reach the inferior corpus quadrigeminum (hence called the inferior or lateral fillet; part of it, the most mesial part of all, turns in a ventral direction to join the crusta.

crura cerebri the dorsal division of the nervous axis (called the tegmentum) is distinctly separated from the ventral division (called the crusta or pes pedunculi), by a layer of darkly pigmented cells called the substantia nigra.

But these arrangements are complicated by—

3. The strands of fibres which form the connections of the cerebellum. These form three sets of pillars or crura (α) below the pons, the inferior cerebellar peduncles or restiform bodies which, diverging from the medulla upon either side of the fourth ventricle, convey fibres from the cord and medulla below up to the cerebellum (β) at the level of the pons, the middle cerebellar peduncles, which constitute the majority of the transverse fibres of the pons, and serve partly to connect the two lateral lobes of the cerebellum, partly to connect each lateral lobe with the opposite crus cerebri (as will be subsequently described); (γ) above the pons, the superior cerebellar peduncles, which converge from the lateral lobes of the cerebellum, upwards and towards the middle line of the tegmental division of the crura cerebri.

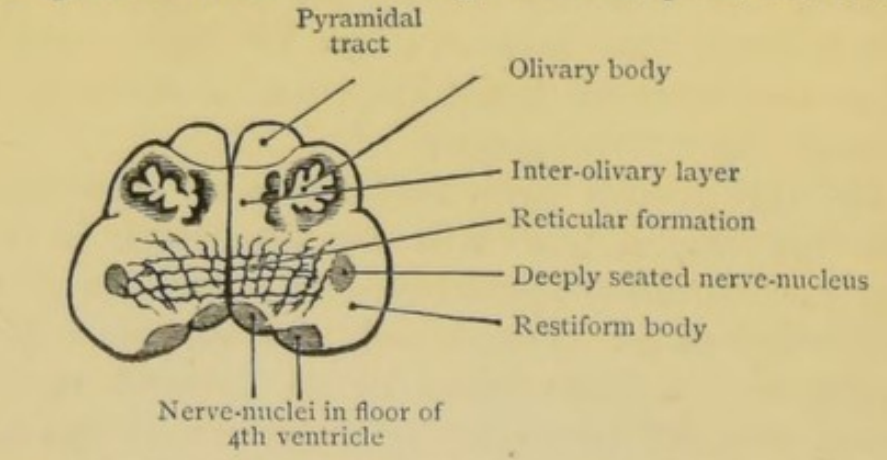
4. By the nuclei and root-fibres of the various cranial nerves. These will be described in detail presently. Roughly speaking, the nuclei form longitudinal columns of cells, which are disposed around the upper end of the spinal canal, around that continuation of it in the region of the crura cerebri which is called the Sylvian aqueduct, and between these levels, either in the floor of the fourth ventricle or somewhat deeper in the formatio reticularis. Some of the motor nuclei (*e.g.*, the hypoglossal and sixth) lie close to the middle line in the floor of the ventricle, having on their outer side in the more lateral parts of the ventricle sensory nuclei (*e.g.*, the pneumogastric, glosso-pharyngeal, and auditory). Another set of motor nuclei—viz., the pneumogastric, glosso-pharyngeal, facial, trigeminal—lie, as we have said, more deeply in the medulla, and farther from the middle

line. This latter set represents that part of the anterior grey horn which has been cut off and thrust aside by the decussation of the pyramids, whereas the former set represents that part which has not been thus decapitated, while the sensory nuclei external to them represent the posterior spinal cornua thrown forwards and outwards by the opening out of the ventricle. The fibres from the nuclei mostly pierce the pons and medulla, and emerge either at its front or sides, excepting the fourth nerve, which emerges at the back and runs round to the front outside the central nervous axis, and one root of the auditory nerve which similarly runs round the restiform body.

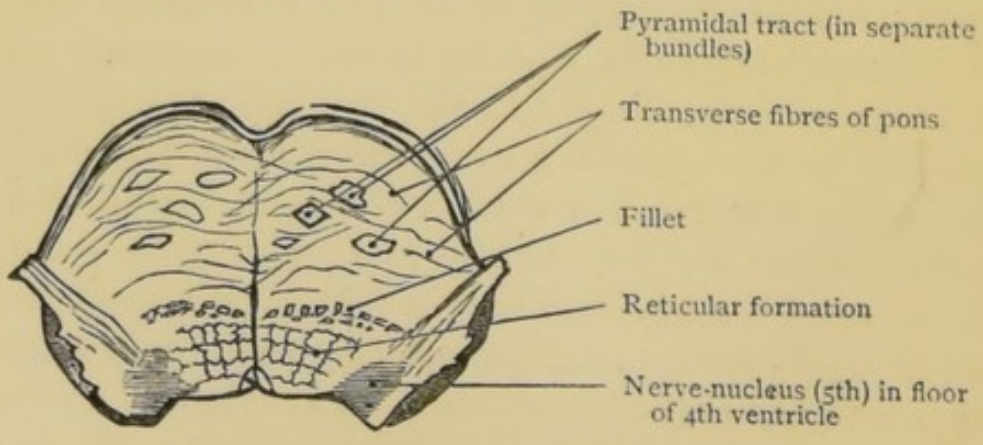
5. By certain comparatively small longitudinal nerve-bundles—viz., the ascending and descending roots of the fifth nerve—which connect this nerve

FIG. 9.

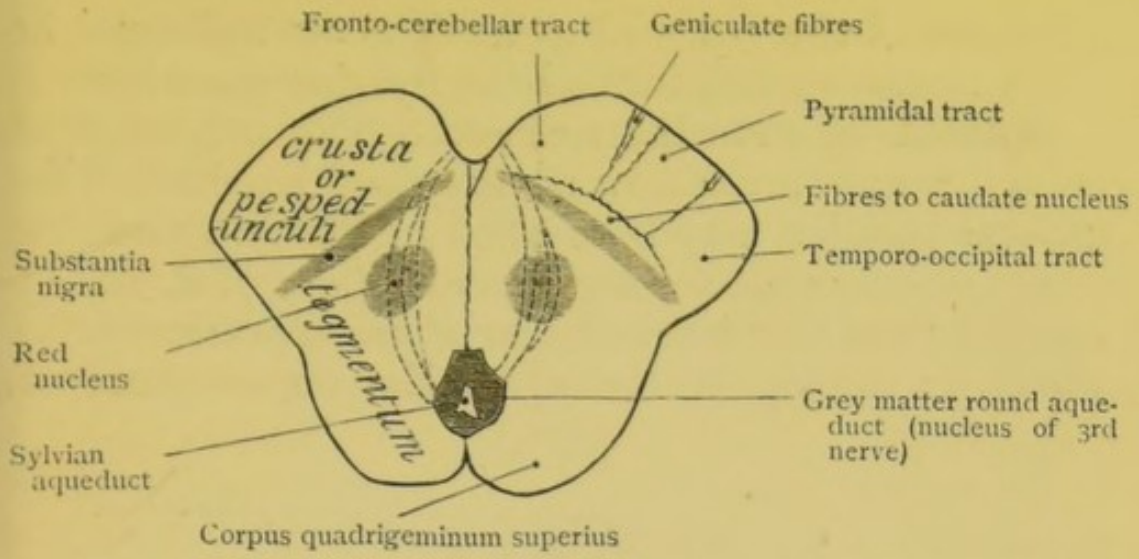
Diagrammatic sections through medulla, pons, and crura.



A. Through medulla at lower part of 4th ventricle.



B. Through pons at level of 5th nerve.



C. Through crura cerebri at level of superior corpora quadrigemina.

with the parts above and below it; the posterior longitudinal bundle, and the posterior respiratory bundle, which probably have some similar relation to the oculo-motor and glosso-pharyngeal nerves respectively.

The general features of the medulla and pons may be seen in the diagrams (Fig. 9). They are as follows—

1. In a transverse section through the medulla—anterior or ventral part consists of anterior pyramids (motor tract), olivary bodies, inter-olivary layer—posterior or dorsal part consists of reticular formation, nerve-nuclei (here hypoglossal and pneumogastric), restiform bodies.

2. Through the pons—anterior or ventral part consists of transverse fibres of pons interlaced with the scattered longitudinal fibres of the pyramidal tracts—posterior or dorsal part consists of reticular formation, nerve nuclei, 5th, 6th, facial or auditory, according to level of section. Between the anterior and posterior parts are the longitudinal fibres of the fillet.

3. Through the crura—anterior (ventral) and posterior (dorsal) divisions are well marked, being divided by the "substantia nigra." The tracts of nerve-fibres will be described later. The nerve-nuclei

are those of the 3rd and 4th. The central canal has again closed up and formed the Sylvian aqueduct.

Nuclei of cranial nerves.—The origins of the cranial nerves must now be described in detail. Their general position may be studied in Figs. 10 and 11.

The nucleus of the hypoglossal (Figs. 7, 8, 10, 11), reaches from the level of the striæ acousticæ downwards, to the top of the pyramidal decussation. In

FIG. 10.

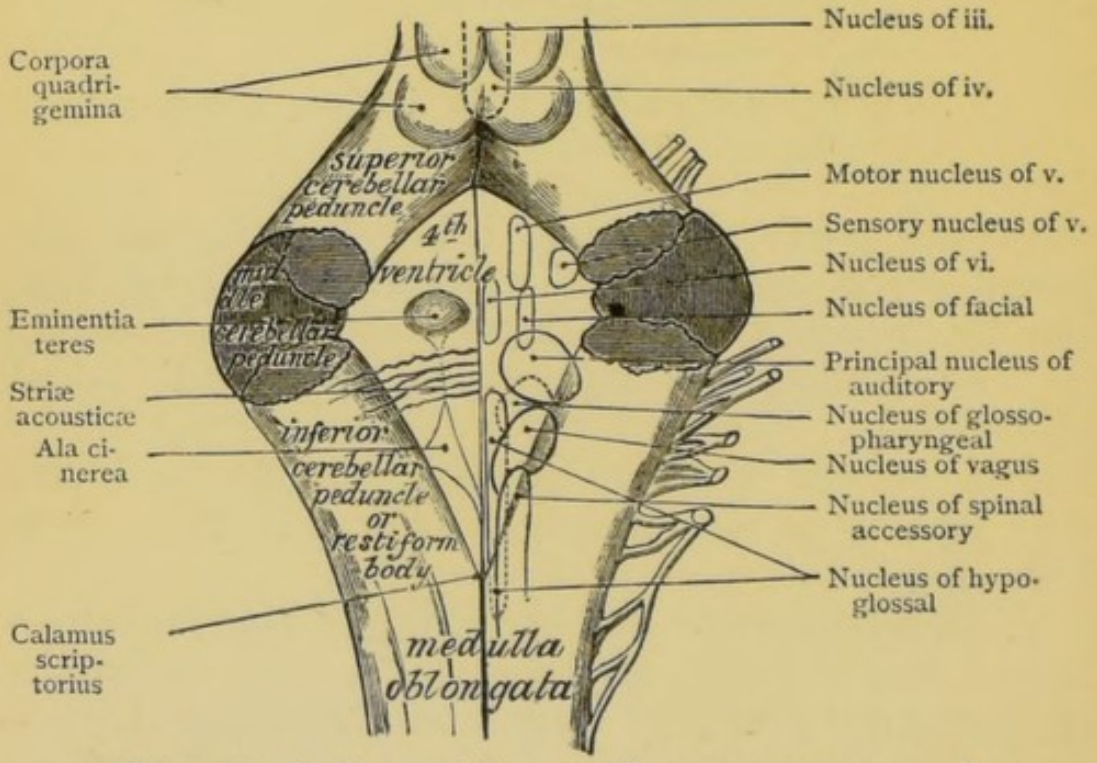


Diagram of 4th ventricle and adjacent parts, as seen from dorsal aspect, to show positions of nerve-nuclei. These are marked on the right-hand half. (After Erb.)

its lower part it lies in front of the central canal, in the floor of the 4th ventricle it lies next to the middle line. The nerve-fibres run outward and forward, and appear on the surface between the olive and anterior pyramid. The nerve-trunk leaves the skull by the anterior condyloid foramen, and is distributed to the muscles attached to the hyoid bone, particularly to those of the tongue.

The main nucleus of the *spinal accessory, pneumogastric, and glosso-pharyngeal* nerves (Figs. 7, 8, 10, 11, 13), may be reckoned as a continuous column which has

nearly the same extent from above downwards as the hypoglossal. The lowest part (nucleus of the spinal accessory) lies close to, and a little behind, the central spinal canal at its upper end; the middle part (nucleus of the pneumogastric) lies underneath the ala cinerea near the apex of the fourth ventricle, that is to say, just outside the hypoglossal nucleus; the upper part (nucleus of the glosso-pharyngeal) has a similar

FIG. 11.

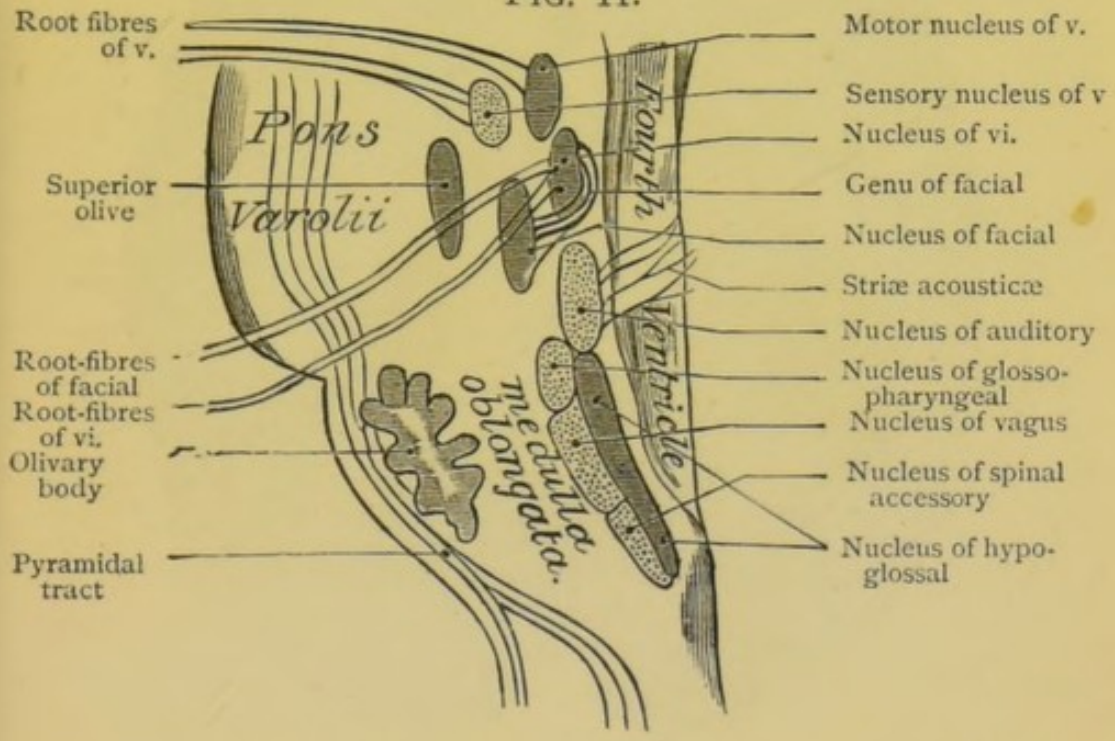
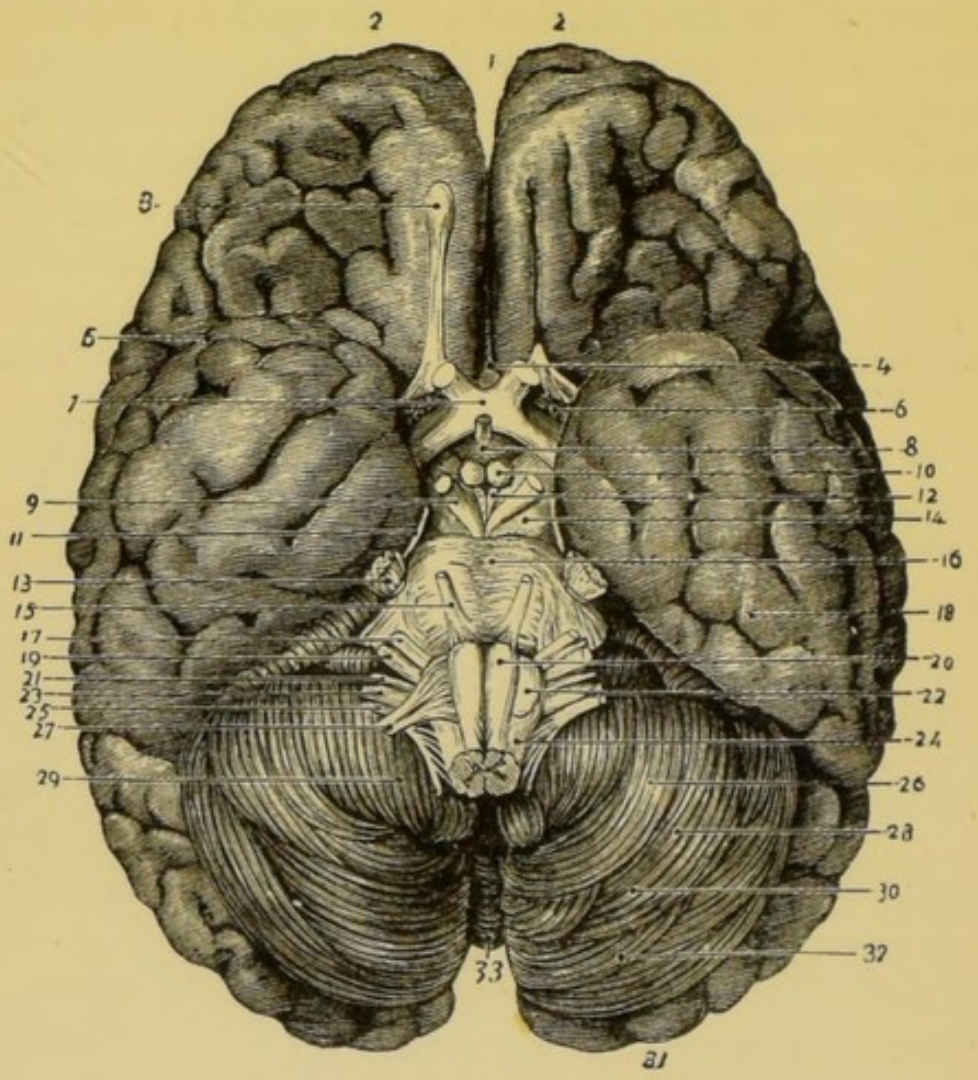


Diagram to show positions of principal nerve-nuclei in pons and medulla, side-view. The organ is supposed to be split down the middle line, and the right half viewed from the mesial side. The most mesially situated nuclei are shaded, the others stippled. (After Erb.)

position higher up in the medulla, being in juxta-position to the auditory nucleus which is outside and above it. The fibres of all these three nerves run forwards and outwards through the lateral parts of the medulla (piercing *en route* the ascending root of the fifth), and appear superficially behind the olivary bodies. They leave the skull by the jugular foramen.

[The spinal part of the spinal accessory nerve is in reality a distinct nerve; its fibres come from the

FIG. 12.



Base of brain (from Heath's "Anatomy").

1. Longitudinal fissure.
2, 2. Anterior lobes of cerebrum.
3. Olfactory peduncle and bulb.
4. Lamina cinerea.
5. Fissure of Sylvius.
6. Locus perforatus anticus.
7. Optic commissure.
8. Tuber cinereum and infundibulum.
9. Third nerve.
10. Corpus albicans.
11. Fourth nerve.
12. Locus perforatus posticus.
13. Fifth nerve.
14. Crus cerebri.
15. Sixth nerve.
16. Pons Varolii.
17. Portio dura of 7th.
18. Middle lobe of cerebrum.
19. Portio mollis of 7th.
20. Anterior pyramid.
21. Glosso-pharyngeal nerve.
22. Olivary body.
23. Pneumogastric nerve.
24. Lateral tract.
25. Spinal-accessory nerve.
26. Digastric lobe.
27. Hypoglossal nerve.
28. Cerebellum.
29. Amygdala.
30. Slender lobe.
31. Posterior lobe of cerebrum.
32. Posterior inferior lobe.
33. Inferior vermiform process.

cervical cord up through the foramen magnum into the skull; it also leaves the skull by the foramen jugulare, and is eventually distributed to the trapezius and sterno-mastoid.]

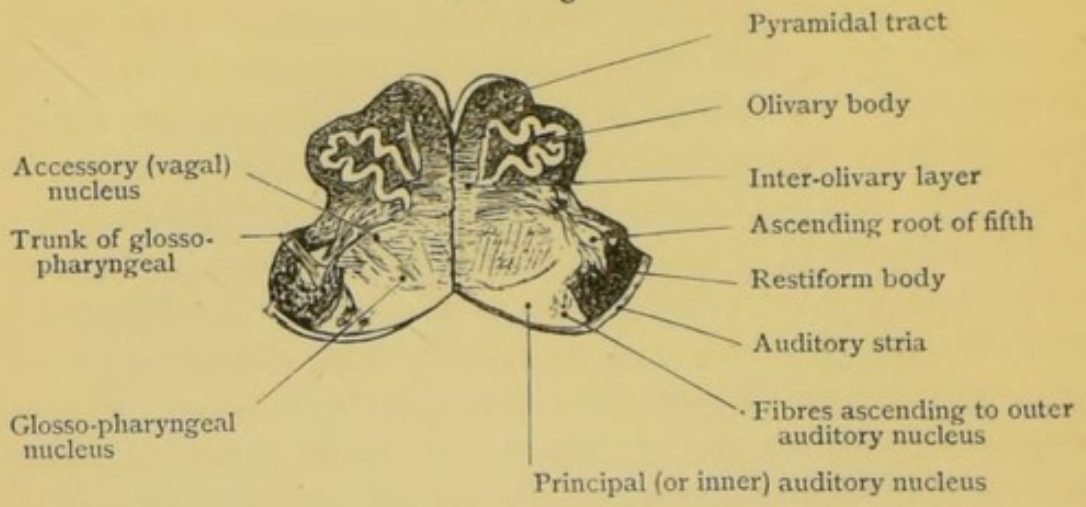
But, in connection with the origin of the three nerves in question, two additional structures must be considered. First, a column of cells (sometimes called the nucleus ambiguus) lying deep in the medulla in front of, and external to, part of the vagal nucleus: these cells (which may be considered, as we have said, as part of the anterior cornu of the spinal grey matter, cut off and pushed sidewards by the decussation of the pyramids) constitute the motor nucleus of the vagus and glosso-pharyngeal, the main nucleus being sensory. Secondly, a small round bundle of longitudinal nerve-fibres placed just outside the main glosso-pharyngeal and pneumogastric nuclei, not far from the surface of the fourth ventricle. It is continuous below with the lateral column of the cord. It is thought to constitute a root of origin for the glosso-pharyngeal, like the ascending root of the fifth nerve. It has received the various names of the slender bundle, the solitary bundle, the posterior respiratory bundle.

Nuclei of the auditory nerve (Figs. 10, 11, 13).

Usually three nuclei are assigned to the auditory nerve: a principal or inner nucleus, containing small cells, lying immediately under the floor of the fourth ventricle in its outer half or two-thirds, and reaching from the level of the auditory striæ downwards upon the outer side of the glosso-pharyngeal nucleus; an outer or superior nucleus, containing large cells, occupying the lateral part of the floor of the fourth ventricle outside the nucleus of the sixth nerve, and thus at a higher level than the principal nucleus. Two tracts of fibres come from these nuclei, the one (superficial root of the auditory) runs round the posterior border of the restiform body just below the pons: the other (deep root) pierces the lower border

of the pons. The two roots thus encircle the restiform body, and between them (or, indeed, partly situated on the superficial root) are cells which make up the

FIG. 13.



Transverse section of medulla, just below pons, and at lower edge of auditory striæ.

third or accessory nucleus of the nerve. Fibres also connect the auditory nerve with the cerebellum. The trunk of the auditory enters the internal auditory canal along with the facial, and is distributed to the internal ear: the superficial root goes to the cochlea, the deep root to the semicircular canals.

Nucleus of the facial (Figs. 10, 11).

This lies deep in the substance of the pons, somewhat to its outer side, and thus has a similar position to that of the motor nucleus of the vagus and glosso-pharyngeal (nucleus ambiguus). It may be seen in sections through the lower part of the pons, and extends upwards to the level of the greatest breadth of the fourth ventricle. The intra-pontine course of the facial is circuitous, just as is its course in the petrous bone. From the nucleus fibres pass upwards and inwards, and then transversely outwards between the nucleus of the sixth and floor of the fourth ventricle (loop of the facial). They then pass forwards and outwards just internal to the ascending root of the fifth, and appear on the surface close to the lower edge of the pons, between the olive and restiform

body. The facial trunk accompanies the auditory into the internal auditory meatus, a small branch (pars intermedia of Wrisberg connecting the two). It enters next the aqueduct of Fallopius, being joined at the upper part of this canal by the large superficial petrosal or Vidian nerve (which connects it with the spheno-palatine ganglion) and a branch from the small superficial petrosal (the communicating nerve between the tympanic plexus and otic ganglion). Near the end of the Fallopian aqueduct the facial gives off the chorda tympani, which connects it with the lingual nerve, the sub-maxillary ganglion, and tongue.

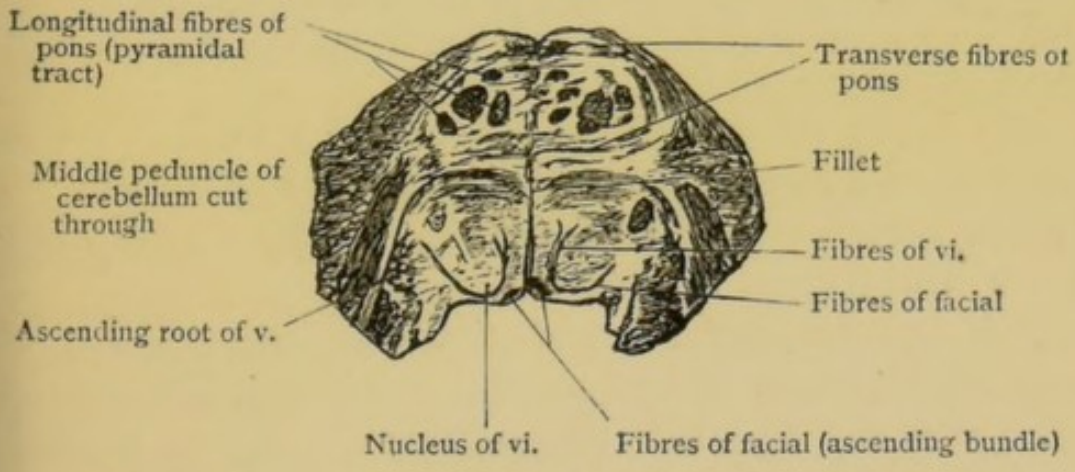
The facial nerve lies close to the inner wall of the tympanic cavity as it passes through the petrous bone; whereas the chorda tympani, passes across the cavity between the incus and malleus.

Not far from the facial nucleus, but deeper still in the substance of the medulla, is a nucleus known as the superior olivary body.

Nucleus of the sixth nerve (Figs. 10, 11, 14).

This lies near the middle line, in the upper half of the fourth ventricle, above the level of the striæ acous-

FIG. 14.



Transverse section of pons through eminentia teres.

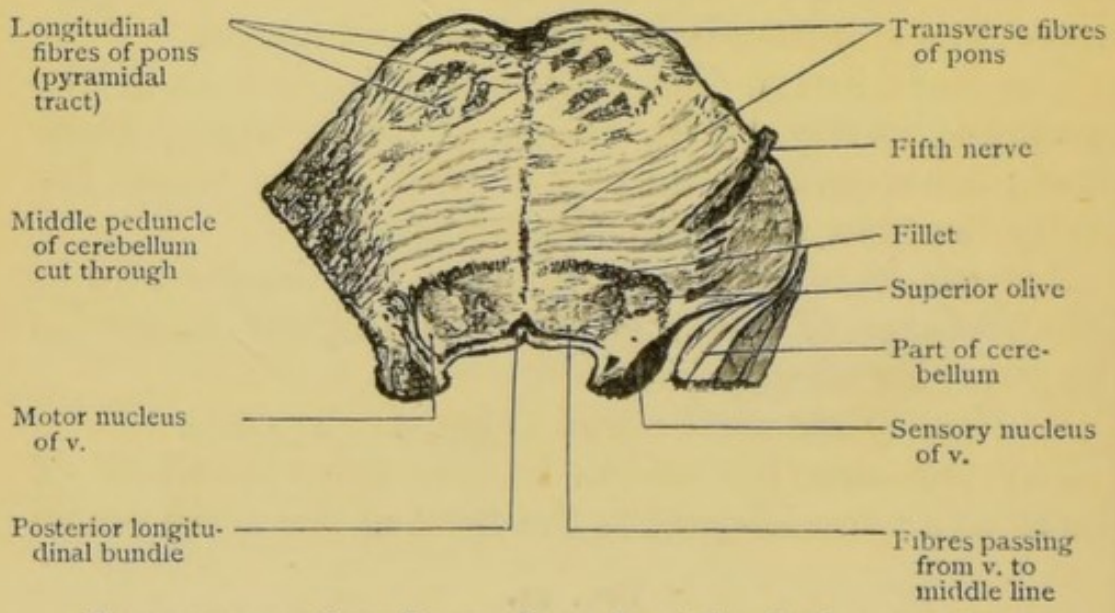
ticæ, underneath a prominence in the funiculus teres, called the eminentia teres. Around the nucleus curve the fibres of the facial, as above described. The fibres of the sixth run forward from the nucleus through

the pons to its superficial origin, which is at the lower border of the pons upon its front aspect, in a line with the origin of the hypoglossal. The nerve trunk runs forward in the floor of the cavernous sinus, and out through the sphenoidal fissure to the external rectus muscle of the eye.

Nucleus of the fifth (*trigeminus*). (Figs. 10, 11, 15.)

The fifth nerve rises in two divisions: a large sensory division and a small motor. Of these two the

FIG. 15.



Transverse section of pons through origin of 5th nerve (from a preparation lent by Dr. Tooth).

sensory is (at its superficial origin) at a rather lower level than the motor. Its deep origins are as follows:—

1. A sensory nucleus beneath the upper and outer part of the fourth ventricle.

2. A descending root, constituted by a band of fibres which runs down to the nucleus through the pons from the level of the anterior corpora quadrigemina. It probably passes into the motor division of the nerve.

3. An ascending root, a bundle which originates below at the upper part of the tubercle of Rolando, just about the junction of the cord and the medulla, and thence runs upwards to the nucleus, being pierced

on its way by the fibres of the spinal accessory, pneumogastric, and glosso-pharyngeal. It probably connects with the sensory division of the nerve.

(4) A third band of fibres, sometimes called the medial root, which runs inward along the floor of the ventricle towards the middle line.

The sensory division of the nerve, passing outwards from the nucleus, appears at the outer part of the pons, not far from its upper border.

The motor nucleus is placed somewhat deeply beneath the floor of the ventricle in its outer part, upon the mesial side of the sensory fibres as they pass to their point of exit. The motor division of the nerve at its superficial origin lies at a slightly higher level than the sensory, but soon passes beneath it. Both divisions pass underneath the dura mater to the apex of the petrous bone, where the sensory part is interrupted by the Gasserian ganglion, from which spring its three large branches, viz. :

1. The ophthalmic, which passes forward with the third and fourth nerve in the outer wall of the cavernous sinus, and out through the sphenoidal fissure.

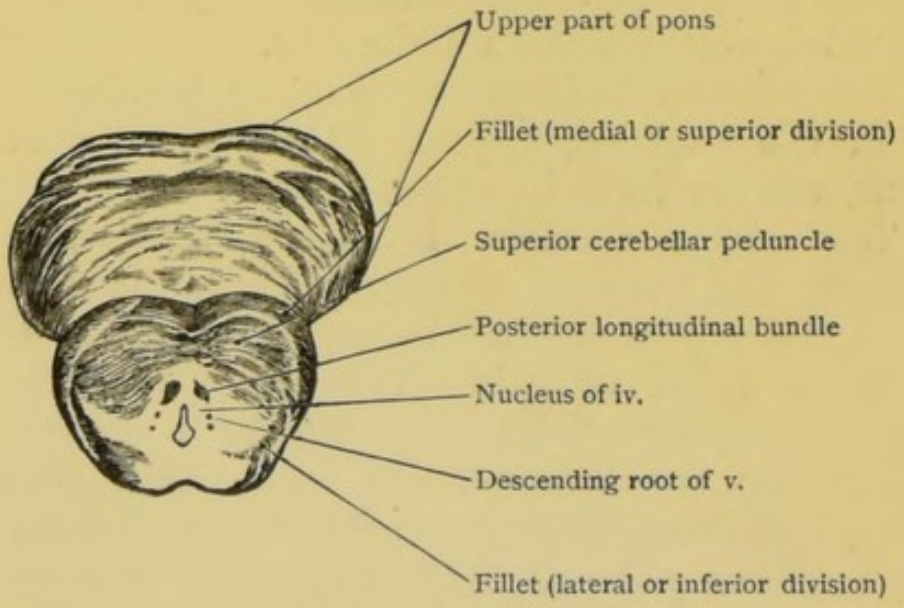
2. The superior maxillary, which pierces the sphenoid bone at the foramen rotundum, and crossing the spheno-maxillary fissure comes out on the face by the infra-orbital foramen.

3. The inferior maxillary, which passes out by the foramen ovale, and is joined by the motor division of the fifth.

Nucleus of the third and fourth nerves (Figs. 10, 16, 17, 18).—This lies in the floor of the Sylvian aqueduct, and extends from midway between the anterior and posterior corpora quadrigemina (this lower part constituting the nucleus of the fourth) up to the posterior part of the third ventricle. The course of the fibres of the two nerves differs remarkably. Those of the fourth run downwards and backwards into the valve of Vieussens, decussate here, wind round the

superior cerebellar peduncles, pass forward in the outer wall of the cavernous sinus, leave the skull by the sphenoidal fissure, and finally end in either superior

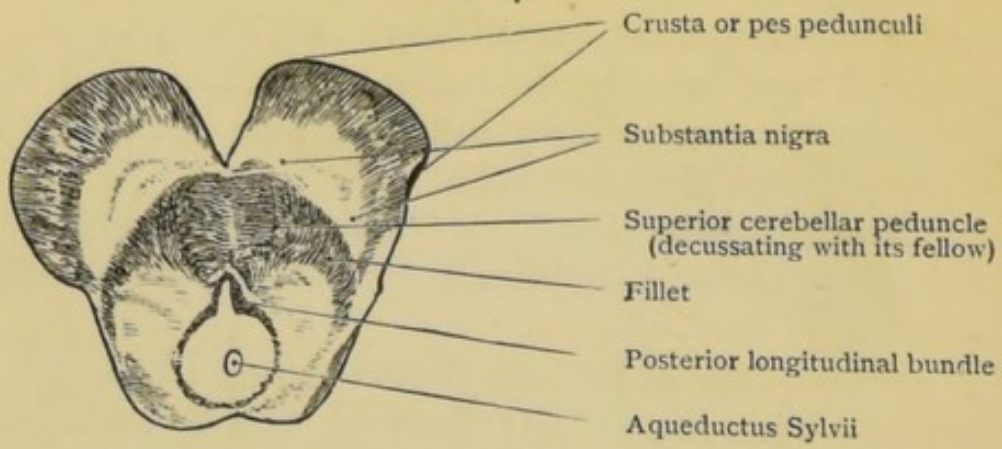
FIG. 16.



Transverse section of pons, at level of inferior corpora quadrigemina.

oblique muscle. The fibres of the third run forward through the crus cerebri, through the "red nucleus,"

FIG. 17.

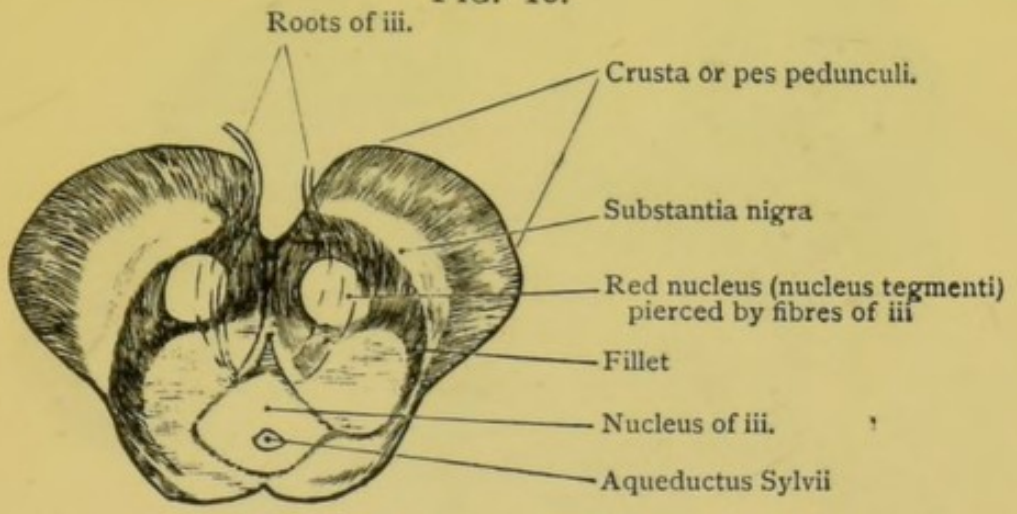


Transverse section through crura cerebri, between superior and inferior corpora quadrigemina (from a preparation lent by Dr. Tooth).

and become superficial between the crura cerebri just above the pons. The basilar artery separates the nerve of the two sides. Each third nerve then travels forward in the outer wall of the cavernous sinus, leaves the

skull through the sphenoidal fissure, and is distributed to the muscles of the eyeball and levator palpebræ.

FIG. 18.



Transverse section through crura cerebri, at level of superior corpora quadrigemina (from a preparation lent by Dr. Tooth).

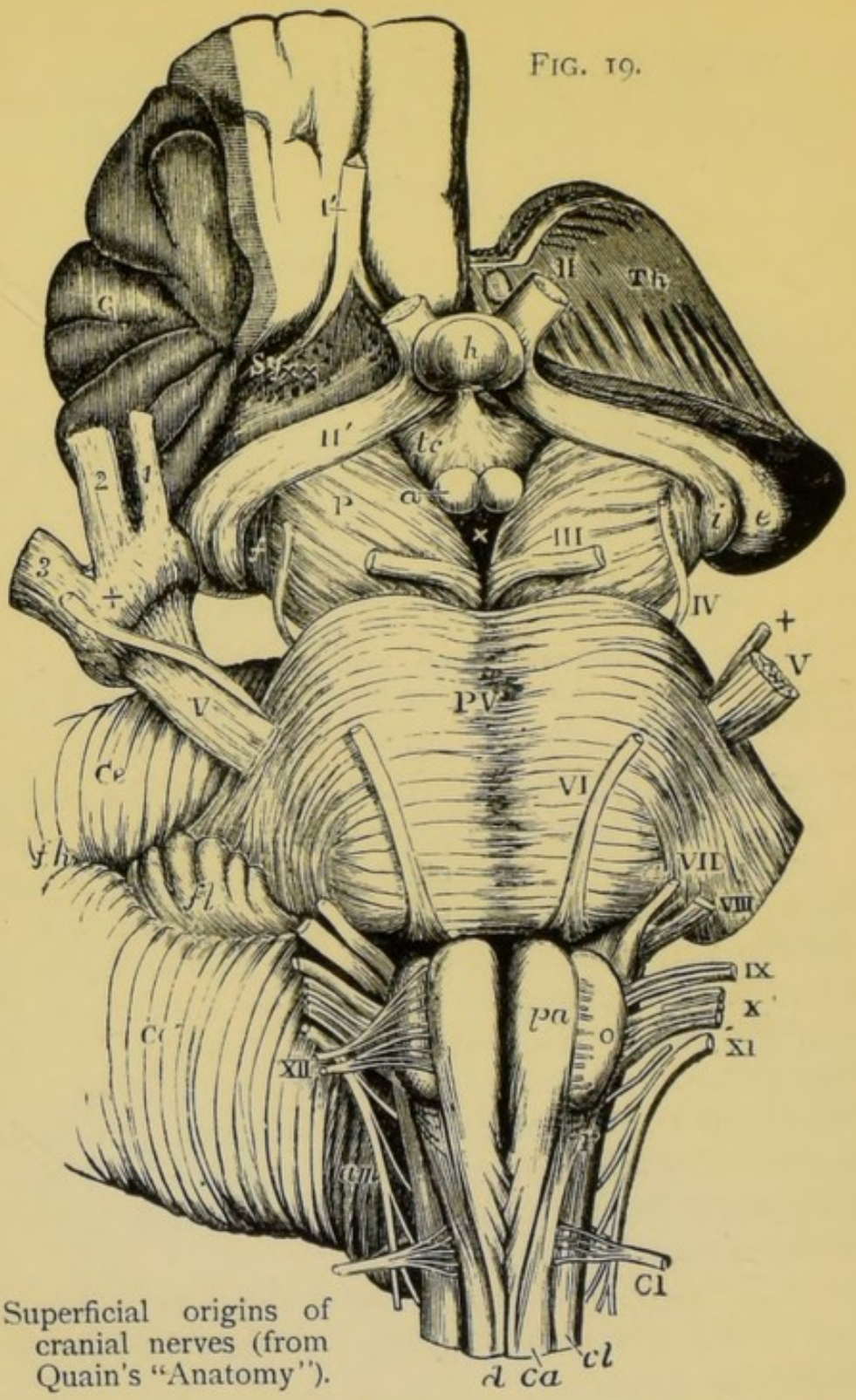
In connection with the oculo-motor nuclei may be mentioned the "posterior longitudinal bundle." This is a small band of white fibres which runs a little distance below the Sylvian aqueduct and the floor of the fourth ventricle, near the middle line and parallel with it. It is thought to connect the nucleus of the third and fourth with that of the sixth nerve. Above, it terminates in the grey matter around the wall of the third ventricle; below, it is continuous with fibres from the anterior columns of the cord; according to some authorities it is traceable for some way down the cord.

The optic and olfactory nerves belong to a higher level of the brain, but may be considered here for completeness' sake.

The optic tract originates from the anterior corpus quadrigeminum (the brachium of this body passes almost directly into this tract), from the "pulvinar" or posterior part of the optic thalamus, and from the external corpus geniculatum.* The tracts wind round

* Such fibres as come from the internal corpus geniculatum are said to be merely commissural, passing through the optic commissure to the corpus of the other side.

FIG. 19.



Superficial origins of cranial nerves (from Quain's "Anatomy").

I'. Olfactory tract.
II. Optic nerve.
II'. Optic tract.
III. Third or oculo-motor nerve.
IV. Fourth nerve.
V. Fifth nerve, sensory root.
V+ Fifth nerve, motor root.
1, 2, 3. Main divisions of fifth.
VI. Sixth nerve.
VII. Facial nerve.
VIII. Auditory nerve.
IX. Glosso-pharyngeal.
X. Vagus.
XI. Spinal accessory.
XII. Hypoglossal.
Cl. First cervical nerve.
C. Island of Reil.
Th. Optic thalamus (the Island of Reil having been removed).
i. Internal corpus geniculatum
e. External geniculatum.
h. Pituitary body.
tc. Tuber cinereum.
a. One of the corpora albicuntia.
Sy. Sylvian fissure.
×× Anterior perforated space.
× Posterior perforated space.
P. Cerebral peduncle
PV. Pons Varolii.
Ce. Cerebellum.
f. Fillet.
fl. Flocculus.
pa. Anterior pyramid.
o. Olive.
d. Anterior median fissure of cord.
V. Laieral tract of medulla.
Ca. Anterior column.
Cl. Lateral column.

the crus cerebri of each side and join at the optic commissure. Here there is a partial decussation (which will be described later), and thus the optic tracts pass into the optic nerves, which run through the foramina optica to the back of the eyeballs.

The ulterior destination of the optic tracts (on the side of the cerebral hemispheres) is to the cortex of the occipital lobe, which it reaches *viâ* the posterior part of the internal capsule, and the fibres which radiate therefrom to the cortex.

The olfactory nerve consists of the olfactory bulb and olfactory tract, and is regarded rather as a lobe of the brain than as a distinct nerve. The bulb lies on the upper surface of the ethmoid, through which fibres pass to the nasal fossa. The tract as it runs backward bifurcates, the one division or root goes towards the Sylvian fissure, the other towards the longitudinal fissure. The first of these divisions probably goes to the tip of the temporo-sphenoidal lobe and the gyrus uncinatus.

Region of crura cerebri.—Each crus cerebri connects its own hemisphere with the cerebellum behind, and the pons and medulla below. The two crura are fused as they approach the pons, and thus roof in the open space of the fourth ventricle, so as to form the aqueduct of Sylvius or iter a tertio ad quartum ventriculum. This region is surmounted by the two pairs of ganglia called the corpora quadrigemina. Deeper down in each crus is placed another ganglion, called the red nucleus. Just below the Sylvian aqueduct lie the nuclei of the oculo-motor nerves.

In a transverse section through the crura (Fig. 9 C, 17, 18) the division into ventral and dorsal parts—called respectively the crusta and the tegmentum—is marked, as we have said, by a layer of darkly pigmented cells called the substantia nigra.

The crusta—*i.e.*, the ventral or lower part—consists of the following tracts of fibres (Fig. 9 C):—

(*a*) In its middle third, the pyramidal or motor

tract, descending from the motor area of the cerebral cortex *viâ* the posterior limb of the internal capsule to the medulla and cord.

(β) In its internal third (*i.e.*, between the pyramidal tract and the middle line) a tract of fibres which descend from the cortex in front of the motor area (prefrontal region), *viâ* the anterior limb of the internal capsule, to the pons and thence to the cerebellum (fronto-cerebellar fibres).

(γ) In its external third fibres which descend from the cortex of the temporo-sphenoidal and occipital lobes, which have the same destination as the last set, but which have not (according to Flechsig) any place in the internal capsule (temporo-occipital cerebellar fibres).*

The tegmentum (Fig. 9 C, 16–18) is a more complicated region. It is surmounted, as just mentioned, by the anterior and posterior corpora quadrigemina. Each of these bodies, anterior and posterior, sends forward a brachium or tract of fibres which runs to that part of the optic thalamus from which the optic tract springs ; indeed, the brachium of the corpus quadrigeminum anterius is almost continuous with the optic tract. The corpora quadrigemina receive fibres from the fillet and from the reticular formation behind them. Probably they are also connected with the underlying oculo-motor nuclei. Their functions are still imperfectly known.

* These are the three principal divisions of the crusta, but there are sundry smaller divisions as follow :—

δ. The inner part of the motor tract that which lies next to the fronto-cerebellar fibres, is sometimes called the "geniculate" tract because it contains the fibres which come from the genu or knee of the internal capsule ; these are destined for the face and tongue (bulbar nuclei), whereas the bulk of the motor tract goes to the limbs.

ε. Deeply placed between the three main tracts and the substantia nigra are fibres which are supposed to run from the caudate nucleus to the pons, and thence to the cerebellum (like the fronto-cerebellar and temporo-occipital cerebellar fibres).

ζ. Fibres which connect the crusta with the fillet.

Beneath them, on section through the tegmentum, we find the Sylvian aqueduct, or iter a tertio ad quartum ventriculum, and around it.

α. Grey matter, part of which constitutes the nuclei of the third and fourth cranial nerves.

β. On either side, the small band of fibres which forms the descending root of the fifth nerve.

γ. Ventrally upon either side the posterior longitudinal bundle.

The main mass of the white substance of the tegmentum consists, however, of the superior cerebellar peduncles and the fillets of either side, the position of these varies according to the level at which the section is taken.

Thus, at the lowest level of this region (viz., at the junction of the pons and crura) each superior cerebellar peduncle on its way up from the corresponding side of the cerebellum is placed at the dorsal and lateral part of the section; but as it ascends brainwards, it tends to the deeper part and towards the middle line (Fig. 16), and reaching this it crosses over and decussates with its fellow (Fig. 17) (at a level midway between the superior and inferior corpora quadrigemina), and finally terminates (at the upper level of the superior corpus quadrigeminum) in a large round nucleus, called, from its colour, the red nucleus, or nucleus tegmenti (Fig. 18). This nucleus is pierced by the arching fibres of the third nerve, and forms a prominent object in a section at the upper part of the crura cerebri. It probably sends fibres upwards to the optic thalamus and lenticular nucleus.

Meanwhile the fillet, which at the upper part of the pons (Fig. 15) lay deeply between the dorsal and ventral portions of the nervous axis, splits up (as we have already said). Its lateral or inferior portion becomes superficial to the superior cerebellar peduncle as this tracks inwards, and winds round its outer edge to gain the inferior corpus quadrigeminum (Fig. 16); its mesial or superior portion passes mainly to the superior

FIG. 20.

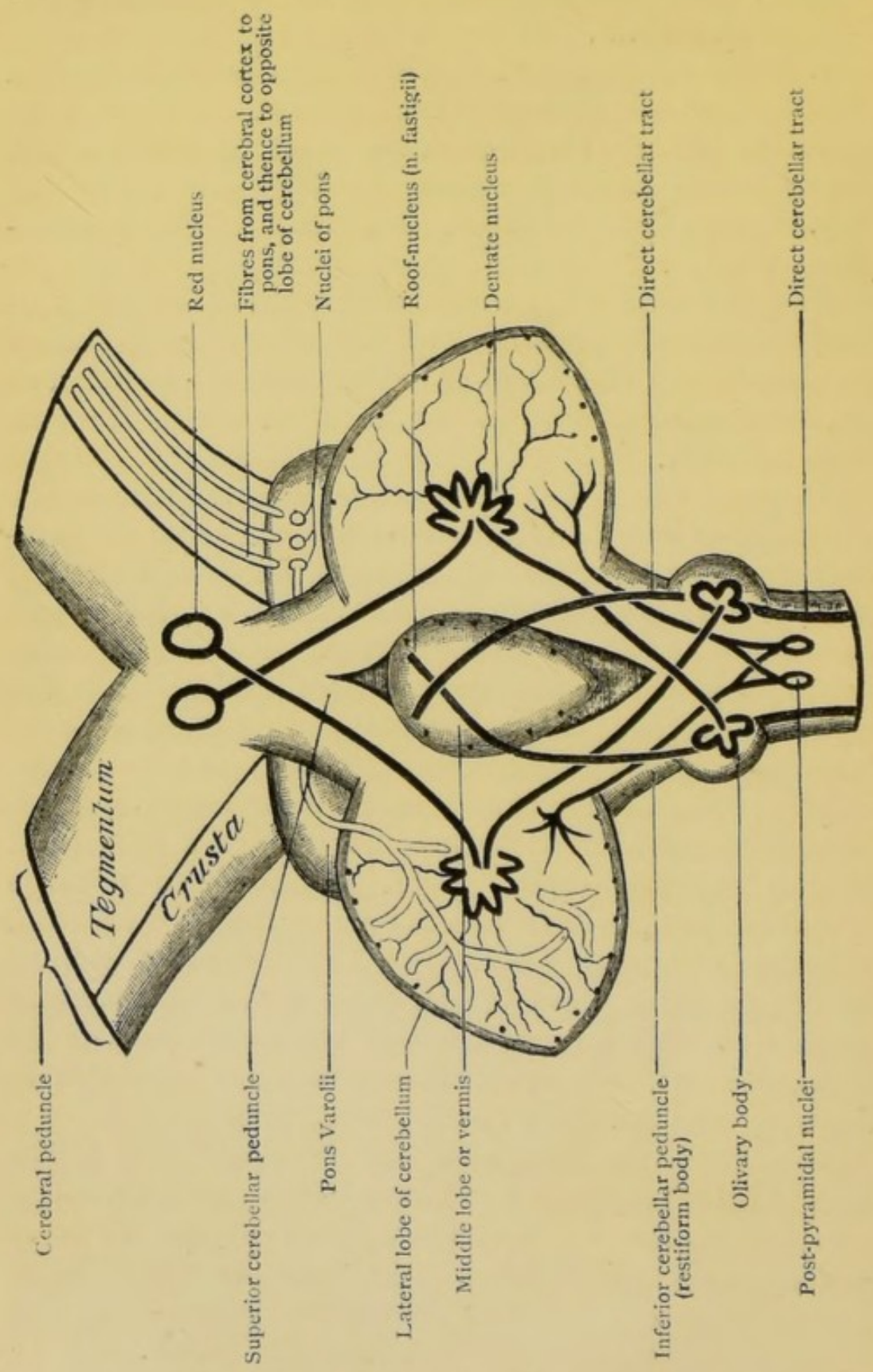


Diagram to show some of the connections of the cerebellum.

corpus quadrigeminum and to the optic thalamus, while a small part joins the crusta of the cerebral peduncle.

Cerebellum.—The cerebellum is an organ of peculiar structure as regards its minute anatomy. Its cortical substance differs singularly from that of the cerebrum, being characterised by the single layer of the large "cells of Purkinje," and the thickly set layer of granules, reminding us of those in the retina. The organ has two lateral lobes and a middle lobe or "vermis." Deeply placed in the white matter of each lateral lobe is a nucleus, consisting of a wavy capsule of grey matter, not unlike the olivary body, and called the dentate nucleus. In the middle lobe are nuclei known as "roof nuclei" (nucleus fastigii), because they lie in that part which roofs in the fourth ventricle.

The functions of the cerebellum are incompletely known. There is reason to think that the division into lateral and middle lobes corresponds to difference in function; and that the particular function commonly ascribed to the cerebellum—viz., the co-ordination of muscular movement required for balancing the body, is limited to the middle lobe.

At any rate, the connections of the cortex of the lateral hemispheres, of the nucleus dentatum of the lateral hemisphere, and of the middle lobe are different. They are illustrated in the diagram, and they are mainly as follows:—

Middle Lobe (vermis).—In the cortex of this lobe terminate the direct cerebellar tracts, which ascend from the cord, *viâ* the inferior cerebellar peduncles (restiform bodies).* Other fibres (not all shown in the diagram) are said to pass, *viâ* the restiform bodies, to the middle lobe—(1) from the formatio reticularis of the medulla; (2) from the auditory nuclei; (3) from the nuclei of the posterior pyramids of both sides.†

* Whether they decussate, as represented in the diagram, or no, is a little uncertain.

† Should be represented in the diagram as going to the middle (not lateral) cerebellar lobe.

The *dentate nucleus of each lateral hemisphere* is joined below to the olivary body of the opposite side by fibres which run in the inferior cerebellar peduncle. Upwards from the dentate body run fibres which form the bulk of the superior cerebellar peduncle; this peduncle, at first placed superficially at the dorsum and side of the pons, soon dips inward, decussates with its fellow in the middle line, and passes into the opposite red nucleus. From the red nucleus arise fibres (not marked in the diagram) which pass upwards probably to the lenticular nucleus and region of the optic thalamus (either into the thalamus itself or through it to the sensory area of the cerebral cortex).

The *cortex* of each *lateral cerebellar hemisphere* sends fibres to its own dentate nucleus. It is also connected with the opposite cerebral hemisphere in the following way (*vide* Fig. 9, C, and p. 38):—

(1) Fibres from the frontal lobe of the cerebrum descend through the internal capsule (anterior limb) and crus cerebri (internal third of the crusta) to the pons; crossing to the opposite side of the pons, they terminate in the cells scattered amongst its transverse fibres.* From these cells other fibres arise which run *viâ* the middle cerebellar peduncle to the cortex of the corresponding cerebellar hemisphere.

(2) Fibres from the temporo-sphenoidal and occipital lobes of the cerebrum descend in a similar way through the external third of the crusta (apparently missing the internal capsule) to the pons and thence to the cerebellum.

(3) Fibres from the caudate nucleus, deeply situated in the crusta, establish a like connection with the pons and cerebellum.

These connections are crossed, it will be noticed; each lateral lobe of the cerebellum is joined to the opposite cerebral hemisphere, the opposite side of the

* Should be represented in the diagram as connecting with cells on the *opposite* side of the pons.

spinal cord, and the opposite olive. Atrophy (or non-development) of one cerebral hemisphere is associated with atrophy of the other half of the cerebellum.

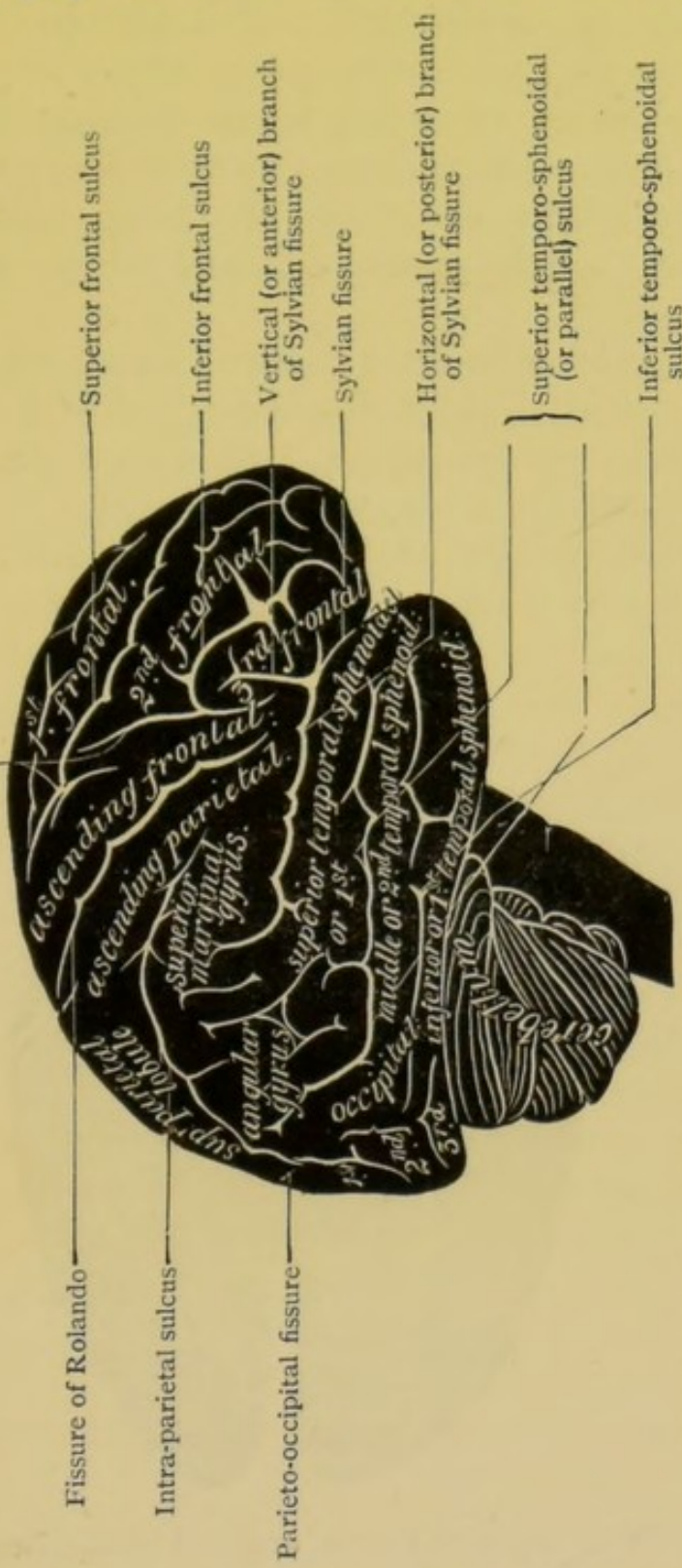


FIG. 21.*

Diagram of external surface of right cerebral hemisphere.

[ERRATUM.—For "*superior marginal*" read "*supra-marginal*" gyrus.

* This and the following diagrams of the cerebrum are on the model of the "Clinical Figures" published by Messrs. Daniellsen.

Similarly, atrophy of one cerebellar hemisphere is associated with atrophy of the opposite olivary body.

Cerebral hemispheres: their surface or

cortex.—Cerebral convolutions.—The cerebral convolutions, with the fissures and sulci which divide them, are indicated on the accompanying diagrams. Their arrangement is sufficiently constant to allow of diagrammatic representation; but every opportunity should be taken of studying them in nature, for the subdivision of the convolutions by subsidiary sulci, and the frequent slight variation of them, render their identification much more difficult than might be imagined.

Next to the longitudinal fissure, which receives the falx cerebri, and divides the two hemispheres from each other, the most important fissures are:—

The fissure of Rolando, which separates the frontal lobe in front from the parietal lobe behind, and upon either side of which are ranged the motor convolutions.

FIG. 22.

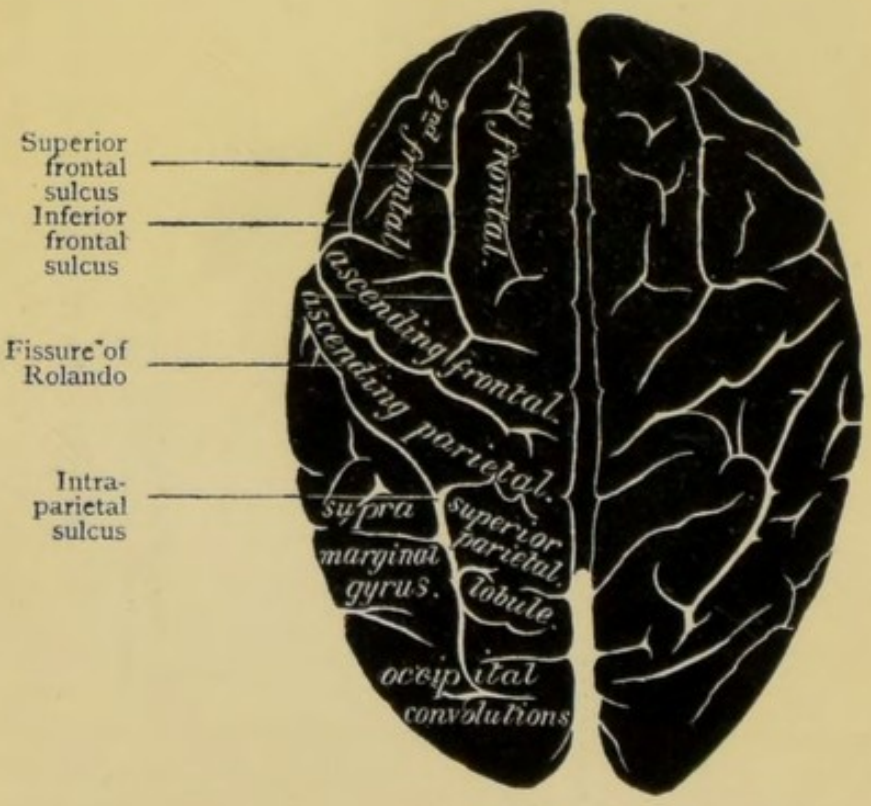


Diagram of upper surface of cerebral hemispheres.

The fissure of Sylvius, which separates the temporo-sphenoidal lobe below from the frontal and parietal lobes above. It begins as a wide-lipped opening

below, and divides into a long horizontal branch and a short vertical one.

The parieto-occipital fissure, seen upon the inner surface of the hemisphere, which separates the occipital lobe behind and below from the parietal.

Taking first the outer and upper aspect of the hemispheres (Figs. 21 and 22), we find on either side of the fissure of Rolando two parallel convolutions—viz., the ascending frontal and ascending parietal. The ascending frontal is separated by an interrupted sulcus (præcentral sulcus) from the three other frontal convolutions which run horizontally. These are called in order from above downwards—the first, or superior frontal; the second, or middle; the third, or inferior. The third frontal curves round the vertical branch of the Sylvian fissure, merging into the lower end of the ascending frontal. (The region where they join is sometimes called the operculum; it covers in the island of Reil below.)

The ascending parietal merges at its upper end into the superior parietal lobule, which occupies the upper part of the parietal lobe. A sulcus, partly horizontal and partly vertical in direction (intra-parietal sulcus), divides the superior parietal lobule above, and the ascending parietal convolution in front, from the remainder of the lobe. This remainder, which is sometimes called the inferior parietal lobule, contains the supra-marginal gyrus, which bends round the extremity of the posterior branch of the Sylvian fissure, and the angular gyrus,* which similarly bends round the end of the next principal sulcus—viz., the superior temporo-sphenoidal or parallel sulcus.

* The term angular gyrus needs an authoritative definition. It is sometimes used to denote the convolution which curves round the end of the parallel sulcus (as in text), sometimes made to include more or less of the space between the parallel sulcus and the termination of the Sylvian fissure, sometimes even to include what in the text is called supra-marginal gyrus. The distance between the ends of the parallel sulcus and the Sylvian fissure is variable in the human brain; in the monkey the parallel sulcus joins the Sylvian fissure.

Beneath the horizontal part of the fissure of Sylvius lies the temporo-sphenoidal lobe, which contains on its outer aspect three horizontal convolutions (superior, middle, inferior). A long sulcus (superior temporo-sphenoidal or parallel sulcus) separates the first or superior from the second or middle convolution. It runs back till terminated by the angular gyrus.

Behind the parietal and the temporo-sphenoidal lobes lies the occipital lobe. The line of demarcation is indefinite, being taken as the line which would be marked out by the parieto-occipital fissure if prolonged over the outer aspect of the brain. In monkeys this fissure actually does reach over the external aspect of the brain, but not in man.

Three occipital convolutions are described—superior, middle, and inferior. They are joined to the parts in front by "annectant" gyri.

On separating the lips of the Sylvian fissure, there are seen the convolutions of the island of Reil (not shown in the diagrams).

FIG. 23.

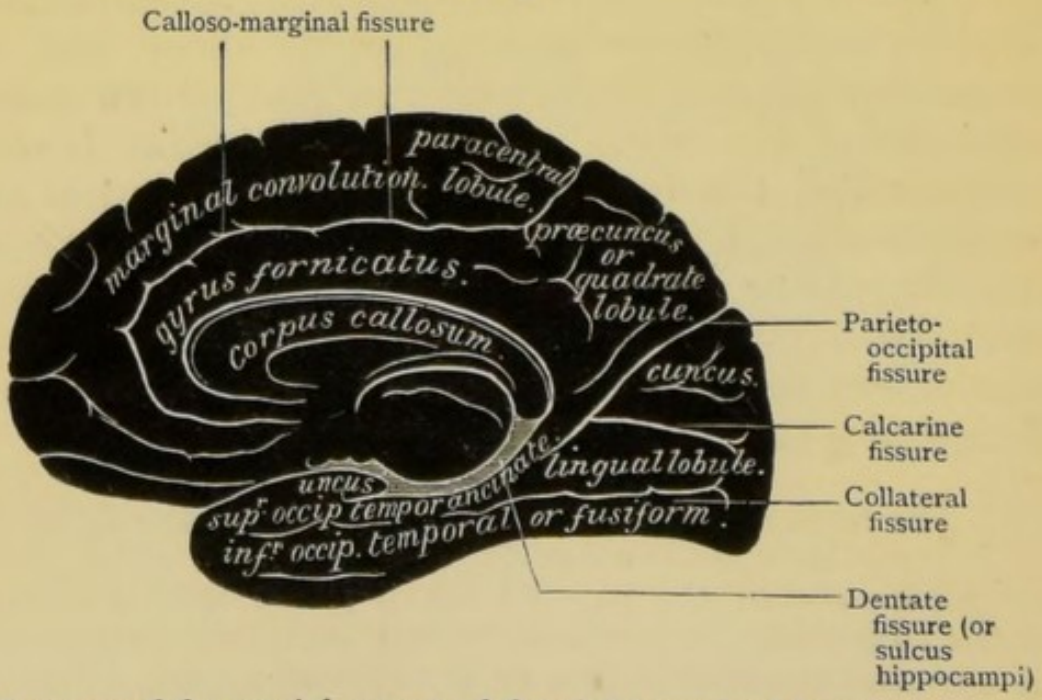


Diagram of the mesial aspect of the right cerebral hemisphere.

The convolutions upon the inner and lower aspects of the brain (Figs. 23 and 24) are more simple.

Immediately above the corpus callosum (the large commissure which connects the two hemispheres) lies the gyrus fornicatus. This runs parallel to the whole length of the corpus callosum, and then curves down behind it, to merge in the uncinate convolution. Parallel with the gyrus fornicatus and next above it, is the marginal convolution; the calloso-marginal fissure separates the two. Posteriorly, the fissure turns up towards the upper aspect of the brain, and thus terminates the marginal convolution. The greater part of this convolution corresponds to the superior or first frontal convolution on the outer aspect of the brain. But its posterior part corresponds to the upper ends of the ascending frontal and parietal convolutions, and this part is called the paracentral lobule. Behind this comes a roughly quadrangular area, corresponding to the superior parietal lobe externally. It is called the præcuneus or quadrate lobule. It is terminated posteriorly by the parieto-occipital fissure, behind and below which the occipital lobe begins. The parieto-occipital fissure runs downwards and forwards, and receives from behind a tributary running horizontally—viz., the calcarine fissure. These two mark off a triangular tract called the cuneus. Below the calcarine fissure, and reaching forwards along the under surface of the temporo-sphenoidal lobe, are the temporo-occipital convolutions. The first, or superior, begins beneath the calcarine fissure, where it is also called the lingual lobule, and fusing with the end of the gyrus fornicatus forms the gyrus hippocampi or uncinate convolution, which borders and extends round the end of the dentate fissure, where it is called the uncus (subiculum cornu Ammonis). The dentate fissure corresponds to the elevation of the hippocampus major within the descending horn of the lateral ventricle; therefore it is also called the sulcus hippocampi. Within it lies the margin where the grey cortical matter of the brain terminates, called the dentate convolution, or

fascia dentata. The second temporo-occipital convolution, also called the fusiform lobule, runs parallel to the first, along the inferior aspect of the brain. The collateral fissure separates the two. The next convo-

FIG. 24.

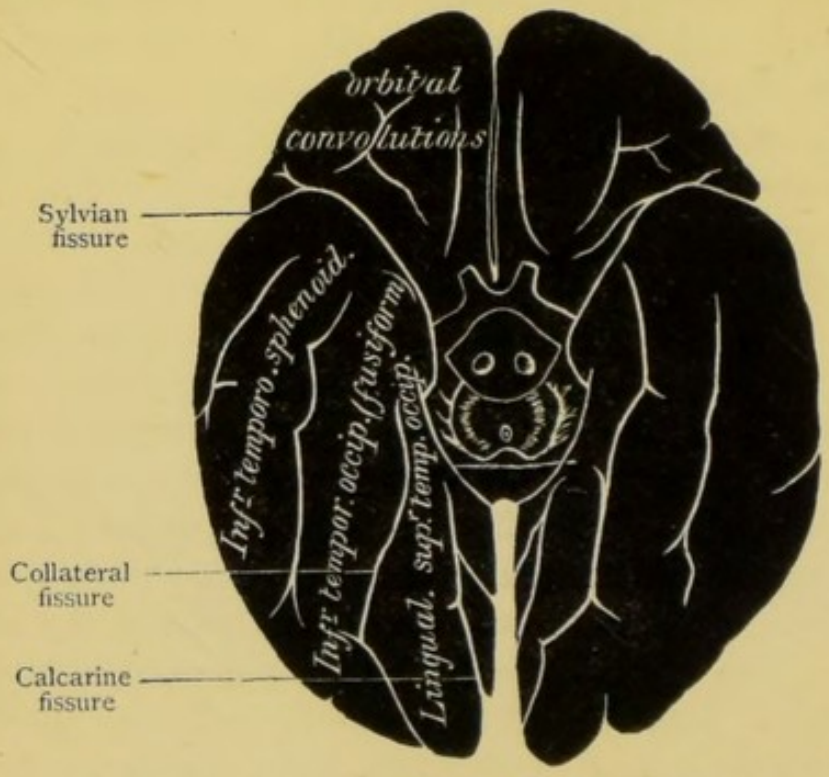


Diagram of lower surface of cerebral hemispheres.

lution upon the outer side of this is the third or inferior temporo-sphenoidal.

Functions of special parts.—The anatomy of the cerebral convolutions derives its importance from the fact that the brain is not (like such organs as the liver) functionally homogeneous, but that each part of the cortex has special functions assigned to it. Such a "localisation of function" in the brain is now denied by few, although the details of the theory are not completely known.

Motor "centres."—The function of voluntary movement is the easiest of investigation, and it is concerning the localisation of this that we have the most certain knowledge. Experiments on animals and observation of disease in man have shown certain parts of the brain to have a special connection with

voluntary movements of the several parts of the body, limbs, face, head, and eyes, &c., so that when these parts of the brain are stimulated such movements are produced; and when they are destroyed such movements become impossible. These parts of the cortex are therefore called the "motor area"; because the net result of activity in this area is movement, though what is the intimate nature of the cerebral processes we do not precisely know. Roughly speaking, the motor area consists of the parts near the fissure of Rolando, but it also extends on to the mesial aspect of the brain. Further, it can be parcelled out into smaller areas, which correspond to movements of particular limbs, and even of particular segments of them; these smaller areas are called the "motor centres" for the arm, leg, face, &c.

The ascending frontal and parietal convolutions are occupied by the centres for the face, arm, and leg (Fig. 25)—in that order from below upwards; each

FIG. 25.

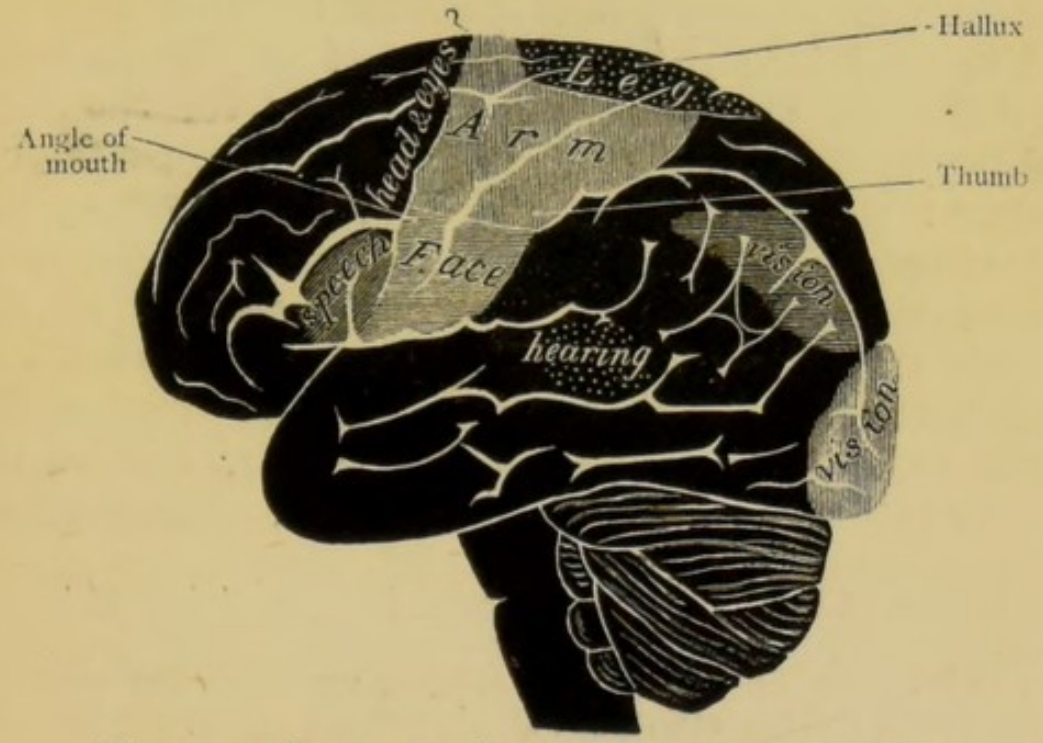


Diagram of motor and sensory centres on external surface of left cerebral hemisphere.

centre occupying (roughly speaking) a third of the whole length of the convolutions. There is no strict

line of demarcation between them, but it has been possible by experiments on monkeys to map them out fairly, and even to find the points corresponding to the several segments of the limbs, and particularly of the thumb, fingers, hallux, &c. Rare cases of limited disease have sometimes allowed a similar minute localisation in man.

The face centre occupies the lower ends of the two ascending convolutions. It is limited above (Horsley) by a line drawn forwards from the lower end of the intra-parietal sulcus parallel with the horizontal branch of the Sylvian fissure. In its upper part are probably represented movements of the upper part of the face, and in its lower part movements of the lower face, lips, tongue and vocal cords.* The posterior part of the third frontal convolution on the left side constitutes "Broca's convolution." Destruction of this part produces that condition of speechlessness which is commonly called aphasia.

The arm centre occupies the middle part of the central convolutions, but it probably runs forward and upward into the posterior part of the superior frontal (here blending with the centre for the leg), and to the mesial aspect of the brain. The particular parts of the arm are said to be represented in the following order from above downwards—shoulder, elbow, wrist, fingers, thumb. Movements of the thumb appear to be represented at the lowest and most posterior part of the centre, just in front of the lower end of the intra-parietal sulcus, and of the fingers just above and in front of this. The position

* Spasm of the angle of the mouth (zygomatici) was in one case dependent on a lesion of the ascending frontal just opposite the origin of the sulcus separating the 2nd and 3rd frontal convolution (Berkeley).

Adduction of the vocal cords (Horsley and Semon) is represented in the monkey at a point just posterior to the lower end of the præcentral sulcus—*i.e.*, in the ascending frontal at the level of the 3rd frontal. There is no centre for abduction of the cords.

of the centre for these parts is important because convulsions frequently commence in them.

The leg centre is at the highest part of the ascending frontal and parietal, probably not reaching down as far as the level of the superior frontal sulcus (Gowers), and extending to uncertain limits anteriorly and posteriorly. On the mesial aspect of the brain it occupies the paracentral lobule. Movements of the hallux are specially represented at a point just in front of the highest part of the fissure of Rolando.

FIG. 26.

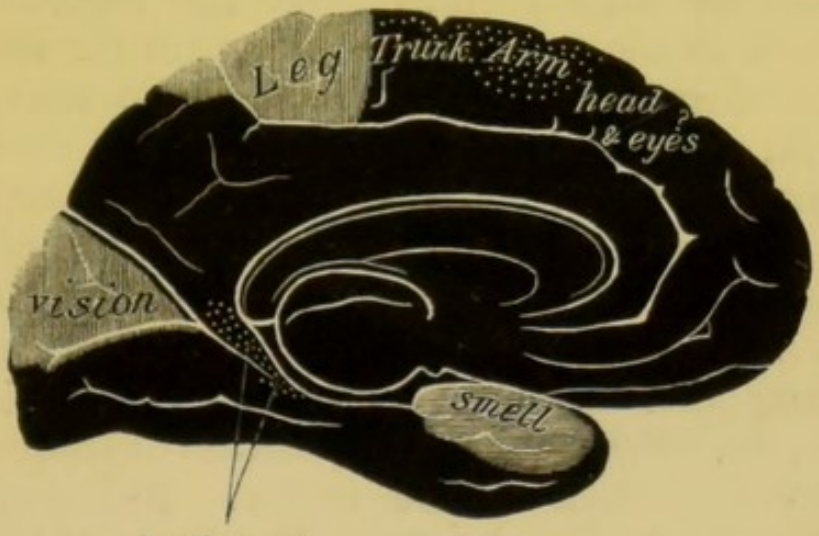


Diagram of motor and sensory centres on mesial aspect of left cerebral hemisphere.*

To complete this account of the motor area on the external aspect of the brain it should be said that there is in the monkey, and probably in man also, a largish area covering the posterior parts of the horizontal frontal convolutions, wherein are represented sideward movements of the head and eyes.

The motor area extends also on to the mesial aspect of the hemispheres (Fig. 26). The marginal convolution in monkeys (Horsley and Schäfer) contains motor centres, which may be enumerated in the following order from before backwards:—

* The centre for touch should probably extend much further forward along the gyrus fornicatus. (*Vide* Savill, *Brain*, 1891.)

1. For the head and eyes (a very small area).
2. For the upper limb (viz., forearm and hand, shoulder).
3. For the trunk (upper part, pelvic part).
4. For the lower limb (viz., hip, knee, foot, toes).

The areas 1, 2, 4 are continuous with those for head, arm and leg on the external aspect of the hemisphere. The centre for the trunk is almost entirely upon the mesial aspect. As might be expected, the centres for shoulder and hip are grouped on either side of the trunk centre.

In man we may surmise that the region of the paracentral lobule completes the centre for the lower limb, and that in front of this may lie centres for the trunk, shoulder, head and neck.

Sensory "centres."—By sensory centres we mean those areas of the cortex, stimulation of which gives rise to sensations such as those of sight, hearing, and the like, and destruction of which causes a corresponding disability of sensation—*e.g.*, blindness, deafness, &c. Concerning the position of these centres we know less than of the motor. The centre for vision was originally placed by Ferrier in the angular gyrus,* destruction of this part produced loss or impairment of vision in the opposite eye (crossed amblyopia). Others, as Munk, placed the visual centre in the occipital lobes, the relation being such that destruction of one (say the right) occipital lobe produced blindness in the opposite (left) half of the field of vision in *both* eyes (crossed hemianopia). As regards man there are facts in favour of both these views, and it may be that they are both true, the occipital lobe constituting a centre for the opposite half-field of vision, the angular gyrus for the vision of the opposite eye.† The centre for hearing is commonly

* Extending also to the supra-marginal convolution: *vide* diagram in Ferrier's "Functions of the Brain," ed. 2, p. 478.

† And probably in a less degree for the eye of the same side also.

placed in the posterior part of the superior temporo-sphenoidal convolution: but this is disputed by some authorities.

The centre for smell is placed at the tip of the mesial aspect of the temporo-sphenoidal lobe (region of the uncus); and that for taste is probably in the same neighbourhood.

Cutaneous sensation is thought to be localised in the uncinate gyrus and it's continuation, the gyrus fornicatus—parts which as a whole have been called the falciform or limbic lobe.

White matter of cerebral hemispheres.—The interior of the hemispheres consists partly of white matter, partly of the grey masses known as the basic ganglia (corpus striatum, and optic thalamus).

The white matter, which we will consider first, serves solely to establish paths of connection between the cortex, the ganglia, and other parts. These connecting fibres are classified into (1) Commissural fibres, passing from hemisphere to hemisphere, which chiefly run in the corpus callosum and commissure of the third ventricle. (2) Longitudinal or "collateral" fibres, which connect different parts of the cortex of the same hemisphere, and which run partly in the fornix, gyrus fornicatus, and other structures, partly immediately beneath the cortex, where they are known as "association fibres." (3) Peduncular fibres, which pass from the cortex to the crura or peduncles of the brain, and thence to the pons, cerebellum, and spinal cord. To these must be added fibres which connect the basic ganglia with the peduncles below, or with the cortex above.

Of these sets the peduncular fibres are the best known. They converge from all parts of the cortex, forming thus what is known as the corona radiata, till they are collected together into a flat band, called "the **internal capsule.**" This important structure is shown as cut transversely in the horizontal section figured in Fig. 27. It lies between the lenticular

nucleus, on its outer side, and the caudate nucleus and optic thalamus on its inner side. It has, in this section, the shape of a V, with its apex pointing to the middle line, and its limbs spread out to an obtuse angle. There is thus an anterior and a posterior limb

FIG. 27.

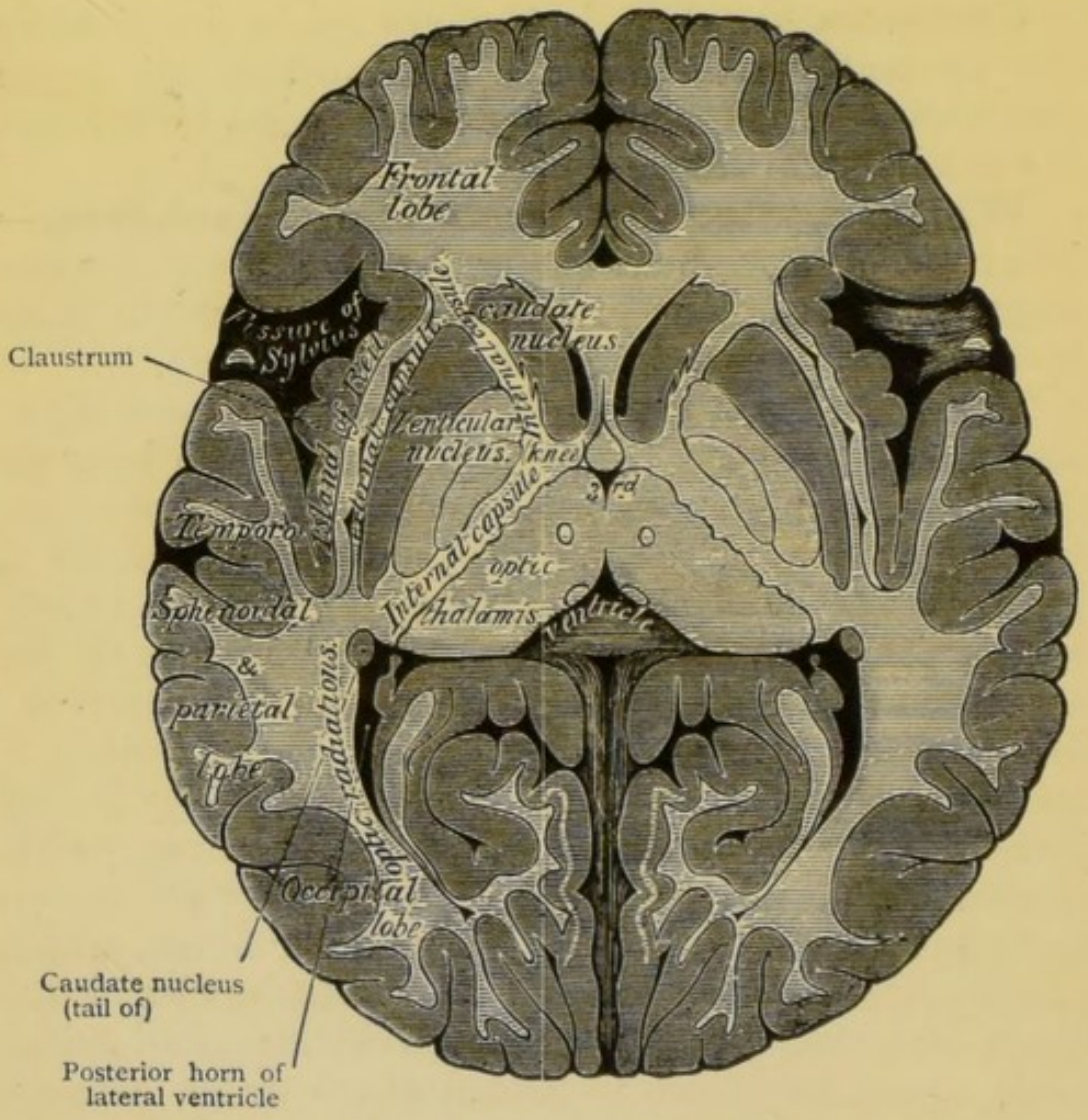


Diagram to show the parts exposed by a horizontal section through the hemispheres, at the level of the basic ganglia.

or division in the capsule, and their point of junction, apex of the V, is called its genu or knee. The fibres in its anterior limb come from the cortex in front of the motor area (prefrontal area), and descend, as mentioned above (p. 38, β, and p. 42, 1), through the crusta of the cerebral peduncle to the pons and opposite lobe

of the cerebellum. The genu and anterior two-thirds of the posterior limb of the capsule are occupied by fibres which descend from the motor area of the cortex to the spinal cord. In the capsule these motor fibres are arranged in a certain order from before backwards, roughly speaking, thus : those for the face, tongue, &c., at the genu, then those for the upper limb, and then those for the lower ;* so that what is inferior in the

FIG. 28A.

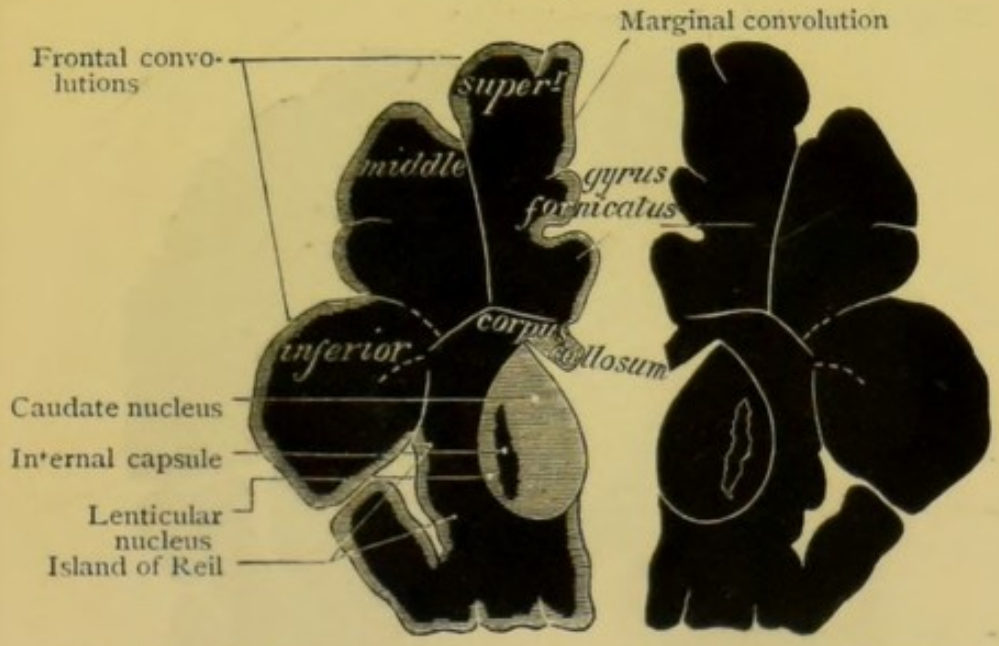


Fig. 28.—Transverse vertical sections (diagrammatic) through the cerebral hemispheres.
A. Through the posterior part of the horizontal frontal convolutions (pediculo-frontal section of Pitres).

cortex is anterior in the capsule. The remaining (posterior) third of the capsule consists of fibres going upwards to the sensory parts of the cortex.

The general direction of the fibres of the internal capsule—viz., as a tract which connects the corona

* Beevor and Horsley in experiments on monkeys found that the fibres had the following relative position from before backward :—

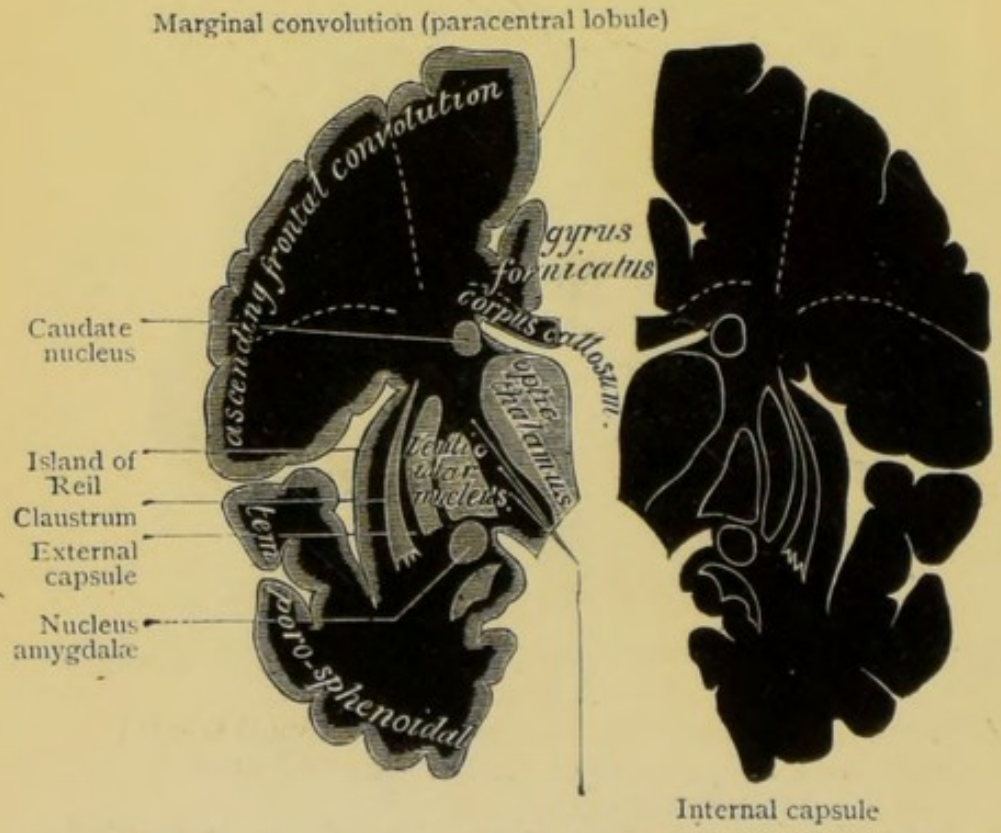
(1) Fibres for movement of the eyes.
(2) " " head.
(3) " " tongue.
(4) " " mouth.
(5) " " upper limb.
(6) " " lower limb: hip, knee, toes.

radiata above with the crura cerebri below, is shown better in vertical sections (Figs. 28B, 28C).

Further back than the internal capsule, curving round the posterior horn of the lateral ventricle, towards the occipital cortex, is a tract of fibres known as the optic radiations. It connects the cortical centre for vision with the termination of the optic tract below (p. 37).

The upper surfaces of the large **basic ganglia**—viz.,

FIG. 28B.



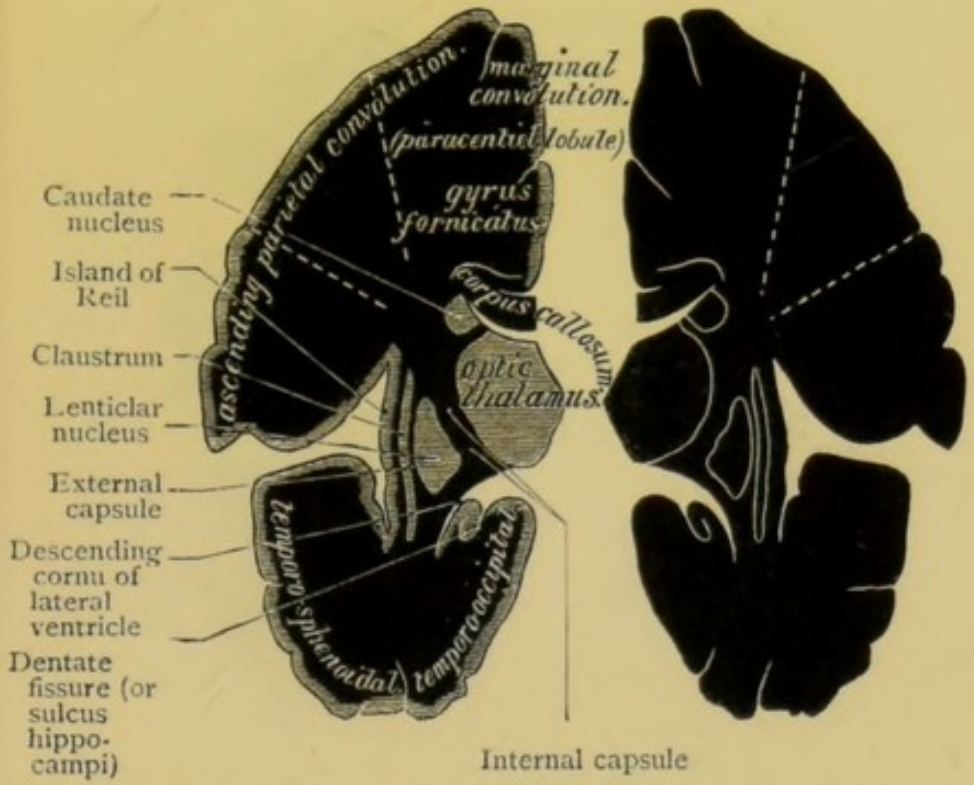
B_2 Through the ascending frontal convolution (frontal section of Pitres).

the corpus striatum and optic thalamus, are seen as soon as the ventricles are exposed from above. For further examination the horizontal and vertical sections (Figs. 27 and 28) may be used. The so-called corpus striatum consists of two parts, caudate nucleus and lenticular nucleus. The **caudate nucleus** is the only one that appears in the floor of the ventricle. The main part of this lies anteriorly, and is separated from the lenticular nucleus by the anterior limb of the internal capsule. But it sends a long tail or cauda

backwards, which arches over the optic thalamus and curves downwards so as to appear in the wall of the descending cornu of the lateral ventricle. At its anterior part the caudate nucleus is partly fused with the lenticular nucleus.

The **lenticular nucleus**, placed externally, fills up the angle made by the diverging limbs of the internal capsule: the posterior limb of the capsule separates it from the optic thalamus and the anterior

FIG. 28C.



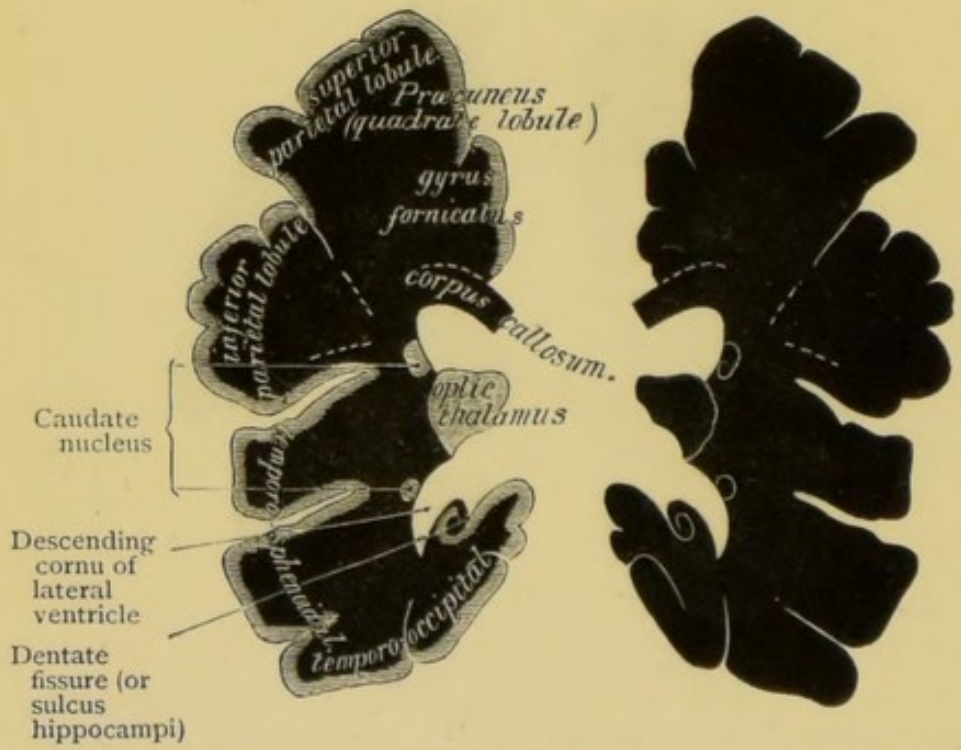
C. Through the ascending parietal convolution (parietal section of Pitres).

limb separates it from the caudate nucleus. The inner part of the lenticular nucleus is less dark than the outer, and by some authors is called the "globus pallidus"; the outer part is called the "putamen." The caudate nucleus (like the præfrontal cortex) is connected to the pons, and ultimately to the cerebellum, by fibres which descend through the crusta of the cerebral peduncle (*vide* p. 42, 3, and foot-note to p. 38, ϵ). To the lenticular nucleus, or at least to the globus pallidus, runs a band of fibres from the tegmentum

of the peduncle. This band is called the lenticular loop. It goes transversely across the internal capsule and underneath the optic thalamus, and connects the lenticular nucleus with the red nucleus and superior cerebellar peduncle, and also with the fillet.

The **optic thalamus** is extensively connected above with all parts of the cerebral cortex by fibres which spread out in the corona radiata; fibres also run in the fornix, which connect it with the hippocampal

FIG. 28D.



D. Through the supra-marginal gyrus and superior parietal lobule (pediculo-parietal section of Pitres).

gyrus or uncinate convolution. The posterior extremity of the thalamus is called the "pulvinar." It exhibits the two eminences known as the external and internal corpus geniculatum (Fig. 19). This region is important as constituting the origin of the optic tracts. Upon the side of the cerebral peduncles the optic thalamus is connected chiefly to the tegmentum—viz., to the red nucleus and superior cerebellar peduncle, brachia of corpora quadrigemina, reticular formation and fillet (?).

The **functions** of the basic ganglia are still doubtful. Till recently it was thought that they were placed—the corpus striatum upon the path of the efferent fibres from the cortex, the optic thalamus upon the path of the afferent fibres; so that motor impulses underwent some modification in the corpus striatum, and sensory impulses in the optic thalamus. But we know now that a large bulk of motor fibres proceed straight downwards from the cortex *viâ* the internal capsule, to the pons and cord, without any interruption in the corpus striatum. Very probably sensory fibres ascend in the same way, without interruption in the optic thalamus. It has been suggested that the caudate nucleus and the external part at least of the lenticular nucleus are really infolded portions of the cortical grey matter. As to the optic thalamus, the connection of its "pulvinar" with one important sense, that of sight, cannot be denied; but the function of the remainder is doubtful.

External capsule: claustrum, &c.—Before leaving these regions, we may add, to complete the description of Figs. 27 and 28, that the layer of white matter just outside the lenticular nucleus is called the external capsule. Its function is not known. Outside this, again, is a thin layer of grey matter known as the "claustrum," also of unknown function, but supposed by some to be essentially part of the grey matter of the island of Reil, under which it lies.

Vessels of cerebrum.—The vascular supply of the cerebrum is important, because some of the commonest cerebral diseases originate primarily in the vessels and not in the nerve-tissue, and therefore the distribution of the disease follows the distribution of the vessels. The arteries of the cerebrum all spring from the circle of Willis, that is, from the well-known arterial ring formed at the base of the brain by the communications between the basilar artery behind and the two internal carotids laterally. The trunks which arise from the circle of Willis are the two anterior

cerebral arteries which run on the inner aspect of either frontal lobe in the longitudinal fissure; the two middle cerebrals which are practically continuations of the carotid trunks, running in the fissures of Sylvius; and the posterior cerebrals which wind backward and outward over the crura cerebri to the occipital lobes. These main trunks are distributed in two ways—(1) By "cortical" branches, which ramify in the meninges and are distributed to the cortex and subjacent white matter; (2) By "central" arteries, small twigs which pierce the base of the brain directly and are distributed to the large ganglia and their neighbourhood. These central arteries form no anastomoses; hence occlusion of any such artery is followed by softening of all the brain-district supplied by it. Whether the cortical arteries anastomose is uncertain; but at least their anastomoses, if such exist, are not so large and regular as those in other parts of the body.

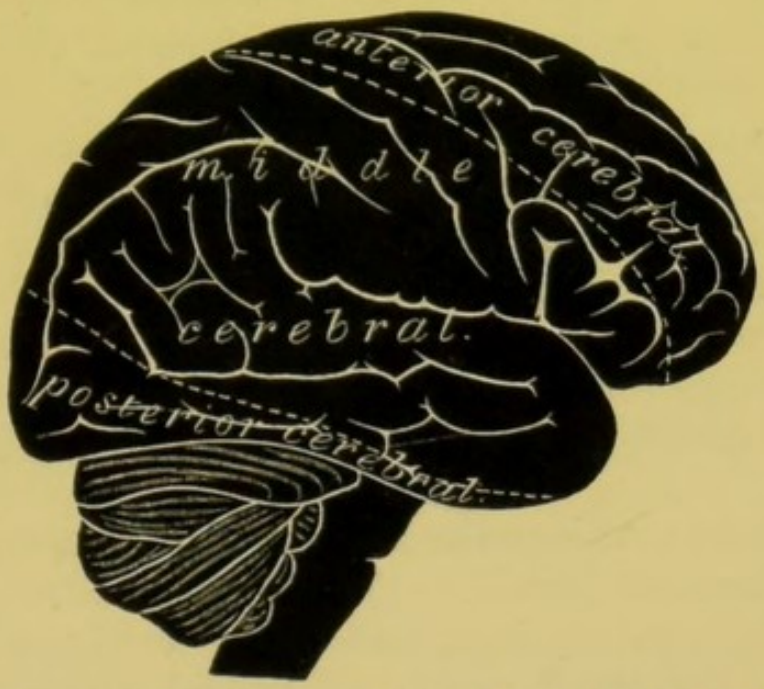
The *central* distribution of the anterior cerebral is small, taking in only the head of the caudate nucleus. That of the posterior cerebral includes the crus cerebri, choroid plexus, and posterior part of the optic thalamus and internal capsule. That of the middle cerebral is most important, for this artery supplies the corpus striatum (except the anterior part of the caudate nucleus), the optic thalamus and internal capsule (except their posterior extremities). This is effected by two sets of branches—(*a*) small twigs which run straight up from near the origin of the middle cerebral, (*β*) larger branches which course outwards in the external capsule, skirting the lenticular nucleus. One of these latter, from its great liability to give way in disease, has been called by Charcot the "artery of cerebral hæmorrhage."

The *cortical* distribution of the three arterial trunks is represented in Figs. 29 and 30.

Externally, the branches of the middle cerebral cover the largest area—viz., the ascending frontal

and parietal convolutions (except the upper part of the former), the roots of the middle and inferior

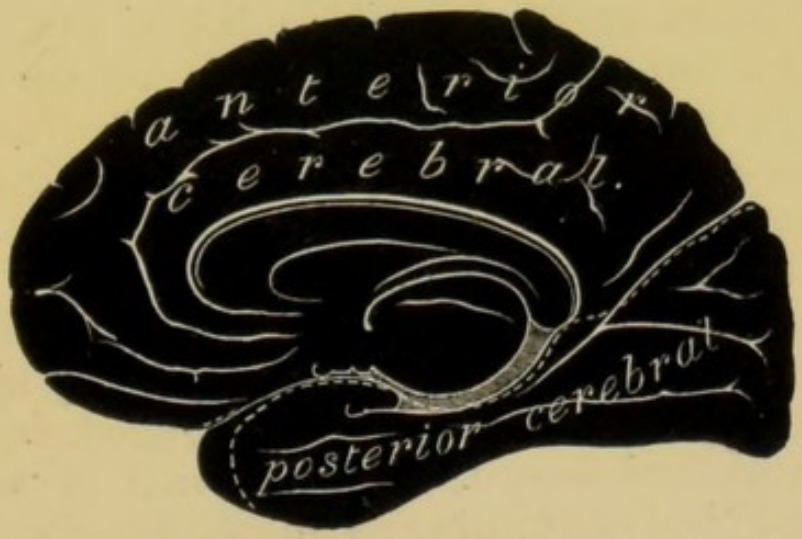
FIG. 29.



Distribution of arteries on external surface of cerebral hemisphere.

frontal, the superior and inferior parietal lobules, the upper two temporo-sphenoidal convolutions, and the

FIG. 30.



Distribution of arteries on mesial surface of cerebral hemisphere.

island of Reil. That is to say, the whole external motor area (except part of the lower limb centre) and

the speech centre on the left side, and a large part of the supposed sensory area (auditory and angular). The rest of the temporo-sphenoidal lobe and the occipital lobe are supplied by the posterior cerebral; the anterior and upper parts of the frontal lobe by the anterior cerebral.

On the mesial surface the conditions are reversed, for the middle cerebral supplies none of this surface; it is divided between the other two arteries; the posterior cerebral supplies the occipital ("half-vision" centre) and temporo-sphenoidal convolutions; the anterior cerebral all the convolutions in front of this, embracing therefore the motor area of the mesial surface.

Relations of cerebral convolutions to points on surface of skull.—The anatomical relations of the several convolutions and fissures to the external surface of the skull must be studied with exactitude by surgeons who engage in operations on the brain.

The simplest rules upon this subject are those given by Hare, and it may be sufficient to repeat them here.

The glabella is the root of the nose in the middle line opposite the upper border of the orbits.

The inion is the external occipital protuberance. A line drawn from glabella to inion through the vertex is called the sagittal line. It corresponds to the longitudinal fissure. Its length is from 11 to 13 inches as a rule. Its middle point is called the mid-sagittal point.

Half an inch (Thane), or three-quarters of an inch (Makins), behind the mid-sagittal point, lies the upper end of the fissure of Rolando, or, strictly speaking, the point where this fissure, if prolonged, would cut the longitudinal fissure. The direction of the fissure of Rolando is downwards and forwards, so as to form an angle of 67° with the sagittal line. This may be conveniently marked upon the skull by Horsley's method. Two strips of soft metal are joined so as to make the required angle; they are then moulded to the skull,

with the inner border of one along the sagittal line and the angle $\frac{1}{2}$ or $\frac{3}{4}$ inch behind the mid-sagittal point; then the inner border of the other will correspond to the fissure of Rolando.

The fissure of Sylvius is found by drawing a line backwards from the external angular process of the frontal bone backwards to the external occipital protuberance, and marking a point on it $1\frac{1}{8}$ inch from the external angular process. This point marks the origin of the fissure, and a line drawn from it to the centre of the parietal eminence marks the course of its horizontal branch.

The parieto-occipital fissure is two inches behind the upper end of the fissure of Rolando.

CHAPTER II.

MORBID ANATOMY OF NERVOUS SYSTEM.

We shall endeavour in this chapter to give a short account of the commonest morbid processes which affect the nervous system; limiting ourselves to those which produce definite anatomical changes.

Such diseases may either (1) arise primarily in the nerve elements or parenchyma, or (2) may begin in its envelopes, vessels, or interstitial tissue, the nervous tissue suffering secondarily.

DISEASE OF NERVOUS PARENCHYMA.

Primary parenchymatous disease is perhaps the least common of the two. It may be either inflammatory or degenerative. The line between the two classes cannot always be drawn.

Acute inflammation and its results.—Acute inflammation of nerve-tissue is more easily recognised by its results than during the early stages of the process. In the brain we may perhaps take as evidence of inflammatory hyperæmia, a general pink suffusion or discoloration, with numerous and well-marked "puncta cruenta." Mere fulness of the meningeal veins, particularly if limited to the posterior part of the head, is no criterion, since it may depend on post-mortem gravitation of the blood.

Cerebral softening.—But the first result of inflammation—viz., softening—has more definite

characters. The tissue has a reddish colour from the admixture of blood and blood pigment, the contour of white and grey matter is ill-defined, the nerve-substance is soft and can be washed away with a stream of water. The red colour tends to disappear when the condition has existed for any length of time; "white softening" rather than "red softening" is then said to exist. The colour is indeed from the beginning not an essential point, depending merely on the amount of blood in the tissues. In such softened patches, when the detritus is examined microscopically, may be seen remains of nerve-elements, particularly of white fibres, in which the myeline is breaking up; swollen and indistinct axis cylinders and nerve-cells; large corpuscles containing fatty granules, which doubtless come from the degenerating nerve-fibres.

Acute inflammation with softening may occur in the brain or cord; acute inflammation of a nerve-trunk affects the connective tissue primarily in most instances; but in the small nerve-branches acute parenchymatous disease occurs, concerning which we can hardly say whether it should be called inflammatory or degenerative.

Abscess of brain.—A further result of inflammation, in the brain at any rate, is abscess. Such abscesses may occur in various parts of the brain; perhaps the commonest seats are the cerebellum and the temporo-sphenoidal lobes. The principal conditions that lead up to intra-cranial abscess are—injuries to the head, disease of the cranial bones, especially of the petrous bone in connection with old middle-ear disease, otorrhœa, accumulations of pus in the pleura, or in dilated bronchi, pyæmia. Such an abscess may attain to a considerable size, and last a long time without giving rise to definite symptoms. When of long standing it has a distinct wall, which separates it from comparatively healthy brain; in more recent or progressive cases the sides are ragged and formed of diffusely softened brain tissue. The contents are often

very foul. Such abscesses may prove fatal by setting up acute inflammation and softening in their neighbourhood, or by perforation into the ventricles, or into the meninges, setting up an acute meningitis.

In **chronic inflammation** of the nerve-centres, the results of the process are chiefly evidenced by changes in the interstitial tissue. The neuroglia becomes thickened, and its nuclei increased in number. The small vessels are enlarged and multiplied. The nervous elements are broken up as in acute inflammation, but there is more time for the removal of their products; hence little may be seen of them, save spaces left empty in the thickened neuroglia, or filled with broken-up myeline. The result of the interstitial overgrowth is to produce a condensation, rather than softening, of the tissues, to which the term "sclerosis" is sometimes applied. A similar result is produced when there has been a slow destruction of the nerve-elements from other causes; thus, when there has been a primary degeneration of these elements, interstitial overgrowth follows, so that before long it is impossible to determine microscopically whether the disease originated in the parenchyma or the interstitial tissue.

Degeneration.—We will next consider this process of degeneration, which is one of great importance in nervous pathology.

The simplest instance of degeneration is that which occurs in a motor nerve-trunk after section. This has been called "Wallerian degeneration" of **nerve-trunks**, after Augustus Waller, the physiologist who first described it. The motor fibres of the nerve-trunks issue, as we have seen, from the large cells of the anterior grey horns of the spinal cord. One function of these cells is to maintain the nutrition of the fibres, and of the muscles to which they are distributed. Hence, when this trophic influence is removed, either by the experimental process of cutting the nerve, or pathologically by destruction of the cells

(acute anterior polio-myelitis), or destruction of the nerve-trunk (acute neuritis or injury from without), degenerative changes set in as follows:—

The axis cylinders, according to most authorities, degenerate and disappear.

The medullary substance breaks up, first into blocks, then into rounded masses of various sizes; then it undergoes gradual absorption.

Meanwhile, the nuclei within the sheath of Schwann multiply and enlarge; their protoplasm increasing apparently at the expense of the disappearing myeline.

The final result of the process is the disappearance of all the proper nerve-elements, leaving only an empty and shrivelled sheath of Schwann.

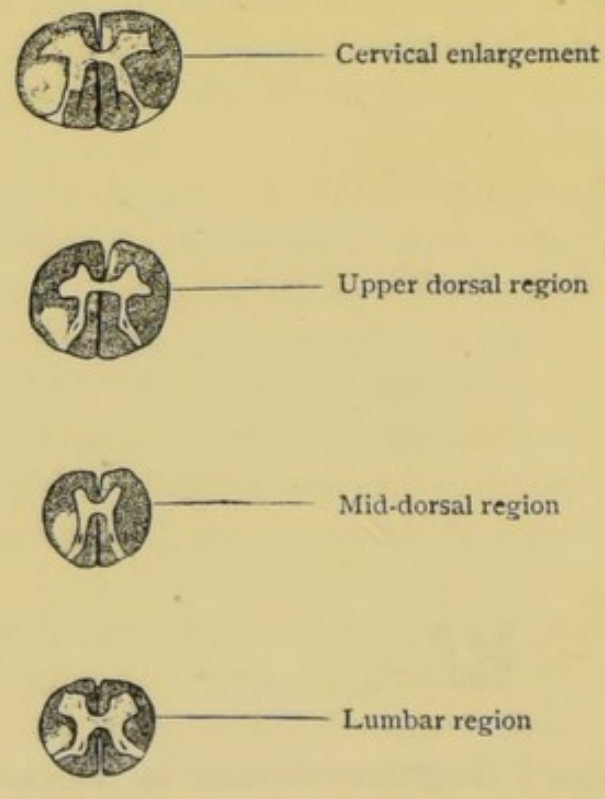
Changes also go on in the muscles, the terminal nerve-organs (end-plates) atrophy; the nuclei of the muscle fibres multiply, while the contractile substance loses its striation; the interstitial substance increases, and eventually, if the process continues unchecked, replaces the muscle tissue.

The conditions which produce this degeneration are destruction of the trophic cells, or a break in the axis cylinders which connect them with the parts below.

Secondary degeneration in nerve-centres.—Analogous changes take place in the fibres of the central nervous system. Taking first the best known set of fibres—viz., the motor—we observe that at their origin in the cortex cerebri are placed large nerve-cells, which have the same relation to them as have the anterior cornual cells to the motor fibres of the peripheral nerves. If these cells are destroyed by disease of the cerebral cortex, or their connection with the fibres below severed, as by cerebral hæmorrhage or softening, involving the internal capsule, or by transverse lesion of the cord, then degeneration commences in the fibres below the lesion. The process consists in changes in the axis cylinders, which first enlarge, then break up; breaking up of the myeline

into a granular detritus, then fusion of the diseased axis cylinders and myeline sheaths; subsequently, the neuroglia increases, and at length replaces the nerve-elements (sclerosis). These changes are analogous to, but not identical with, the Wallerian degeneration of nerves, the difference being that (1) in the nerve-trunks there is an active (? inflammatory) process—

FIG. 31.



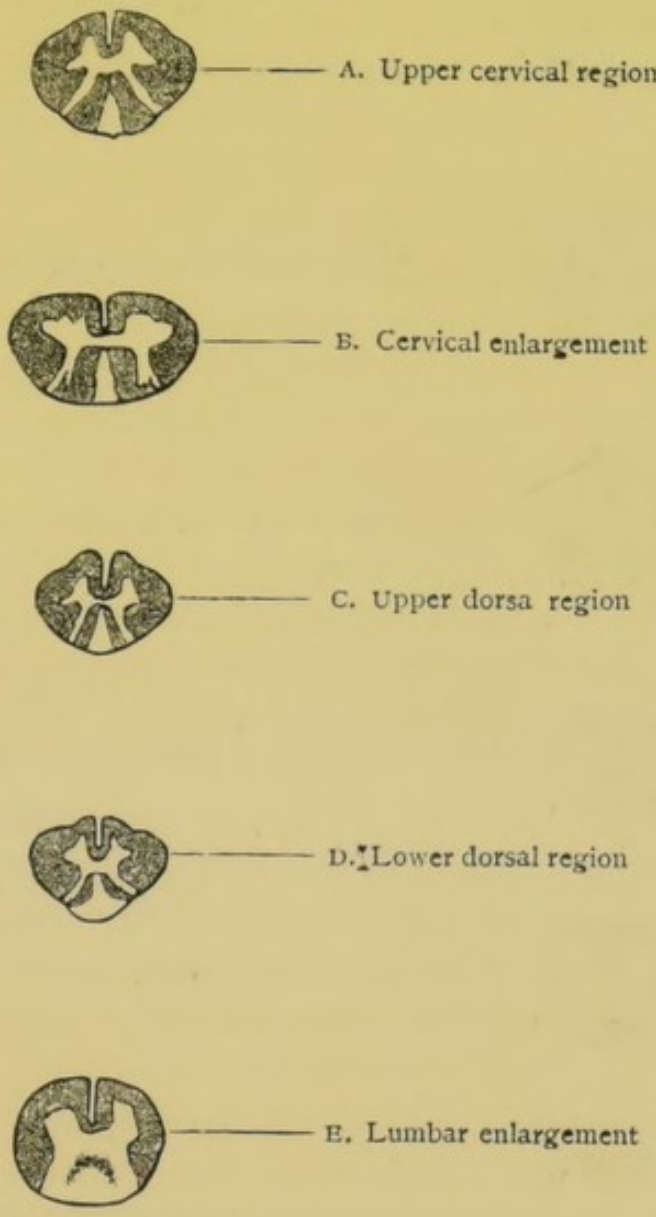
Secondary descending degeneration of pyramidal tract (crossed and direct) from a case of cerebral tumour.

viz., the multiplication of the nuclei of the sheath of Schwann, while in the cord this sheath is wanting; (2) in the cord the earliest changes are in the axis cylinder, whereas in the nerve-trunks the condition of the axis cylinder is a little uncertain; (3) that in the nerve-trunks regeneration may take place, whereas in the cord this has never been demonstrated. The length of fibre affected by this degeneration in the cord extends from the level of the lesion to that of the cells of the anterior grey horns, and here it stops.

The same thing may take place in afferent nerve-fibres. For instance, suppose the tract known as the

posterior median column is cut off from its cells of origin, which are probably situated in the ganglia of the posterior nerve-roots, either by disease of these

FIG. 32.



Secondary ascending degeneration of the postero-median columns.

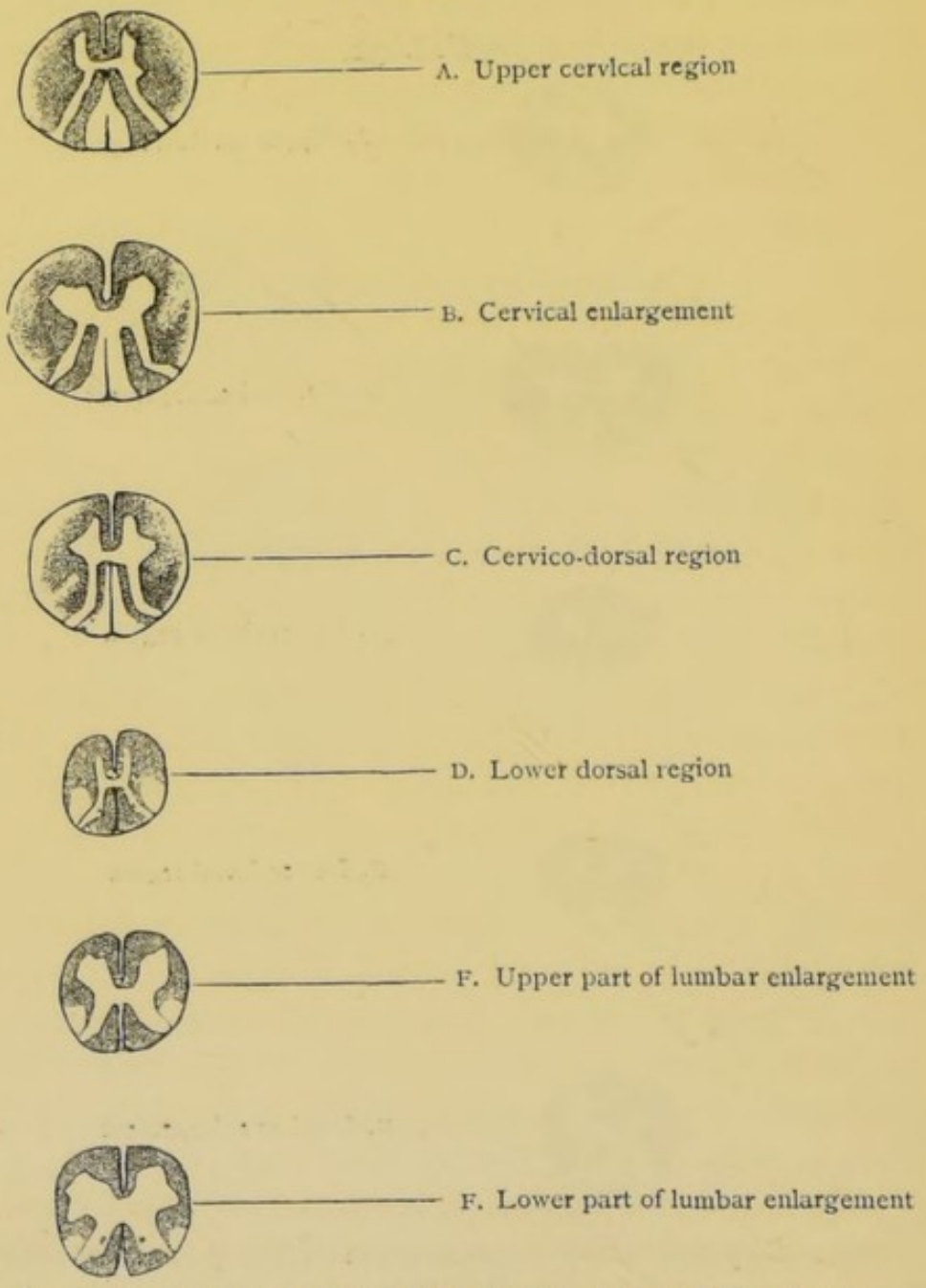
From a case of tumour involving the cauda equina and lower part of the cord.

In E the postero-lateral columns are involved, above this only the postero-median. The cerebellar and antero-lateral tracts are healthy.

nerve-roots or by a transverse lesion of the cord, degeneration of the tract sets in, extending upwards from the lesion to the cells of the post-pyramidal

nuclei in which the tract terminates. Similar degeneration occurs in the fibres of the cerebellar tract,

FIG. 33.



Secondary degeneration, after transverse myelitis of 3rd dorsal segment of cord.

In A, B, and C the degeneration is ascending, and occupies the postero-median columns, the direct cerebellar, and antero-lateral tracts.

In D, E, and F the degeneration is descending, and occupies the crossed pyramidal tracts.

and of the antero-lateral tract under appropriate conditions. Such degeneration is called "ascending"

when it occurs in afferent fibres, "descending" when in efferent fibres, from the direction, up or down the cord, in which it is propagated.

The process in all these cases is known as "secondary degeneration," and it is governed by the following laws:—

1. That fibres degenerate when separated from their cells of origin, or when those cells are destroyed.

2. That the degeneration reaches in the direction in which the fibres normally conduct, towards centre or periphery, as the case may be—*i.e.*, there is no degeneration of afferent fibres below a lesion, nor of efferent above it. So invariable has this rule been found, that conversely the function, afferent or efferent, of a nerve-tract may be inferred from the direction in which its fibres degenerate.

3. That the degeneration stops short at the nerve-cells in which the fibres terminate.

This limitation of the disease to definite nerve-tracts, and the definite direction taken by it in the line of their normal conduction, seem to show that its primary seat is in the nerve-elements and not in the interstitial tissue. Yet, as has been said, the perishing of the nerves appears to involve overgrowth of the neuroglia, so that when the process has existed for any time it may be absolutely indistinguishable, so far as microscopic appearances go, from chronic inflammatory disease affecting the neuroglia primarily. "Sclerosis" is the result in either case.

Primary degeneration in nerve-centres.—In addition to this "secondary" process of degeneration, caused by disease or injury which separates the fibres from their trophic centre, it appears that degeneration may arise independently of such lesion. It is then called "primary." Such primary degeneration may be "systematic" or "diffuse." It is (*a*) **systematic** when one or more definite nerve-tracts are picked out by the disease, much as in secondary degeneration. Thus the tracts of the pos-

terior columns degenerate in ordinary tabes, and the pyramidal tracts in amyotrophic lateral sclerosis; sometimes they both degenerate together. The more strictly "systematic" such disease is, the greater is the probability that it is primarily nervous and not interstitial. But on this point some uncertainty may prevail—first, because such primary degeneration does not limit itself to known nerve-tracts with such accuracy as does the secondary form; secondly, because our knowledge of its minute anatomy in the early stage is imperfect, and that of the later stages, as pointed out above, does not help us.

(β) **Diffuse** degeneration is unselective, irregularly situated, and spreads along no definite lines. The typical example of this is the disease known as insular or disseminated sclerosis, in which patches of degeneration are found, irregular in shape and situation, and affecting any part of the nervous centres or even the peripheral nerves. They exhibit the microscopic characters of interstitial overgrowth with nerve disintegration. Another example of diffuse degeneration is, a widely spread induration and atrophy which occurs principally in the cortex cerebri in young children, who are the subjects of hereditary syphilis. In such examples it is difficult to say how much may be due to chronic inflammation, and how much to primary nervous degeneration.

So far we have chiefly spoken of degeneration of the white matter, wherein it can best be studied. Degeneration may affect the nerve-cells also. Thus, in the "spinal" forms of progressive muscular atrophy there is found a degeneration of the anterior cornual cells. This is often associated with similar disease in the white matter of the pyramidal tracts. Acute disease of the same cells occurs in infantile spinal paralysis, but this is most probably inflammatory.

The causes of nervous degeneration, other than secondary degeneration, are very obscure. Probably

they must be sought among some such influences as these—antecedent constitutional diseases, such as syphilis, and perhaps other specific diseases; toxic influences—*e.g.*, lead, alcohol, ergotism, &c.; inherited or congenital vulnerability of the nervous structures which are affected; over-use, or actual injury.

Disease originating outside the nervous parenchyma — (*a*) **Vascular disease.** — Many diseases of the nervous system depend, as already said, on causes extrinsic to the nervous substance. A large proportion of these arise in the vessels.

Cerebral hæmorrhage.—Hæmorrhage is common in the cerebral hemispheres. It usually originates from a point just outside the lenticular nucleus—*i.e.*, from one of the external divisions of the central branches of the middle cerebral artery (*vide* p. 60). The size of such a hæmorrhage may vary greatly. As it increases it presses upon the basic ganglia and internal capsule from without inwards, ploughs up the tissues composing them, and finally may rupture into the lateral ventricle, flooding with blood this ventricle, and perhaps also the third and opposite lateral ventricle, and even penetrating through the Sylvian aqueduct to the fourth ventricle. A large hæmorrhage of this description may find its way out at the descending cornu of the lateral ventricle, or break through the floor of the third ventricle, and so fill with blood the sub-arachnoid space at the base of the brain. Such a large hæmorrhage is necessarily fatal; smaller hæmorrhages in course of time become encysted and gradually dry up, and may eventually leave only a yellowish scar. Hæmorrhages originate either from rupture of an atheromatous artery, a condition in association with which we commonly find granular kidneys and cardiac hypertrophy, or from small aneurysms, or from vessels which have undergone fatty degeneration. Aneurysms are found not only in the central arteries, in which position they are usually small and multiple, and are known as miliary

aneurysm, but also upon the arteries of the circle of Willis or of the meninges, where they are more frequently of larger size and single. Again, cerebral hæmorrhage may occur in a vascular new growth, and such a growth should always be looked for when the hæmorrhage is in an unusual situation, as, for instance, the sub-cortical white matter. Hæmorrhage in the pons is not uncommon; in the spinal cord, apart from injury or new growth, it is rare.

Plugging of cerebral vessels.—Plugging of the cerebral arteries may be due either to embolism or thrombosis. In either case, if there be complete obstruction, and if the artery affected, as is the case with the central arteries, have no anastomoses, the result is softening of the district supplied by it, a necrosis and disintegration of the nerve-tissue with corresponding abolition of function. **From embolism.**—In embolism this plugging is sudden, and is due to the impaction by the blood-stream of some foreign body, most frequently a fragment of blood-clot or a cardiac vegetation. Thus cerebral embolism is often a sequela of valvular disease. Sudden cerebral symptoms in the course of such valvular disease may, however, be due to hæmorrhage, for a previous embolism may have caused weakening of the vascular wall, resulting in aneurysm. The middle cerebral artery is usually the seat of embolism, and that of the left side, it is said, more frequently than the right.

From thrombosis. — Thrombosis of an artery follows as a secondary process upon embolism, sealing and completing the obstruction; or originates from disease of the arterial wall, such as the roughening of atheroma, or the partial occlusion which results from syphilitic deposit. The process is gradual, though the actual nervous symptoms may be sudden, and it may have a tendency to spread. Thrombosis of the veins and sinuses results in deep punctate congestion, with softening of the district whence the veins are derived. It may occur in the proximity of

diseased bone, or in such constitutional states as the cancerous cachexia, gout, anæmia, the puerperal condition.

Vascular disease as a source of spinal softening or spinal sclerosis.—In the spinal cord softening is common, just as hæmorrhage is rare. It may be impossible to say whether it is the result of previous inflammation or of deficient blood-supply. Softening of the lower parts of the cord has been explained, on the latter supposition, by the lengthy course taken by the vessels of this district along the elongated nerve-roots. Again, a form of spinal sclerosis has been described by some authors in connection with vascular disease. It is said to spread from diseased arteries as a focus, and thus to be distributed longitudinally in the cord, so as to simulate in a measure the "systematic" scleroses which we have described above.

(β) **Disease of nerve envelopes**—(1) **Bones.**—Disease of their envelopes—viz., the bones and meninges—seriously affects the nerve-centres themselves. Caries of the vertebræ, usually associated with angular curvature (Pott's disease), sets up an inflammatory thickening of the membranes beneath, and this in its turn injures the cord itself, partly by compression, partly by spread of inflammation to its substance. This has been called compression myelitis. Disease of the cranial bones—necrosis, caries or syphilitic—may cause thrombosis of the adjacent sinuses, meningitis, or an intra-cranial abscess. Chronic purulent catarrh of the middle ear, with disease of the petrous bone, often has one of these results.

(2) **Meninges.**—Primary affections of the dura mater are not very common. Hæmatoma of the dura mater occurs chiefly in the cerebral dura, and presents the appearance of a flat cake upon the inner surface of the membrane, having the general appearance of blood-clot. Whether it is simply blood-clot more or

less encapsuled and organised, or whether it is the result of an inflammatory exudation into the meshes of which blood has been extravasated, is still uncertain. In the spinal cord, particularly in the cervical region, the dura mater is liable to a form of chronic inflammation which causes much thickening of its inner surface; this has been called hypertrophic pachymeningitis. Chronic affections of the cerebral dura are most common among the inebriate, the syphilitic, and the insane.

Affections of pia and arachnoid.—Disease of the inner membranes, pia and arachnoid, is more important, both because of their greater proximity to the nerve-substance, and because the pia carries the nutrient vessels. In the brains of alcoholic subjects a diffuse milky thickening of the arachnoid is found, with excess of sub-arachnoid fluid, and shrunken hard convolutions beneath. In children a chronic or subacute meningitis may arise limited to the parts around the occipital foramen. Syphilitic meningitis is subacute. It occurs in the brain in the form of patches, over which the membrane is thickened and infiltrated with thick yellowish lymph; the inflammation may spread to the cortical substance beneath. A similar process may occur in the cord; here it may extend over a very considerable length of membrane, affecting also the superficial layers of the cord (meningo-myelitis), and the nerve-roots as they pass outwards. **Acute meningitis** is often secondary to disease of the adjacent structures, the scalp, bones, dura mater, or sinuses. A very frequent cause is ear-disease, with disease of the petrous bone. Disease of the nasal cavities and of the ethmoid bone may have a similar result. Acute specific diseases form another cause for meningitis. Under this head come pyæmia, cerebro-spinal meningitis, tuberculosis, and perhaps other acute diseases. Pyæmic meningitis is suppurative, and may be spread over the whole cerebral surface. **Tubercular meningitis** has limitations as to its seat, and may show

other characteristic appearances. Thus, as a rule, it is limited to, or at least most marked in, the following places: the base of the brain, the fissures of Sylvius, the upper surface of the cerebellum. Tubercular meningitis of the convexity only is decidedly uncommon. There is more thickening and less purulent effusion than in the other acute forms of meningitis; and the effusion consists of scanty sticky lymph held in the interstices of the membrane rather than of actual pus. Grey tubercles can often be seen studded along the vessels of the meninges, particularly in the Sylvian fissures. These, however, may be so minute as scarcely to be recognised by the naked eye; in that case the diagnosis must be made by the microscopic examination of the membranes, the localisation of the meningitis, and the existence of tubercle, or caseous matter in other organs. For tubercular meningitis is usually part of a general tuberculosis. The brain substance in the neighbourhood of the meningitis is frequently reddened and softened; the choroid plexus may share in the inflammation of the meninges; where there is a large effusion of fluid into the ventricles, these become greatly distended, and on the surface of the hemispheres the sulci become obliterated, the convolutions flattened, the cortex bloodless from pressure of the effusion, while the brain-substance in the neighbourhood of the ventricles becomes quite soft and macerated. In some instances, perhaps more frequently than is usually thought, tubercular meningitis affects the cord as well as the brain. In the acute specific disease known as cerebro-spinal meningitis this is the rule.

Lastly, it must be remembered, for it is important clinically, that disease of the bones and meninges affects not merely the nerve-centres but also the nerve-trunks and nerve-roots of the neighbourhood. Thus syphilitic disease at the base of the skull causes varied paralyses of the cranial nerves; caries of the petrous bone causes facial paralysis; disease of the spinal column

and meninges causes symptoms of pain and paralysis referable to the adjacent spinal nerve-roots.

(γ) **Tumours.**—Tumours are a frequent source of nervous disease. Under this head we may enumerate—

I. Such foreign bodies as hydatids, which may be found in the brain or, still more rarely, in the spinal cord; simple cysts, of unknown origin, which appear to be not uncommon in the cerebellum.

II. Inflammatory new growths, which form the local manifestation of a specific disease. Under this head come the nodular thickenings or fusiform swellings of the nerve-trunks found in leprosy. Syphilis also may produce not only meningeal and arterial disease, as indicated above, but also an infiltration or thickening of nerve-trunks, or, still more commonly, distinct gummatous tumours. These latter may occur either in the brain or spinal cord; they are said usually to be so placed as to manifest some connection with the pia mater or the choroid plexus. Hence, the cortex cerebri is a not unusual site for them. Since the *rôle* of syphilis in the production of nervous disease has been recognised, and anti-syphilitic remedies used almost as a matter of routine, gummata have been less frequently seen post-mortem. Tubercular disease may present itself in the form of caseous masses, deeply situated in the nervous centres. These are perhaps most frequent in the cerebellum and its neighbourhood. They may be single or multiple, may run to a considerable size, and may exist for a length of time.

III. Tumours in the stricter sense—*i.e.*, new growths of definite microscopic type, and with tendency to increase. These are mainly carcinomata and sarcomata. Carcinomata are perhaps the least common of the two, and when found in the nerve-substance are secondary to growths elsewhere. But they may also spring from the dura mater or the bones. Of sarcomata, a common sort is that known as glioma, a small

round-celled growth which originates in the neuroglia. It may affect any part of the nervous centres. In glioma of the pons Varolii the whole of this organ may be enlarged, unequally perhaps, and with lobulation and some distortion of its surface, but still preserving its general shape. On section, the whole pons is found to be diffusely infiltrated with new growth. Other forms of sarcomata—large-celled, spindle-celled, or with cells of mixed type, myxo-sarcomata, cystic sarcomata—are also found. They may be primary or secondary; single, multiple, or even symmetrical; and may affect almost any part of the nervous system or its envelopes. Among the more curious sites may be mentioned the nerve-trunks at the base of the brain; a spinal nerve-root; the spinal meninges, enwrapping the cord for a considerable distance; the dura mater in several of the cerebral fossæ simultaneously. The nerve-substance of the cerebral hemispheres is perhaps their commonest seat. And here it may be remarked (1) that a new growth may be almost indistinguishable by the naked eye from softish cerebral tissue; (2) that cerebral new growths are often very vascular, and their vessels may suddenly give way, and hence both the clinical symptoms and the post-mortem appearances may simulate to a certain extent those of simple cerebral hæmorrhage.

CHAPTER III.

ON CERTAIN GENERAL SYMPTOMS AND METHODS OF INVESTIGATION.

Before describing the several varieties of nervous disease, it may be well to consider certain symptoms which are common to many of them. These symptoms may be roughly grouped into anomalies of the motor or sensory functions, such anomalies being either on the side of defect (paralysis), excess (irritation), or perversion; anomalies of reflex or of co-ordinative movements or of the intellectual functions.

Motor paralysis in its relations to lesions at different sites.—Loss of the power of voluntary movement (motor paralysis), and in a less degree loss of the power of sensation (sensory paralysis), supplies us with the most definite symptoms for purposes of diagnosis, and particularly diagnosis of the locality of the disease. A recapitulation of the course of the motor and sensory fibres will aid us in understanding the forms of motor and sensory paralysis.

Course of the motor tract.—The motor fibres commence in the ganglion cells of the cerebral cortex. Here (it will be remembered) the nervous mechanism for each half of the body is separated in its respective hemisphere, and, further, the centres for the various parts of the same side—viz., face, arm, leg, &c.—are spread over a tolerably wide area. But as they descend through the corona radiata the fibres from these centres gradually approximate, till they reach the

posterior limb of the internal capsule. Here they lie in close juxtaposition, though they are not intermingled. Proceeding downwards through the crus cerebri they reach the pons, where they still lie within a small area, though broken up and separated by the transverse fibres which cross between the cerebellar hemispheres. At the level of the pons some of them—viz., those for the eyes, face, and tongue—cross, decussating with those of the opposite side, and end in the nuclei of their respective cranial nerves. The remainder continue their course till they reach the junction of the medulla and cord. Here they also cross to the opposite side (decussation of the pyramids). Thence they pass down in the opposite half of the cord, mainly within the posterior part of its lateral column—*i.e.*, as the crossed pyramidal tract—(some running also without decussation in its anterior column as the direct pyramidal tract) till they terminate in the large cells of the anterior cornu, and principally at the levels where these cells are most abundant—viz., at the cervical and lumbar enlargements. Thus, from the cortex cerebri to the motor nuclei of the cord and medulla the fibres are uninterrupted by ganglion cells. From these spinal nuclei motor fibres start afresh and pass outwards from the central nervous axis, either as cranial motor nerves or anterior spinal nerve-roots. These latter soon join the sensory roots, fuse with them into a nerve-trunk of mixed sensory and motor function, and thus run till they approach their distribution, where filaments are again separated out for the supply of the various muscles and cutaneous areas.

Course of the sensory tract.—Concerning the course of the sensory fibres within the central nervous axis much less is known, but this much may at least be said: those which come from the one side of the body enter the cord by the corresponding posterior nerve-roots, and soon after entering it cross to the

opposite side of the cord.* Their exact position as they run upwards in the cord we do not know. But at the level of the pons and the crura cerebri the sensory fibres probably lie in the tegmentum (viz., the posterior or dorsal part of these regions), just as the motor fibres lie in the crusta (viz., the anterior or ventral part). Higher still, at the level of the internal capsule, the sensory fibres, including now those from the organs of special sense, are gathered into a bundle which lies behind the bundle of motor fibres, and constitutes the posterior third of the posterior limb of the internal capsule. Thence they probably diverge, spreading through the corona radiata to reach the sensory areas of the cerebral cortex.

From this arrangement of the motor and sensory tracts there follow some important facts regarding the distribution of paralysis.

Paraplegia.—When the spinal cord is affected by a transverse lesion, say in the dorsal region, sensation and motion are lost in all the parts below. This is called "paraplegia." The bilateral distribution of the symptoms is almost characteristic of spinal disease. The paraplegia may be complete or partial in degree, both as regards motion and sensation; or motion only may be affected, since the two motor tracts lie not far apart, and are for some unexplained reason more easily affected than the sensory.

Hemiparaplegia.—If the lesion does not reach right across the cord, but across one (lateral) half of it only, then motor paralysis results (of the parts below) upon the side of the lesion, because the motor tract for one-half the body (say the right) runs in the corresponding (right) half of the cord; but there is sensory paralysis of the opposite side, because the sensory fibres of one-half the body (say the left) cross after entering the cord, and run in its opposite (right)

* Even this statement, in the light of some recent researches, may need revision.

FIG. 34.

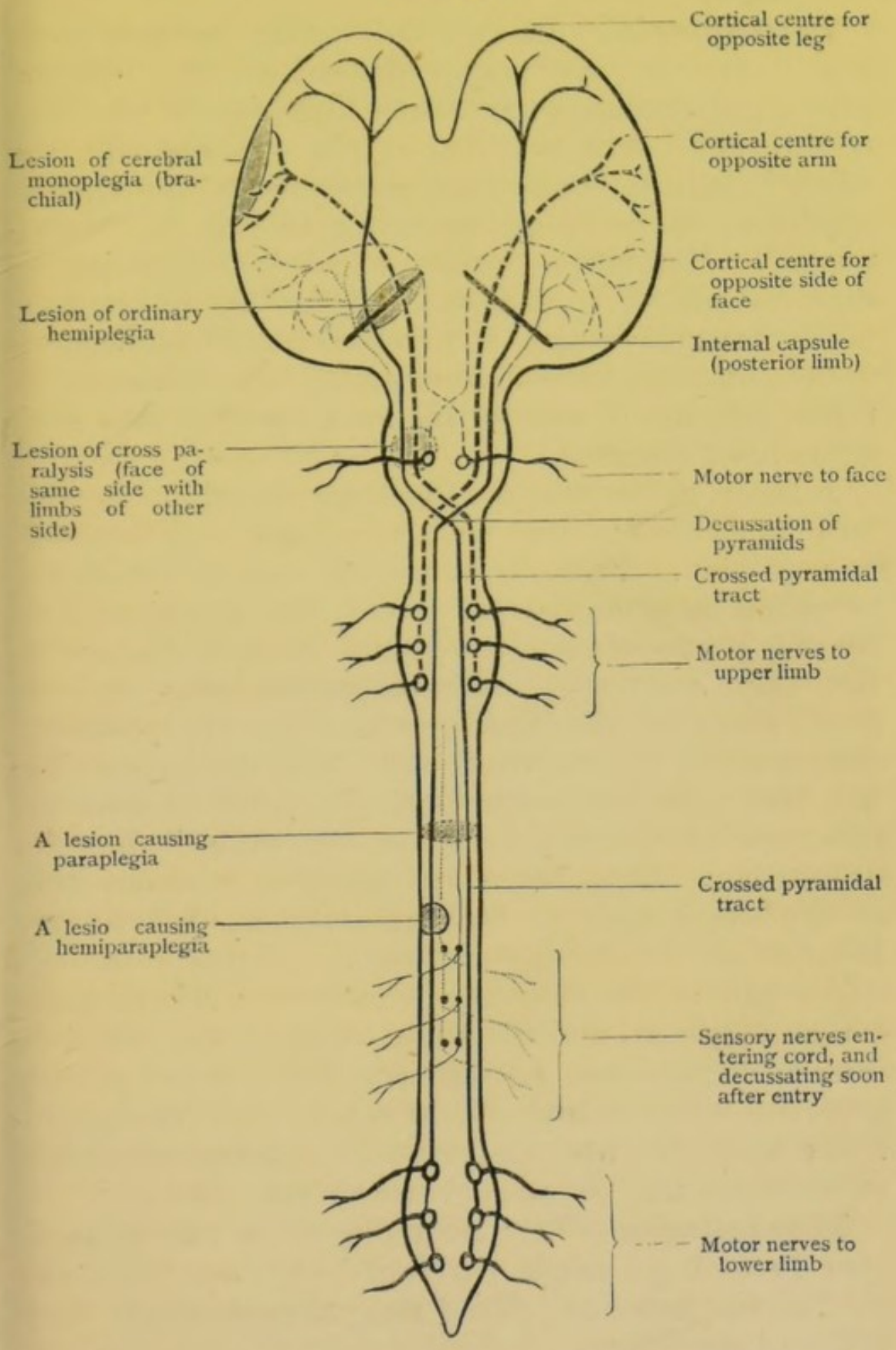


Diagram to show the general arrangement of the motor tract, and the effect of lesions at various points.

half. This condition, a rare one, is called "hemi-paraplegia."

Hemiplegia.—Passing now to the brain, if there is a lesion in the neighbourhood of one internal capsule (a common event), then the motor fibres going to the face, arm, and leg of the opposite side are likely to be interrupted, and there ensues the familiar condition of one-sided paralysis known as "hemiplegia." Hemiplegia points to a cerebral lesion as does paraplegia to a spinal. According to the part of the capsule which is involved, the hemiplegia is motor, sensory, or both combined. The abolition of motor function is usually the most striking and most permanent symptom.

A lesion in the pons may be large enough to involve both sides of it; then both arms and both legs will be paralysed. Probably there will also be symptoms referable to some cranial nerve; the commonest of these is contraction of the pupils (from irritation of the third nucleus?). **Cross-paralysis.**—A one-sided lesion of the pons produces the characteristic symptoms of "cross-paralysis." This depends on the fact that a cranial nerve is caught after its decussation, and the motor tract of the limbs before its decussation. Thus the nerve paralysis is on the side of the lesion, and the limb paralysis, as in ordinary hemiplegia, on the opposite side. In disease of the upper part of the pons the facial nerve of the same side is paralysed and the limbs of the other side; in disease of the crus cerebri, the third nerve of the same side with the limbs of the other. The hemiplegia, if the lesion lies near the posterior (tegmental) aspect of the pons, may be sensory rather than motor.

Monoplegia.—By "monoplegia" is meant paralysis involving a single part only—*e.g.*, one limb, one side of the face, &c. This may happen under very different conditions.

A. The lesion may be cerebral, affecting either (1) the cortex or sub-cortical white matter of the

motor area before the motor strands have joined in the internal capsule; or (2) the capsule, but to such a limited extent as to pick out one only of its constituent strands, the face, arm, or leg-fibres.

B. The lesion may be spinal. If it be limited to a moderate length of the anterior grey cornua of one side, paralysis of one limb or one set of muscles in a limb will result. Unlikely as such a limitation might appear *à priori*, it often occurs practically, as in cases of infantile paralysis.

C. The lesion may be neural, affecting either a nerve-trunk or the nerve-endings. Let us defer for simplicity's sake the consideration of disease of nerve-endings (peripheral neuritis), which is indeed more often bilateral than monoplegic, and let us consider the points of diagnosis between the other forms of monoplegia.

Cerebral monoplegia (A) differs from that of spinal (B) and neural (C) origin in these points mainly: in the two latter forms there are disturbances of nutrition in the paralysed muscles, evidenced by wasting, flaccidity, and alteration of electrical reactions, owing to interference with the function of the anterior cornual cells; whereas in the cerebral form the electrical reactions are normal, wasting, if any, is less marked and less early; and there is a tendency to rigidity. In the spinal and neural forms, the paralysis is absolute; that is to say, volitional, reflex, and associated movements are all abolished, whereas in cerebral paralysis the leg may move when tickled, or the arm when the patient yawns. Tendon reactions, too, are abolished in the spinal and neural forms, preserved or exaggerated in the cerebral. (There may be also differences in the distribution of the paralysis within the limb.)

Spinal monoplegia (B) due to disease of the anterior cornual cells differs from neural (C), due to disease of a nerve-trunk, in the following respects. In the former there is no anæsthesia; in the latter, except

in disease of a purely motor nerve-trunk such as the facial, we should expect anæsthesia. In the former, again, the distribution of the paralysis should follow the grouping of the spinal segments; in the latter it should follow the anatomical distribution of the nerve. There will remain, however, for both forms the common characteristic of muscular wasting or flaccidity, plus electrical changes, except in the few instances where the disease is either very mild or very recent.

Cerebral monoplegia (A) may arise from a lesion either (1) in or near the cortex, or (2) in the internal capsule. The former is more common, because at the cortex the motor tract is spread over a wider area and its constituent centres more separate than in the region of the internal capsule. Small capsular lesions may, it is true, pick out one of the several bundles for face, arm, leg, &c. But since such a lesion is usually a hæmorrhage, the immediate shock of which extends beyond its anatomical limits, we should expect a history of hemiplegia, which in time has narrowed down to monoplegia. Cortical lesions, on the other hand, which are frequently progressive (tumours, gummata, meningitis, and the like) begin with monoplegia, which may spread into a hemiplegia.

Examination of motor paralysis. — Motor paralysis, if of any degree, is a symptom sufficiently obvious both to the physician and the patient. It may be necessary, however, to distinguish it from weakness consequent on mere exhaustion, such as occurs after acute diseases, from inability to move caused by pain on movement, or by joint disease, or (in children) by separation of an epiphysis. A hemiplegia, when the patient is in a state of coma, may sometimes be made out by the flaccid way in which the limbs of the paralysed side drop when we raise them, and then allow them to fall; by the distortion of the face towards the non-paralysed side, or by the puffing out of the cheek upon the sound side.

Paralysis of a minor degree is often called "paresis." The effect of such minor paralysis upon the finer movements of the hands may be confused with the awkwardness produced by loss of sensory or of co-ordinative power. Incomplete loss of power in the leg is shown by the patient dragging the limb as he walks; the toes of the boot may sometimes be noticed to be worn down thus. An instrument called a dynamometer is sometimes used for estimating the amount of power retained by the hands (strictly speaking, by the flexors of the fingers and thumb). It is an oval spring, which the patient is made to squeeze; the resistance which he thus overcomes is recorded by a dial and index fixed in the centre of the spring. It is useful for estimating the relative strength of the two hands: but since there is a certain knack in squeezing it far, it is less trustworthy for absolute measurements.

Loss of power in individual muscles is detected in some of the following ways. Knowing the normal action of the muscle, we tell the patient either to fix the limb in such a position or bring it into such a position as involves the contraction of the muscle, while we ourselves apply a counter-force. Thus, for the biceps cubiti we bid him keep the elbow semiflexed while we try to straighten it out; for the ilio-psoas we bid him while sitting to bring the thigh towards the body while we apply pressure downwards on the knee. Or we may guess the nature of the paralysis from the position produced by the unbalanced action of antagonist muscles, witness the claw-hand in paralysis of the interossei, or from the expedients the patient adopts in performing certain movements, witness the way in which a child with weakness of the erector spinæ climbs up his knees in the endeavour to get up from the ground.

Examination of sensory paralysis.—Sensory paralysis, commonly called anæsthesia, is more complicated in its nature and more difficult to test than

motor. We must distinguish various modes of sensation.

(1) Tactile sense—*i.e.*, the feeling experienced when the skin is touched. This may be tested by light touches with the feather of a quill-pen, or by touching the hairs of the part examined. The eyes should be securely closed, the surface tested should not be cold, the fingers, &c., should not be moved. Lastly, allowance must be made for the patient getting puzzled, and for impatience on his part (or on the part of the operator). In certain cases the patient may feel the touch but cannot say where it is; or localises it in the wrong place. In rare cases the patient will refer the touch to the corresponding part of the opposite side of the body; this has been called "allochiria." It is frequent to hear complaints of numbness, deadness, and the like, pointing no doubt to minor degrees of anæsthesia, and yet to be unable to detect anything by examination.

(2) Sense of temperature. This may be tested with hot and cold spoons, or sponges; or by test-tubes filled with water of different temperatures; or by alternately blowing and breathing on the part. The patient may be unable to discriminate between the sensations, or may take the one sensation for the other. Sometimes he will start away from the application of a cold surface and declare it burnt him.

(3) Sense of pain. This may be tested by pinching, pricking with a needle or pen-point, or by the faradic current applied with a wire-brush. The touch of the object may be felt sometimes, but not the pain (analgesia); or, again, the pain may not be felt till after a distinct interval owing to delay in transmission of the pain-sense.

(4) Muscular sense. This expression apparently means the appreciation we form of muscular tension, whether such tension be active, as when we contract our muscles against resistance; or passive, as when the

limbs are placed in varying positions. The muscular sense may be tested in the following ways :—

(*a*) By noting the patient's power to discriminate differences in weight. This may be done by making him hold in his hand balls of similar external appearance, but differing in weight.

(*β*) By making him shut his eyes, and tell when his limbs are put by us into different postures. In this latter test, at any rate, the impressions derived from the muscles are supplemented by those from the joints, fasciæ, tendons, &c.

"Muscular sensibility" is sometimes distinguished from "muscular sense," as meaning the conveyance from the muscles of ordinary tactile impressions when they are handled, or of painful impressions when they are pinched, cut, or severely faradised.

The various modes of sense that we have enumerated may be affected separately, or together. It seems probable that different impressions may be conveyed by different routes within the cord, but we do not certainly know the route of any one of them.

Distribution of anæsthesia.—From the distribution of anæsthesia, just as from the distribution of motor paralysis, certain inferences may be drawn as to what part of the nervous system is involved. In disease of the nerve-endings (multiple peripheral neuritis) the anæsthesia involves the extremities of the limbs, like a glove or stocking, irrespective of the distribution of the main nerve-trunks : in disease of a nerve-trunk the sensory like the motor palsy follows the distribution of the particular nerve-trunk. In disease of the nerve-roots, or of the cord itself, the value of the sensory symptoms depends a good deal on the level of the lesion in the cord. Thus, in a transverse dorsal myelitis, owing to the simplicity of the cutaneous distribution of the dorsal nerves, the level reached by the anæsthesia enables us to infer with considerable accuracy the segment of the cord which is affected. But in a myelitis affecting the cervical enlargement,

or the lumbar or sacral region, the distribution of the anæsthesia in the limbs is less easily understood. We shall refer the reader to the statements already given as to the functions of these parts. The import of sensory and motor palsy, when they involve opposite sides of the body, spinal hemiparaplegia, has been sufficiently pointed out. Anæsthesia depending upon lesion of the sensory (extreme posterior) part of the internal capsule involves the whole of the opposite side of the body, face, trunk, limbs, and special senses. Anæsthesia from disease of the cortex is a subject incompletely known; but it would appear that the loss of sensation from this cause affects the limbs, segment by segment, involving the hand up to the wrist, the forearm to the elbow, the arm up to the shoulder, &c., so that oddly enough the distribution of it may resemble that of the anæsthesia from disease of the nerve-endings.

There is this much, however, to be said about sensory as compared with motor paralysis, that (except in cases when a sensory tract rather than a motor is definitely singled out by disease) sensory paralysis is the less marked and the more transitory symptom of the two, whether the lesion lie in the brain, cord, or nerves.

Other anomalies of motor function.—The functions of motion and sensation instead of manifesting deficiency (paralysis), may be morbidly exalted or perverted.

Convulsions. — Of such motor exaggerations there are several important kinds. The convulsions of true epilepsy consist of sudden widespread purposeless muscular spasms, usually tonic at first (*i.e.*, the muscular contraction is continuous), and then clonic (*i.e.*, the affected muscles, relaxing and contracting alternately, produce a vibratile movement of the affected parts). In the convulsions called epileptiform, the movements begin in a definite member and involve the rest of the body gradually. These latter attacks are of special interest, since it has been shown

that they are caused by local disease in the motor area of the cerebral cortex; that the principal focus of disease is in the cortical centre for the member first attacked by spasm; and that the spread of the convulsions corresponds to the spread of irritation over successively adjacent motor centres. Thus, suppose a convulsion begins in the great toe, it is likely to spread thence up the leg and thigh, to the upper limb and face, because the irritation will involve successively the leg, arm, and face centres.

Chorea.—The movements of chorea consist of restless sudden twitches, which vary from mere starts to wild jactitation of the limbs. They are not paroxysmal, like epileptic convulsions, and the movements themselves are more varied and intermittent. They are not rhythmical, like the tremors we shall presently notice; they occur both while the patient is at rest, and as an interruption to his voluntary movements. From the facts that the twitchings and grimacings of chorea resemble sudden voluntary movements, and that they may be limited to one side of the body, we may surmise that their source is the cerebral hemispheres.

Athetosis.—In the late stages of hemiplegia, particularly when this is incomplete in degree and has originated in childhood, certain slow continuous movements of the affected limbs may be observed, chiefly hyper-extension and wavy movements of the fingers, with perhaps drawing movements of the arm. Such a condition, aptly described by Gowers as a "mobile spasm," is called athetosis.

Tremors.—Several varieties of movement are classed under the term "tremors," meaning by this a more or less rhythmical shaking. The first consists of such as are seen in disseminated sclerosis. Here the limbs are still when at rest, but on attempting to use them irregular movements set in. In a well-marked case these movements are wide and irregular in extent, and they involve the whole hand or arm.

To these it is usual to oppose a second class—the tremors of paralysis agitans. The movements in this disease, at least at an early stage, are most noticeable when the limb is at rest and are checked by voluntary movement. They are smaller in extent (finer tremors, that is), and affect primarily the small muscles—viz., those of the thumb and fingers—and are more truly rhythmical both in time and in extent. There are many other diseases in which tremors occur, and in which the characteristics to which we have alluded should be noted; that is, whether they are most marked during rest or during effort; whether they are fine or coarse movements, rapid or slow, rhythmical or irregular. Such diseases are senility, general paralysis, hysteria, Graves' disease, mercurialism, alcoholism.

Fibrillary twitchings.—Another variety of muscular over-action may be seen in cases of progressive disease of the anterior cornual cells of the cord. Small bundles of the muscles which are undergoing atrophy twitch from time to time, so as to raise the skin above them, and sometimes even so much as to cause a start of their tendon. This is called fibrillary tremor, or (where larger parts are involved) fascicular tremor of the muscle.

Spasm, either continuous or intermittent, may sometimes be distinctly limited to the distribution of the nerves, as in the wryneck produced by contraction of the sterno-mastoid and trapezius, or in motor "tic" affecting the facial muscles. Or, again, individual muscles, or sets of muscles, may be affected.

Another very common and very important form of muscular over-action is the rigidity which accompanies certain chronic stages of paralysis. This we shall defer till we speak of tendon reactions.

Anomalies of sensory function—Pain.—Exaggerations and perversions of the sensory functions furnish less definite indications of disease than do those of the motor. The commonest, pain, is obviously of too wide import to be a direct index of nervous

disease. But there may be such characters about a pain as indicate that the nervous system is specially involved. It may be limited to the district supplied by a particular nerve, as in neuralgia, or it may have a peculiar lancinating character, as the "lightning pains" of tabes dorsalis, or it may be remarkably paroxysmal. The name "causalgia" has been given to a peculiar burning pain, which occurs in partially anæsthetic limbs, particularly where there has been injury or disease of a nerve-trunk. **Tenderness** to touch may also be limited to a nerve-trunk, or to certain points in its distribution. Thus, in trigeminal neuralgia tender points may be found where the nerve makes its exit from the bones upon the face; and in nervous women tender points are found over the intercostal nerves, under the mammæ, in a line with the angle of the scapula, over the vertebræ, &c. Particularly well marked, in this latter class of case, is the tenderness to pressure in the inguinal regions, which has been called "ovarian" tenderness. When tenderness is so marked that merely touching the skin gives rise to pain, there is said to be "hyperæsthesia," more properly it would be called "hyperalgesia." **Paræsthesia.**—Sensations described by patients as crawling ("formication"), pins and needles, numbness, deadness, and the like, are not infrequent, and may indicate, as above noted, a mild degree of sensory paralysis. **Sensory auræ.**—Lastly, epileptiform attacks are sometimes heralded by sensations instead of by limited convulsions. These sensory "warnings" are presumed to point to a focus of disease in the sensory area of the cerebral cortex, and to be thus strictly comparable to the motor warnings which we have noticed above.

Reflex actions form a very important item in the diagnosis of nervous disease. These we may roughly divide into three classes: the organic reflexes, the superficial or skin reflexes, and the so-called deep or tendon reflexes.

The "reflex arc."—The nervous structures concerned in a reflex action consist of three main parts: (α) afferent fibres which convey the stimulus from the periphery to (β) the centre, consisting of the grey matter of the cord, medulla, pons, or cerebrum. In this centre the stimulus is modified and rearranged so that it issues as a motor impulse along (γ) the efferent fibres, which convey it to the muscle or other organ which has to be called into activity. If a reflex action is present, we may infer that all these parts (together called the "reflex arc") are intact. If it is absent, this may be due either to disease of the arc itself, or to some perturbing influence exercised by some higher part of the nervous centres, or to the fact (not unfrequent in skin-reflexes) that the reflex is naturally ill-developed, or has become obsolete from disuse. The several fragments of the cord with their attached nerves constitute a series of reflex arcs; and each of the reflex centres—*i.e.*, each segment of the cord—has a recipient (posterior) part and a motor (anterior) corresponding to the anterior and posterior nerve-roots.* Disease of any part of the arc, central or peripheral, may annul the reflex; disease of the anterior part of the spinal segment, or disease of its efferent nerves, causes motor palsy as well as loss of reflex; disease of the afferent nerves causes loss of reflex and loss of sensation; but disease of the posterior part of the spinal segment may cause loss of reflex without loss of sensation, the probable reason being that within the cord the afferent fibres take a different course according as they are going to the reflex centre or to the sensorium.

The control exercised by the higher centres over reflex actions is a subject of which our knowledge is imperfect; but that such control exists is shown by the facts that reflex actions can often be prevented by the will, and that they may be exag-

* *Cf.* p. 9.

gerated when cerebral influence has been cut off by disease.

Organic reflexes.—Let us consider those reflexes which have a special bearing on questions of diagnosis, and, first, some of the so-called "organic reflexes." The centres for micturition and defæcation are situated in the lowest parts of the cord.*

Micturition.—The act of micturition is complicated. So far as it is purely reflex, its mechanism is this: the urine, gradually accumulating in the bladder, supplies, after sufficient accumulation, a stimulus to the spinal centre; the activity of the centre thus aroused causes a double action in the bladder—viz., contraction of the detrusor urinæ and relaxation of the sphincter, and in this way the bladder is emptied. But, further, the spinal centre is in communication with the brain; hence the patient is conscious of the stimulus which leads to micturition, and can, on the other hand, prevent the act of micturition if he chooses.

Were this prevention or inhibition the only result of the influence exercised by the cerebrum, we should expect that removal of this influence would simply leave the spinal centre free to empty the bladder, unconsciously and involuntarily, at stated intervals. But as a matter of fact in cerebral hæmorrhages, crushing of the cord above the lumbar region, and other injuries and disease which remove the cerebral influence, we usually find the bladder distended. Hence it seems that the cerebrum acts not only in the way of inhibiting, but also of stimulating, the micturition centre. If the spinal centre for micturition be itself destroyed, distension of the bladder results with still greater certainty; for though the sphincter is paralysed as well as the detrusor, there remains in the urethra and parts around it sufficient resistance to the exit of urine to cause distension. The bladder rises

* Probably in the 3rd and 4th sacral segments.

above the pubes as an oval, dull, fluctuating swelling; urine may dribble from the urethra, but this is simply a sort of overflow, which does not empty the organ.

Defæcation.—The mechanism of defæcation is very similar. In health the tonic contraction of the sphincter prevents the continuous passage of fæces. During defæcation the sphincter is relaxed, and the peristaltic action of the bowel, reinforced by the contraction of the abdominal muscles, expels the fæces. Defæcation, like micturition, is not in health a purely reflex act: the spinal centre is obviously under the control of the cerebrum, which can inhibit or permit its action. We may notice, however, that some of the muscles which contribute to the expulsion of fæces, notably the diaphragm and abdominal muscles, are innervated from a higher level than the sacral cord, in this respect differing from the muscles of the bladder. And again, that when the rectal sphincter is paralysed by destruction of the sacral centre, there is not (as in the case of the urethra) sufficient resistance in the soft parts to produce retention. Hence, in such paralysis involuntary evacuation of fæces is the rule.

There exist other lumbar centres whose condition may afford us information as to the state of the cord. Thus, by destruction of the lumbar cord the act of erection is rendered impossible, while in transverse lesions of the cord higher up we often find troublesome priapism owing to over-action of the centre for erection.

It need scarcely be added that the organic reflexes, which are most essential to life, have their centres in the medulla (cardiac, respiratory, gastric reflexes). Affections of these, as evidenced (for instance) by continuous vomiting, inability to swallow, disturbances of the respiratory or cardiac rhythm, are most important in prognosis; they show that disease is advancing towards a vital point of the nervous system.

Superficial or skin reflexes.—"Skin" reflexes, or superficial reflexes, are the movements which follow when certain portions of the skin are tickled or irritated. The principal skin reflexes are as follows:—

The plantar—tickling the sole causes movement of the toes or drawing up of the leg; the centre is in the upper sacral region. The glutæal—irritation of the skin over the glutæi causes contraction of those muscles; centre, in lower lumbar region. The cremasteric—stroking the skin of the inner part of the thigh produces retraction of the testicle on the same side; centre, upper lumbar region. The abdominal—stroking one side of the abdomen causes contraction of the abdominal muscles upon that side; centre, lower half of dorsal region; and stroking the skin over the lower ribs causes a twitch of the epigastric region; this is called the epigastric reflex, centre, about mid-dorsal region. The scapular—upon irritating the skin between the scapulæ there follows contraction of the muscles beneath the skin; centre, in cervical enlargement. The conjunctival—touching the conjunctiva produces winking; centre, nuclei of fifth and seventh nerves with their fibres of connection.

The pupillary reflexes will be described in connection with the eye—*vide* p. 122.

Absence of these skin reflexes is not necessarily due to disease. They are, indeed, usually present in healthy children, but in adults they cannot always be obtained. Some of them—*e.g.*, the plantar, cremasteric, abdominal, conjunctival—are more constant than others. In cases of unilateral cerebral disease, especially at an early stage, it may be found that the superficial reflexes on the paralysed side are absent, or diminished as compared with those on the sound side. In hysterical paraplegia, the plantar reflex may be, so to speak, delayed; that is, we have to tickle the sole a long time before the reflex action follows.

Tendon-reactions, otherwise called tendon

phenomena, tendon reflexes, deep reflexes, or "jerks," are more constant phenomena in health, and are of extreme importance in the diagnosis of disease. Let us first consider the most typical example of this class—viz., that which is variously called the patellar tendon reflex, the knee-phenomenon, or knee-jerk—and which consists in the sudden contraction of the quadriceps extensor cruris, and (by consequence) sudden extension of the knee-joint, which is evoked by a blow upon the ligamentum patellæ. **Methods of examination.**—In most cases we can obtain it thus: direct the patient to sit with one knee comfortably supported across the other, then strike a sharp blow (even through the dress) upon the ligamentum patellæ. This may be done with the finger-tips, with the edge of a thin book, or, best of all, with the ear-piece of a stethoscope which is shod with a stout india-rubber ring. (These are sold by instrument makers for the purpose of percussing the chest.) The quadriceps cruris responds by a sharp single contraction, which jerks the foot upwards. The essential points in the examination are, that the knee-joint should be partially flexed (a position midway between extension and semiflexion is a good one), so as to put a certain amount of passive tension on the quadriceps; that the other muscles, particularly the hamstrings, should be relaxed, and not consciously or unconsciously held rigid; that the blow should be sharp but elastic, and should fall accurately on the tendon, not on the bone. (Usually a point a little to the inner side of the belly of the tendon is the most sensitive.) If no "jerk" of the foot be obtained, the knee and thigh must be bared; we can then better localise the blow, and can see or perhaps feel the answering contraction of the muscle, for this may be present, though not strong enough to raise the foot. The patient may also be made to sit on the edge of a table with the thighs supported as far as the knee-joints, and the legs dangling loosely over the side; or, in children espe-

cially, we may "stirrup" the foot by taking it in one hand and flexing the knee with a light upward pressure. Should the patient be in bed, we must remember to flex the knee-joint by lifting it in one hand, and we can at the same time feel with this hand whether the hamstring muscles are duly relaxed. Should we fail after all precautions in obtaining a knee-jerk, we may adopt a plan introduced by Jendrassik, sometimes called the "arm-tension" method, or method of "reinforcement"—viz., tell the patient to hook the fingers of either hand into each other and then pull strongly as if to pull them apart, while we test the knee-jerk. This distracts his attention, and possibly reinforces the contraction by increasing the muscular innervation throughout the body.

Difficulty in eliciting the knee-jerk (in the absence of disease) is generally caused as follows: either (1) the patient, either from nervousness or from a mistaken idea of helping the operator, does not allow the limb to hang flaccidly; or (2) in corpulent people with thick limbs and a short ligamentum patellæ, it is difficult to strike the tendon sufficiently accurately and smartly. In this latter case, a firm blow with the edge of the hand may act better than the smart tap with the finger-tips or with the india-rubber-shod stethoscope.

The theory of the knee-phenomenon is still unsettled; some maintain that it is a true reflex action; others that it is a purely muscular contraction, evoked directly by the sudden tension put on the muscle by the blow; others that, though the actual contraction is not a reflex act, yet that it requires for its development a certain muscular tonus, the maintenance whereof is dependent on reflex action. These theories we need not discuss, because, clinically speaking, it conforms in all the conditions which accompany its presence and absence to a true reflex, whatever may be its essential nature physiologically.

Meaning of abnormalities in tendon-

reaction.—The conditions which cause variation in the knee-jerk we will now briefly enumerate. *Absence of knee-jerk*—If after careful and repeated testing the knee-jerk cannot be obtained, disease is probably indicated. In most instances the disease is in the reflex arc itself. The constituent parts of this arc we have given above.* We will now mention some diseases which, by falling upon the various parts of this arc, may abolish the knee-jerk. The whole of the lumbar enlargement may be attacked by myelitis (AE);† the lumbar motor cells by anterior cornual myelitis (E), as in infantile spinal paralysis; the intra-spinal afferent nerve-fibres by posterior sclerosis, as in tabes dorsalis (A); the extra-spinal nerve-roots by meningitis (A or E or AE); the nerve-trunks by neuritis (AE); the nerve-endings by peripheral neuritis (A or E or AE); the muscles by primary myopathy (E).

On the other hand, the knee-jerk may be present but may be feeble or exaggerated; but as it varies much in different persons, even in health, experience alone, aided by a careful consideration of the other factors of the case, must determine whether the feebleness or exaggeration is morbid. Mere feebleness (unless it be unilateral, or unless the feebleness has developed under observation) is not as a rule of much diagnostic importance.

* *Vide* p. 10. The spinal centre (which must be intact to allow of the presence of the knee-jerk) is in the upper lumbar region (2nd to 4th segment : Gowers).

† In this enumeration (A) denotes that the disease attacks the afferent part of the arc, (E) the efferent, (AE) both parts. Disease of the efferent part is usually accompanied by muscular weakness or wasting, or electrical abnormalities. Disease of the efferent part may or may not be accompanied by sensory disturbances. In peripheral neuritis the nerve-endings to the skin may escape while those in the tendons are involved; and again in intra-spinal disease the afferent fibres to the brain may escape while those to the reflex spinal centre are diseased. In such cases the tendon-reflex may disappear, without any loss of cutaneous sensibility.

Exaggeration of the knee-jerk implies, at least, that there is no destructive lesion of the reflex arc, and probably also that the reflex centre is over-active. The precise cause for such over-activity is doubtful. The conditions under which it is found are the following: either (1) without concomitant organic disease, as in hysteria, in certain stages of epileptic fits, in early hemiplegia, or in strychnia-poisoning (Charcot); or (2) with organic disease, commonly in the form of sclerosis of the pyramidal tract (lateral sclerosis). Such sclerosis may be bilateral, either secondary to a transverse lesion of the cord higher up, or (possibly) idiopathic and primary in both lateral columns. The tendon-reflexes are then exaggerated on both sides. Or the sclerosis may be unilateral, secondary to a lesion in the opposite cerebral hemisphere; the tendon-reflexes are then exaggerated on one side only.* With such lateral sclerosis three main symptoms are usually associated: (1) paralysis or paresis from interruption of the motor tract, (2) exaggeration of the tendon-reflexes, and (3) when the sclerosis is well-developed, rigidity of the paralysed limbs.

Rigidity or contracture of muscles.—Rigidity is a striking symptom and must be further considered. It is closely associated with exaggeration of tendon-reflex, which latter symptom not only accompanies, but often is the precursor of rigidity. Rigidity, like exaggerated tendon-reflex, may appear independently of anatomical lesion in the cord, or else may be the index of lateral sclerosis. Both varieties may sometimes be seen in a case of cerebral hæmorrhage. In the early stage of this disease, before the cord has become sclerosed, there may be rigidity which will probably pass off. This is presumably due to cerebral irritation, and is called "early rigidity." "Late rigidity" is a subsequent and more permanent pheno-

* In yet another form of sclerosis—namely, disseminated sclerosis—the tendon-reflexes are usually exaggerated.

menon, and comes on when secondary lateral sclerosis of the cord has followed upon the original cerebral lesion. This rigidity depends on continuous muscular contraction, which exists to a certain extent in all the paralysed muscles, but predominates in some groups. Thus, in the leg the usual effect is to produce extension of the leg and thigh with some adduction and internal rotation of the thigh; in the upper limb, semiflexion with semipronation of the forearm, adduction of the upper arm, with flexion of the fingers into the palm. Thus rigidly extended lower limbs with the knees pressed together are suggestive of bilateral spinal sclerosis; stiffness of one leg, with the attitude just described of the corresponding upper limb, is characteristic of old hemiplegia.

Abnormalities of tendon-reaction apart from disease of the reflex arc.—In considering the association of lateral sclerosis with the knee-jerk, we have already passed beyond the consideration of the reflex arc itself. There is indeed little doubt that the tendon phenomena can be profoundly influenced by the higher (intra-cranial) centres. Experimental physiology would lead us to think that such influence is inhibitory—*i.e.*, that those higher centres hold the spinal reflex centres in check, and that the removal of their influence allows the reflex-action to become excessive. In man this may be also true: for, as we have seen, the knee-jerk is excessive in lateral sclerosis, and one effect of such sclerosis is to obstruct the downward path from the brain. But other facts indicate that the cerebral influence is necessary, not merely for control, but also for production of tendon-reflexes. Thus, after an epileptic seizure there is a stage when the tendon-reflexes are absent, which precedes the stage of exaggeration. Again, absence of tendon-reflexes may be sometimes seen in cerebral tumour, and still more commonly in cerebellar tumour. Lastly, Bastian, Bowlby, and Thorburn have shown that if the spinal cord be completely divided by disease

or injury, the lower limbs are flaccid and the tendon-reflexes absent, and this, though the lumbar centres be intact, and even though there be descending lateral sclerosis.

Ankle-clonus.—Tendon-reactions, like skin reflexes, may be elicited in very various parts of the body. In the lower limb, next to the knee-jerk in importance comes the phenomenon of "ankle-clonus."* This when present is elicited as follows: let the patient's knee be slightly flexed, and support it if necessary with one hand in that position. Then with the other hand press the toes rather suddenly upward so as to cause dorsiflexion of the foot and toes, and maintain a certain pressure on them. The calf muscles will contract in response to the pull on their tendon, and will point the foot down again. They then relax; but the continued upward pressure of the hand causes them to contract again. Thus a rhythmical oscillation of the foot is kept up, which is called "ankle-clonus" or "foot-clonus." Sometimes the mere pressure of the toes on the ground may start such a clonus; and it may continue till the calf-muscles are permanently relaxed by forcibly pointing the foot downwards. Unlike the knee-jerk, ankle-clonus is not present in health; it is to be regarded as an exaggerated tendon phenomenon.

The "front-tap" contraction, also an exaggerated tendon-reaction, is produced by percussing midway between the tibia and fibula about two-thirds down the front of the leg; the response is a contraction of the calf-muscles, and consequent downward pointing of the foot.

In the upper limb there are certain tendon phenomena often to be seen in health, and marked in cases of disease. The triceps cubiti may be made to contract as follows: support the upper arm so that it makes an angle with the trunk, and so that the olecranon is

* Centre at junction of lumbar and sacral regions.

directed outwards, letting the forearm hang vertically; then percuss the triceps tendon. Or a jerk may be obtained at the wrist thus: support the forearm in a position of slight flexion, and tap the lower end of the radius, the biceps cubiti and supinator longus will contract, and jerk the forearm and hand slightly upwards.

In cases of exaggerated tendon-reaction, a contraction has been elicited in the temporal and masseter muscles, by depressing the lower jaw with a smart tap. This has received the euphonious name of "jaw-jerk."

Inco-ordination or ataxy of movement.—Movements even of a simple kind are rarely executed by the contraction of one muscle, but by the combined contraction of several. Unless these various contractions be properly adjusted to each other in time and in force, the movement becomes awkward, irregular, or even impossible. Such a want of adjustment is called "inco-ordination," or sometimes "ataxy." It may be manifest not only in movement (motor ataxy), but in the combination of muscular contractions necessary to retain a fixed position, say that of standing upright; and it may then be called "static ataxy." Both kinds of defect may be shown in walking (locomotor ataxy), because for walking the balance of the trunk must be kept, and the legs moved harmoniously at the same time. Inco-ordination of movement may be caused by defects of various kinds. In the case of such movements as are primarily voluntary and subsequently reflex, it is seen at the stage when the reflex centres are imperfectly educated. Thus an infant's walk is ataxic. But after the movement has been perfectly acquired (that is, become reflex), the balance of muscular action may be disturbed (1) by defect on the motor side, in consequence of which the call made by the centre on the muscles is unequally responded to; (2) by defect in the afferent apparatus, in consequence of which the

centre is misinformed of the position of parts, and therefore directs wrongly. In this latter case the muscular sense, whereby we know the position of our limbs, is usually at fault, and the patient will try to correct this by the aid of sight; but if he be made to shut his eyes, his ataxia becomes evident. If, as frequently happens, the sense of touch is at fault also, the ataxia will be still further aggravated. Incoordination may be also seen in movements which we should at first call purely voluntary. There may be two reasons for this. First, because the defect may be, as just said, motor, and therefore interfering with calls from higher and lower centres alike; or secondly, because in almost every action the muscular adjustment is a reflex matter, though the stimulus to action may be a matter of volition.

Vertigo is the feeling we experience when our perceptions of our relations in space to surrounding objects are discordant or confused. In this general sense it probably corresponds to the popular term "giddiness"; but in a more definite sense vertigo is said to mean the sensation that we ourselves are moving, or objects around us moving, when no such motion exists. When vertigo is severe, the abnormal sensation is followed by actual movement, and the patient who felt himself turning actually does rotate, or he who feels giddy actually falls to the ground. In inquiring after vertigo we should endeavour to make the patient distinguish giddiness from a mere sense of faintness; and if he actually experiences a sense of movement, we should inquire in what direction this movement seems to be.

There are varieties of vertigo which depend on different causes. **Ocular** vertigo results from partial paralysis of an ocular muscle. Our judgment of the direction of an object largely depends on the effort made in the ocular movement for fixing it. In partial paralysis of an ocular muscle, the patient makes an extra effort to fix an object and hence judges

wrongly of its position; if he tries to touch it quickly, he misses it. If he walks, the misjudging of his surroundings makes his gait unsteady, and (subjectively) causes him vertigo. These symptoms disappear when the paralysed eye is closed. **Aural** vertigo is very common; it is a purely sensory defect. One division of the labyrinth—viz., the semicular canals, is appropriated to the perception not of sounds, but of the varying positions of the head according as we lie down, stand upright, or incline the head in any direction. The varying pressure of the endolymph in the ampullæ is thought to furnish the stimulus, and the impressions are probably conveyed by a definite part of the auditory nerve. The constant and correct reception of such impressions is obviously most important for the maintenance of equilibrium. Disease of the middle ear which affects the labyrinth secondarily, disease of the labyrinth itself, of the auditory nerve, or of its central connections, may therefore produce vertigo. This may either take the form of a chronic giddiness and unsteadiness, usually with exacerbations; or there may be paroxysmal attacks, sometimes of great severity, in which the room or the bed, or the patient himself, seems to him to be turning round, tossing up and down, or rushing through space. There are additional symptoms—(1) vomiting and collapse, perhaps due to extension of the irritation to the nuclei of the vagus; (2) tinnitus and deafness, which indicate the aural origin of the affection. Such paroxysmal attacks of vertigo with tinnitus, vomiting, and collapse, associated with a progressive deafness for which no cause can be found in the middle ear, constitute the train of symptoms known as **Menière's disease.** Aural vertigo is not remedied by closing the eyes, it may occur even while the patient is in bed. Movements of the head (*e.g.*, turning over in bed) may bring on the attacks. Except in the mild cases where it is diluted down to a mere giddiness, the sense of movement is well-marked

and definite in direction. Where there is a sense of rotation, this is usually towards the side of the diseased ear.

Gastric vertigo (vertigo a stomacho læso) is said to arise from digestive disturbance, but its existence is denied by some eminent authorities.

Epileptic vertigo occurs either as the prelude to a fit, or as the substitute for a severe fit (petit mal). It has these resemblances to aural vertigo, that it is paroxysmal and often definite as regards the direction of the apparent motion. When associated with definite epileptic fits the diagnosis is easy; but this may be difficult where there is petit mal only. The purely epileptic vertigo will probably be of shorter duration than the aural; there will be no other aural symptoms, but there may be pallor, slight loss of consciousness, and perhaps some spasm even in mild attacks, whereas it is only in severe aural cases that symptoms of this kind occur. But we must not forget that the two causes, epilepsy and ear-disease, may both co-operate. The actual rotation of head, eyes, or body that follows an attack of epileptic vertigo, is usually towards the side to which the patient fancied himself or his surroundings to be turning. The pupil of this side may be a little larger than the other.

These are the most definite forms of vertigo, but the symptom may occur in a variety of nervous diseases. It is evident that the function of equilibration depends on a large number of peripheral organs, and must therefore be represented in a large area of the central nervous system.

Speech defects.—Defects of speech make an important contribution to the symptomatology of nervous diseases. We will, first of all, distinguish three classes of such defect.

(1) **Articulatory.**—The first class embraces speech affections which depend solely on difficulty of articulation. The examples are somewhat various; the

nasal indistinct speech (especially indistinct for labials) of bulbar paralysis; the drawling inarticulate speech which often accompanies hemiplegia; the hesitating confluent speech of general paralysis; and the peculiar "scanning" or "syllabic" speech of disseminated sclerosis, in which words and syllables are brought out slowly and with separation. To these articulatory defects the name "aphemia" has been given. (2) In the second class of speech defects the patient's articulation (for such words as he may still possess) is not at fault; but he has lost the power of expressing his thoughts by language. Thus he is unable (in extreme cases) to say any words, or his speech is limited to some routine words or phrase, or he misuses words. But he still has a correct mental vocabulary, for he understands what is said to him, knows when he himself has spoken wrongly, and his reasoning powers are unimpaired. Only he cannot use his words for purposes of speech; the "way out for words" (as it has been said) is blocked. This defect is called "**aphasia**" (in the limited sense of that term), or sometimes "motor aphasia;" or "Broca's aphasia," because the anatomical lesion corresponding to it—viz., in the posterior part of the third frontal convolution of the left cerebral hemisphere—was first localised by Broca. (3) The third class of defects is called "**amnesia**" (more fully amnesia verborum), because the patient has lost the memory of spoken words. Words addressed to him are no longer symbols, calling up appropriate groups of ideas; nor can he group his own ideas under their appropriate symbols in speech. Therefore, though he hears what is said to him he does not understand it: though his power of utterance is not lost, like the aphasic, his utterances may be confused and unintelligible both to others and himself; and his reason is impaired through failure of the mental imagery of words. This condition has also been called "word-deafness," or "sensory aphasia." The position of the lesion is less certain

than in aphasia proper: probably it lies somewhere near the auditory centre in the neighbourhood of the posterior end of the fissure of Sylvius, on the left side of the brain.

Thus, with respect to spoken language, we may reasonably recognise two cortical areas or "centres," the one devoted to the reproduction of words in speech, and therefore presiding over the (lower) articulatory centres; the second devoted to the reception and storage of spoken words, being fed, so to speak, from the organ of hearing. For written language we are tempted to assume a similar pair of centres, the one for the expression of thought in writing, the second for the storage of visual memories of written (or printed) words; seeing that similar defects have been described on the visual side—viz., an inability to communicate ideas by writing, or "agraphia," corresponding to aphasia; and an inability to comprehend written or printed matter, due to loss of the visual memories of words, or word-blindness.

It remains to be added that for perfect speech not only is the action of some such centres required, but their combined actions. Hence there may be (4) varieties of speech-defects due to lesions, not in the cortical centres themselves, but in the fibres connecting them ("commissural aphasia").

Coma.—A patient who appears to have lost consciousness, and to be able neither to feel, nor speak, nor move voluntarily, and who on recovery has no memory of his past condition, is said to be in a state of coma. There are various degrees of such a state. In the minor forms, called hebetude or stupor, there is little more than dulling of the mental faculties; the patient may tell his name when asked, may put out his tongue when told, &c., but he volunteers nothing, cannot give long answers, and tends to lapse into a somnolent condition. In a state of deeper unconsciousness he neither answers nor appears to hear, his eyes wander aimlessly when opened, but he still may

stir when touched, or even perform quasi-voluntary actions, and the reflex actions still go on. In still deeper coma the breathing becomes stertorous from paralysis of the palate, the optic axes may diverge, and reflex actions begin to fail, so that he cannot swallow, and has no longer control over the sphincters. This stage is usually the prelude to cessation of the respiratory and cardiac movements—*i.e.*, to death. Congestion of the lungs, accompanied by a continuous and considerable rise of temperature, is often the immediate cause of death.

The causes of coma may be very various, and the prognosis of a case varies much, according to the cause at work. But from the physical state of the patient, without an accurate history of the conditions which led up to the attack, it may be difficult or impossible to diagnose the cause. The only safe rule in such a case is to wait and watch, taking such measures to empty the stomach and intestines as will not, by too violent action, aggravate possible cerebral disease. The causes of coma may be roughly classed as functional, toxic, and organic. Under the head of functional coma come cases due to hysteria, epilepsy, insolation, and to diseases such as disseminated sclerosis, general paralysis, and the like, which indeed present organic lesions, but none sufficient to account for the coma at that particular time. The prognosis in most functional cases is favourable. Toxic coma forms a common class of cases, all the more important because prompt remedies, such as emptying the stomach or bowels, or the administration of antidotes, may be needed. Acute alcoholism is perhaps the commonest; next, poisoning by opium and other narcotic drugs. Under this head also may be ranked uræmic and diabetic coma. Lastly, coma may be caused by almost any organic disease of the brain; and it is the more likely to follow in proportion as the cerebral disease is (1) sudden in onset, (2) extensive. Cerebral hæmorrhage is a very common cause; cerebral

embolism or thrombosis a less frequent cause; meningitis, tumour, abscess, produce coma chiefly in their later stages. Into the differential diagnosis of the various conditions which we have enumerated we cannot now enter.

There are other general symptoms due to morbid states of the nervous centres to which we need only make allusion, such as delirium, a symptom fully treated of in books on general medicine; and insanity, which forms the subject of special treatises.

CHAPTER IV.

SYMPTOMS REFERABLE TO THE ORGANS OF SPECIAL SENSE.

THE EYE.

Ocular paralysis.—It is obvious that to understand paralyses of the ocular movements we must have some knowledge of the action of the ocular muscles. The following account of their action is taken chiefly from Dr. Bristowe's excellent text-book of medicine. To each eyeball six muscles are attached—viz., external and internal rectus, superior and inferior rectus, inferior and superior oblique. Let us now take three axes drawn each through the centre of the eyeball, when the eye is at rest and looking straight forwards horizontally (Fig. 35)—

Viz., *AA*, a vertical axis;

OO, a horizontal axis, drawn outwards and forwards from the inner side of the optic disc to the outer margin of the cornea;

RR, another horizontal axis, nearly at right angles to *OO*, coming out in front near the inner canthus.

Then the action of the external and internal recti is to rotate the eyeball round the vertical axis AA, the external rectus in the outward direction, the internal in the inward. The oblique muscles rotate it round the axis *OO*, the *inferior* giving it an *upward* movement, the *superior* a *downward*. The superior and inferior recti rotate it round the axis *RR*, the

superior giving it an upward, the inferior a downward movement. The movements imparted to the cornea by these various muscles are as follows: by the external and internal recti, simple outward or inward

FIG. 35.

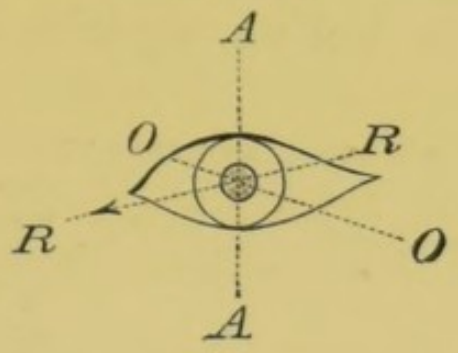


Diagram to show axes of rotation produced by the ocular muscles (left eye). (Modified from Bristowe.)

movement respectively; by the obliqui, movements differing according to the position from which the cornea starts, but always along the concentric circles, in Fig. 36, of which *O* is the centre; by the superior

FIG. 36.

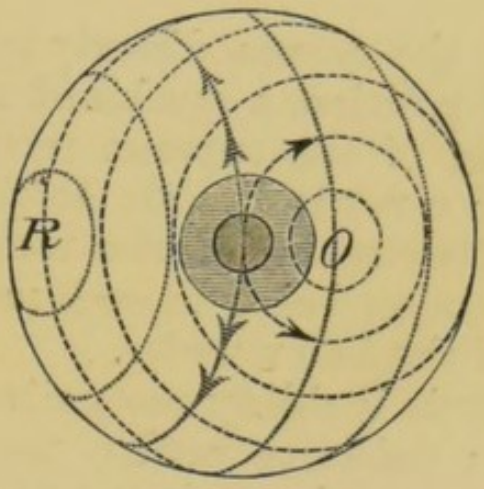


Diagram to show the lines of movement produced by the recti muscles (superior and inferior), and by the obliqui (superior and inferior respectively). Left eye. (After Bristowe.)

and inferior recti movements which vary in the same manner, but always along the concentric circles, of which *R* is the centre. Thus, supposing the eye to be looking along the axis *OO*, the obliqui will give no

movement to the cornea save that of rotation, but the superior and inferior recti will give an up and down movement respectively. Suppose it to be looking along the axis RR, then the superior and inferior recti will only give a rotatory movement, but the oblique will give an up and down movement. Suppose, again, the eye is looking straight forwards, then acting singly,

The inferior oblique	will move the cornea			upward and outward, in the direction of the upward *single* arrow.
The superior oblique	,,	,,	,,	downward and outward in the direction of the downward *single* arrow.
The superior rectus	,,	,,	,,	upward and inward in the direction of the upward *double* arrow.
The inferior rectus	,,	,,	,,	downward and inward in the direction of the downward *double* arrow.

There will be in each case a tilting of the vertical meridian of the cornea; for this meridian preserves the direction of the concentric circle along which the cornea moves.

But to carry the cornea directly upward from the original central position, a combined action is necessary on the part of the superior rectus and inferior oblique; and to carry it directly downward, a similar combination between the inferior rectus and superior oblique; the motion being in each case the resultant of the action of the two muscles.

Ocular paralysis is chiefly recognised by the following symptoms: defective ocular movement; squint or want of correspondence between the optic axes; diplopia or double vision.

Defective movement is recognised by making the patient follow with his eyes an object moved in various directions; the affected eye lags behind the other, when the movement to be elicited requires the action of the paralysed muscle. Thus, when the object is moved in the direction of the muscle's action, it may

be easy, in simple and well-marked cases, to recognise the nature of the paralysis.

The squint, which is simply the result of the defect of movement, is recognised by the same means. Squint may exist, however, even when the eyes are at rest, owing to the unbalanced action of the muscles which antagonise those that are paralysed. The eye squints in a direction opposite to the line of action of the paralysed muscle.

The diplopia is caused by the non-correspondence of the optic axes. Owing to this the images from the

FIG. 37.

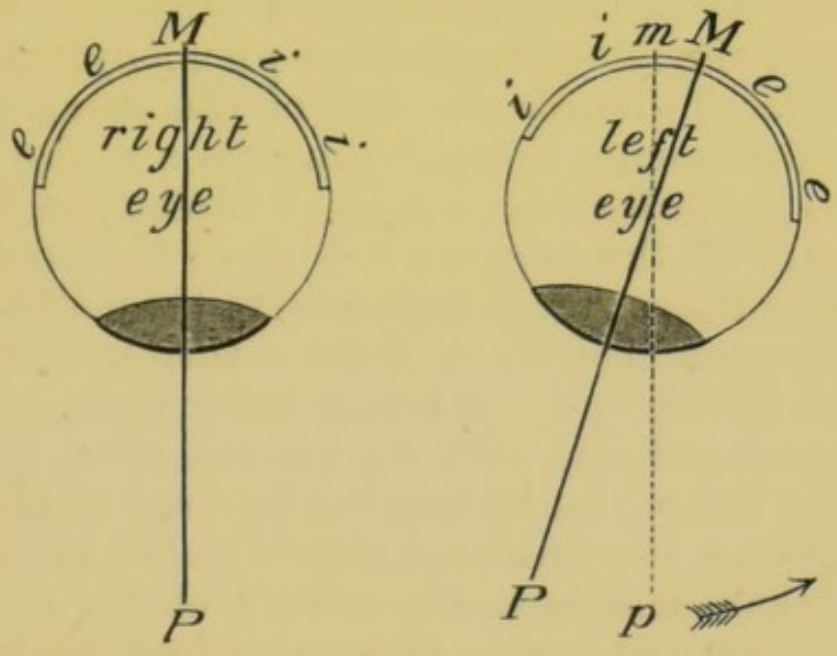


Diagram to illustrate squint from paralysis of left external rectus.

The object fixed is supposed to be in the distance, and the rays from it are parallel. These coincide with the principal axis, *PM*, of the right eye, but not with that of the left.

M, macula lutea.

i i, internal half of each retina corresponding to external half of its visual field.

e e, external half of each retina corresponding to internal half of its visual field.

The arrow shows the direction in which the false image is displaced.

object fixed do not fall on corresponding points in the two retinæ; and are therefore referred to different directions in space. In Fig. 37 the left eye is squinting inwards, and therefore rays from a distant object

which in the right eye fall on the macula lutea, fall in the left upon a point internal to this. But since the internal half of the retina would receive under normal conditions rays from objects in the outer half of the field of vision, the image formed in the squinting eye is referred to a place external to (*i.e.*, to left of) its real position. This is called the false image; it is generally fainter than the other one because it is formed further from the macula lutea.* The displacement of the false image is in the opposite direction to the squint, and therefore in the direction of the line of action of the paralysed muscle. Diplopia is said to be crossed when the false image is displaced towards the side of the non-paralysed eye; homonymous when it is displaced towards the side of the paralysed eye. Just as the amount of squint increases as the patient attempts to utilise the paralysed muscle, so does the divergence of the images; but since the false image is brighter the nearer it is formed to the macula lutea, it may happen that the diplopia is then most troublesome, when the squint is barely perceptible.

We may add this, that the patient often attempts to correct the squint by turning his head in the direction in which the paralysed muscle ought to carry the eye, so that sometimes the nature of his complaint can be guessed at once. Of vertigo from ocular paralysis we have already spoken.

Two examples may be given of paralysis of an individual ocular muscle.

Left external rectus.—Innervation from sixth nerve; action, turns eye horizontally outwards (to left).

Symptoms of paralysis; left eye lags or is immobile when patient looks to left; left eye squints inwards; false image is placed to left of true image (homonymous diplopia), and is on the same plane with it, and is not tilted.

* If there is any doubt as to which image belongs to which eye, it may be settled by using a candle-flame for object and making the patient hold a coloured glass in front of one eye.

Left superior oblique.—Innervation from fourth nerve; action (supposing the eye to start from a position of rest) carries the cornea downwards and to left, at the same time tilting the upper end of its vertical meridian inwards (to right).

Symptoms of paralysis; squint little or none as the eye looks straight forward, but if the patient attempts to look downwards (or, still more, if downwards and to left), the left cornea remains rather above and to right of the position it should take up, and also appears to screw round slightly in the direction of the hands of a watch; homonymous diplopia (on looking downwards and to left), the false image being placed below and to left of the true, and its upper end tilted to right.

All the remaining muscles of the eyeball, and also the elevator of the upper lid, are supplied by the third nerve. Complete paralysis of the third nerve-trunk causes the following symptoms: ptosis—*i.e.*, closure of the eye from inability to raise the upper lid; loss of movement of the eyeball, except the outward movement (supplied by the external rectus and sixth nerve), and the downward and outward movement (supplied by the superior oblique and fourth nerve); external squint with crossed diplopia. Further, the pupil is somewhat larger than that of the sound eye, and does not contract under the stimulus of light or of accommodation effort, because the third nerve supplies the sphincter pupillæ; and the power of accommodation is also lost, because the third nerve supplies the ciliary muscle. Syphilis is a frequent cause of paralysis of the third nerve. This, or indeed any form of ocular paralysis, may occur in the early stages of tabes dorsalis, or less frequently of disseminated sclerosis; in these cases the paralysis is generally transient. Recurrent ocular paralysis may occur in connection with migraine.

Complex ocular paralysis.—But ocular paralysis is by no means always limited to one muscle, or the muscles supplied by one nerve-trunk. It may

affect simultaneously several independent muscles, or muscles of the two eyes, or there may be loss of particular movements rather than paralysis of any particular muscles. Such complicated ocular paralysis may be produced in various ways: (1) By peripheral neuritis in the district of the oculo-motor nerves, an example of which is to be found in the ocular palsy which occasionally accompanies the limb-paralysis of alcoholic neuritis. (2) By central disease affecting either the ocular nerve nuclei, or the root-fibres as they run from the nuclei towards their superficial origin. If such disease be a coarse lesion, such as a tumour, hæmorrhage, &c., it will probably produce other symptoms besides ocular palsy, most likely cross paralysis (*vide* p. 84), and the ocular paralysis will often be bilateral, seeing that the nerve nuclei and root-fibres, at any rate those of the two third nerves, lie near together. If the disease be microscopic—viz., a chronic degeneration of the oculo-motor nuclei, the symptoms are still more likely to be bilateral. Such nuclear disease is usually chronic and progressive, and it gives rise to the set of symptoms known as progressive ophthalmoplegia—ophthalmoplegia externa when the movements of the eyeballs gradually fail; ophthalmoplegia interna when the movement of the pupil and the power of accommodation are affected. Such ophthalmoplegia is analogous to the progressive muscular atrophy which depends on degeneration of the anterior cornual cells of the spinal cord, and indeed it may actually be accompanied by muscular atrophy, or may form a part of some other spinal degeneration, such as tabes. An acute affection of the region of the third nucleus has also been described, of rare occurrence, probably inflammatory in nature, occurring generally in drinkers, associated with symptoms of delirium tremens, and ending fatally.

There may be loss of a combined movement of the two eyes, the muscles engaged being paralysed for that movement only, and not for other movements.

Take, for example, the movement of directing the two eyes laterally to the left. This requires a combined action on the part of the left external rectus and the right internal rectus; and it may be that the two muscles are powerless for this purpose, though at the same time the internal rectus retains its power for other movements, such as that of convergence. For the lateral movement just described the two muscles are said to be "yoked" or "conjugate," and the movement is called a conjugate movement, or, more generally, an

FIG. 38.

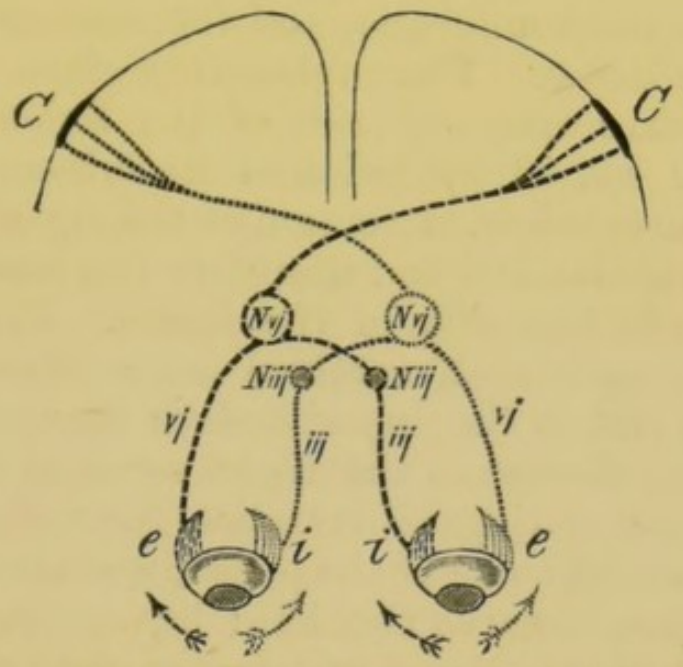


Diagram to illlustrate the mechanism of conjugate lateral movements of the eyes.

C, Cortical centre, stimulation of which causes rotation of both eyes towards opposite side, and paralysis of which allows conjugate deviation to the same side.

Nvj, Nucleus of 6th nerve, connected by the fibres of the 6th nerve (*vj*) with the external rectus (*e*) of the eye of the same side, and through the opposite 3rd nucleus (*Niij*) and 3rd nerve (*iij*) with the internal rectus (*i*) of the opposite side.

associated movement. It is believed that the sixth nucleus has such connections with the external rectus of the same side (*viâ* the sixth nerve-trunk), and with internal rectus of the other side (*viâ* the third nucleus and nerve-trunk) as enables it to produce the aforesaid

conjugate movement of the eyes to the same side. Destruction of one sixth nucleus will consequently produce loss of conjugate movement of the eyeballs towards the side of the lesion.

Such movements are dependent also upon higher centres in the cerebral hemisphere. Experiment shows that in the cortex of each cerebral hemisphere in the monkey there is a centre or centres, the action of which is to cause turning of the eyes and head to the opposite side. In hemiplegia from cerebral disease in man, there is often a turning (conjugate deviation) of the head and eyes. They turn usually away from the side of the hemiplegia, and therefore towards the side of the lesion. The explanation given is that the cortical centre upon the side of the lesion being destroyed, or cut off by lesion of its sub-cortical fibres from the parts below, the action of the opposite cortical centre is unbalanced, and therefore the head and eyes look towards the side of the lesion. Probably the fibres from each such cerebral centre descend, cross, and finally end in the opposite sixth nucleus.* Thus, a *paralysing* disease in the right cerebral hemisphere would cause conjugate deviation towards the right, while in the right side of the pons it would cause conjugate deviation towards the left; but an *irritative* disease (*i.e.*, such as caused undue stimulation) in either locality, would produce just the reverse effects.

There are other associated movements of the eyes which may be lost in disease, such as the movement of convergence, and the movement of looking upwards.

Nystagmus.—Sometimes the eyes, instead of remaining steady when directed towards an object, or instead of following a moving object equably and steadily, exhibit continuous jerks or small oscillations. This condition is called nystagmus. It may arise (apart from actual nervous disease) from defects of

* That is, the fibres which regulate the movement of the eyes: those for the movement of the head would of course end in the nuclei which act on other muscles.

vision either congenital or dating from early childhood, or in connection with some continuous strain upon the ocular muscles, as in "miners' nystagmus." Nystagmus arising from nervous disease is said to manifest itself more when the eyes are used for fixing or following an object than when they are at rest. The minor degrees of it are certainly best demonstrated by making the patient fix an object far to one side of him, thereby putting some strain upon the muscles. These jerky movements are (usually) present in both eyes; they may be horizontal in direction, vertical, rotatory, or quite irregular. The nervous diseases in which nystagmus occurs are: disseminated sclerosis (towards the diagnosis of which it may contribute in an important degree); the variety of locomotor ataxy, known as hereditary ataxy or Friedreich's disease; incomplete paralysis of the ocular muscles, as in tumours of the pons, peripheral neuritis. In disseminated sclerosis a fair comparison may be made between this tremor of the eyes and the tremor of the limbs.

Paralysis of pupil and ciliary muscle.—Leaving the movements of the eyeball, we come to the internal muscles of the eye. These are, (1) the ciliary muscle, (2) the sphincter of the iris, (3) the dilator of the iris. The last-mentioned muscle is innervated from the sympathetic, the other two from the third nerve. Sometimes all these muscles are affected by a paralysis, progressive in character, and depending probably on degeneration of the nerve-nuclei in the pons. This is called ophthalmoplegia interna; it is analogous to, and may be associated with, the ophthalmoplegia externa which we have mentioned. Paralysis of the ciliary muscle only, the symptom being loss of accommodation, is most commonly due to diphtheritic paralysis.* It occurs in conjunction with other symptoms in paralysis of the third nerve. As to the pupils, in the natural state their apertures are circular, and

* Omitting of course presbyopia, and conditions purely ophthalmic.

equal on the two sides; the pupil contracts when light is thrown on the corresponding retina (reflex action), or on the opposite retina (crossed or consensual reflex); it contracts, too, with the effort of convergence or accommodation in looking at a near object (associated action); it dilates when the eye is shaded; and dilates when pain is inflicted, as by severely pinching, pricking, or faradising the skin of the neck. The size of the pupils varies with many circumstances; they are often small in elderly people, large in myopic patients, large in states of excitement, or after the occurrence of an epileptic fit. An undue contraction of the pupils is called "miosis," the reverse condition "mydriasis." An important and early symptom of tabes dorsalis is often furnished by the pupils. The reflex contraction to light is not to be obtained, though there is still a contraction when the patient accommodates. This is called reflex iridoplegia, or sometimes the "Argyll-Robertson phenomenon." Care must be taken in examining for it that the patient looks steadily into the distance all the time, otherwise the accommodation is exerted, and the associated contraction of the pupil appears. In some cases, however, even this associated action of the pupil is abolished, so that it is completely immobile.* Usually, though not always, the pupils in tabes dorsalis are small; "spinal miosis" this is called. The movement of dilatation upon the application of painful stimuli is said to be abolished also, but this may be difficult to test. Tabes dorsalis is by far the most frequent cause of reflex iridoplegia, but it occurs sometimes in general paralysis of the insane, and occasionally in old syphilitic cases.

In paralysis of the cervical sympathetic, the pupil of the affected side is a little smaller than the other one, since the sympathetic supplies the dilator fibres. Other symptoms of paralysis of the sympathetic are as follows: the eyelids droop somewhat, the eyeball

* It should be ascertained that such immobility is not due to adhesions from old iritis, &c.

appears retracted, so that the palpebral opening looks smaller than on the other side. Flushing and heat of the face upon the side of the paralysis may occur. Aneurysms, enlarged glands, tumours at the root of the neck or at the apex of the lung, may cause such paralysis of the sympathetic. The peculiarities of pupil may be seen in disease of the cervical or upper dorsal cord, or of the uppermost dorsal nerve-roots where the cilio-spinal fibres of the sympathetic leave the cord.

Affections of fundus oculi.—It is hardly necessary to insist upon the importance of examining the fundus oculi. It is here only that we can directly inspect a nerve and its terminal expansions. Moreover, the condition of the optic nerve has a more than local importance; it may furnish the best indication of central nervous disease. Thirdly, in the fundus oculi, as in other parts of the body, may appear symptoms of constitutional states, which need not necessarily be nervous in origin, but which may have important bearings on nervous disease, such as syphilis, Bright's disease, &c.

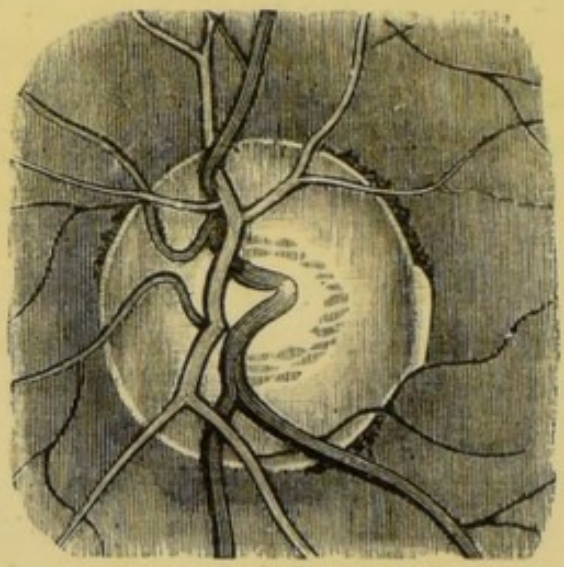
For a description of the methods of ophthalmoscopic examination, and of those conditions of the media which must be taken into consideration in such examination, we shall refer the reader to text-books on ophthalmology. The "direct" method is the easiest and best for the determination of minute changes in the fundus.

The chief morbid conditions of the optic disc or papilla are: (1) Optic neuritis, or papillitis; (2) optic atrophy.

Optic neuritis. — In the normal condition the optic disc is circular, of translucent pink hue, variable in different persons, but almost always paler than the surrounding fundus; its edge is distinctly traceable, generally more sharply marked at the sides than above or below, and particularly on the nasal side; the edge may or may not be bordered with pigment; its centre (the lamina cribrosa) may be slightly de-

pressed, grey, and punctate in appearance; from the centre issue the retinal vessels, which mainly pass upwards and downwards, and then branch over the fundus. The veins are larger and darker than the arteries, and the vessels show a light streak running

FIG. 39.



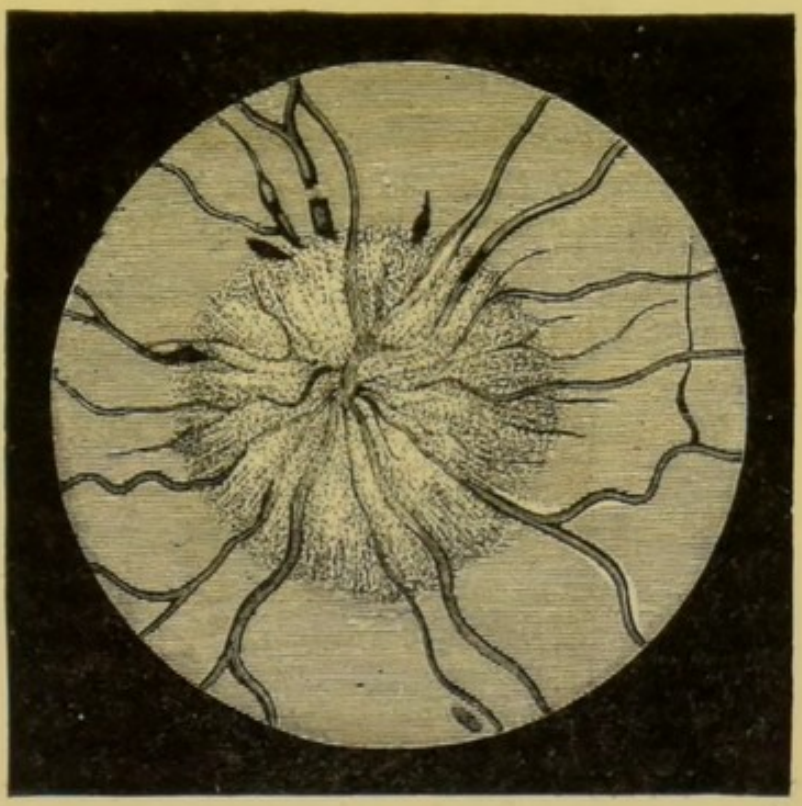
Healthy optic disc, erect image. (From Nettleship, after Jäger.)

along their axis with a coloured streak on each side; this "double contour" being due to the refraction of the light from their rounded surface. The nasal half of the disc is often paler than the temporal.

In optic neuritis the colour of the disc becomes at first deeper, approaching that of the fundus (congested or hyperæmic disc), though upon this alone much stress cannot be laid; next, the edge of the disc, as seen by the direct method, becomes indefinable and hazy; the haze increasing, begins to obscure the vessels here and there; then swelling of the disc sets in, its central pit becomes filled up, the disc as a whole becomes raised above the level of the fundus; its edges look frayed out and spread over the adjacent parts of the fundus. The swelling of the disc is evidenced by the difference of focussing required for it and for the fundus; by the difference in the apparent movement of the vessels on the disc and of those

on the fundus as the observer's eye is shifted, by the curve of the vessels and their loss of double contour as they pass off the disc. As the swelling goes on the veins enlarge and become tortuous, the arteries become smaller; hæmorrhages may appear both upon

FIG. 40.

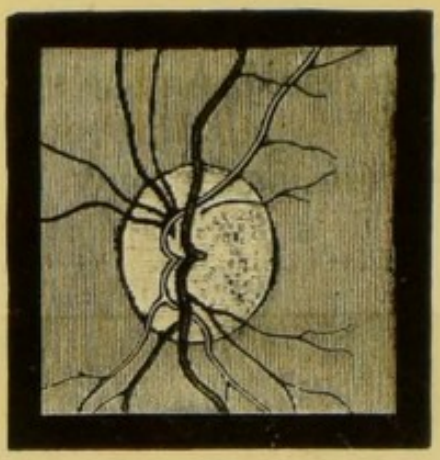


Severe optic neuritis or papillitis. (From Nettleship, after Hughlings Jackson.)

and around the disc; the neighbouring parts of the fundus become involved in the haziness and swelling. This extreme condition of optic neuritis was formerly called "choked disc."

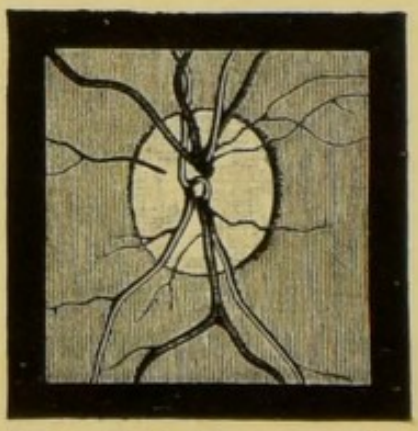
Optic atrophy.—In optic atrophy the disc is no longer translucent, but has a dead opaque colour, either chalky white, or dull grey, or yellowish. The central pit may appear either filled up, or more hollow than usual. In primary atrophy the edges of the disc are sharply cut; when the atrophy is secondary to neuritis (post-neuritic or post-papillitic atrophy), the edges may remain ill-defined, at any rate here and there, and the vessels may be smaller

FIG. 41.



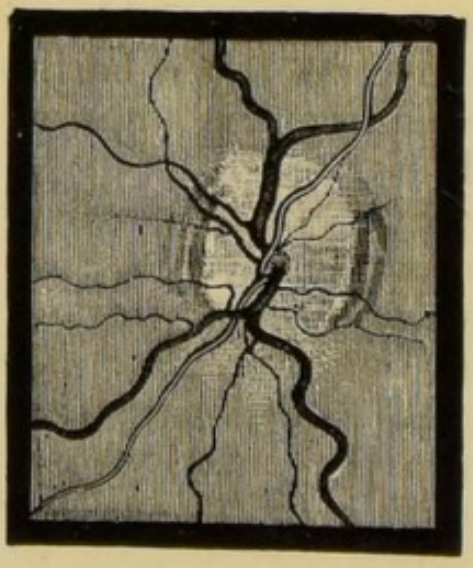
Simple atrophy of disc. Stippling of lamina cribrosa exposed. (From Nettleship, after Wecker.)

FIG. 42.



Atrophy of disc from spinal disease. Lamina cribrosa concealed, vessels normal. (From Nettleship, after Wecker.)

FIG. 43.



Atrophy of disc after papillitis (Nettleship).

than normal; but this is not necessarily the case, and there may be no means of distinguishing the secondary from the primary atrophy. Atrophy may be partial, but it must be remembered that even in a normal disc the nasal half is apt to be paler, and its nasal edge more defined than the rest.

Optic atrophy may be also secondary to various forms of retinitis, to embolism of the retinal artery, and other purely ocular affections.

Indications afforded by optic neuritis or atrophy.—These affections of the optic nerve have a twofold bearing. First, upon the patient's vision. This is a question principally for ophthalmic surgeons, and we shall only remark here that optic atrophy is almost always accompanied by impairment of vision, and may destroy vision altogether; but optic neuritis does not in all cases cause impairment. Therefore, the eyes should be examined, even when there is no complaint of loss of sight. Secondly, upon the presence of nervous disease elsewhere. Optic neuritis, affecting both eyes (double optic neuritis), is generally indicative of coarse organic disease within the cranium. The other symptoms of intra-cranial disease—headache, vomiting, convulsions, and even in some cases paralysis—may be due to purely functional causes; but when optic neuritis is superadded, we may as a rule look for structural lesions, hence its importance in diagnosis. Of such lesions, tumour is the commonest, either new growth in the strict sense (glioma, sarcoma, carcinoma); inflammatory new growths, such as gummata and tubercular masses; or the rarer parasitic growths, such as hydatids, actino-mycosis, &c. The character of the tumour makes no difference to the optic neuritis; and as to locality, neuritis may accompany a tumour in any position. How the tumour causes the optic neuritis we do not fully know. Optic neuritis need not be present in all cases of intra-cranial tumour; but it will probably be found at some stage or other of most tumours, if the

eyes be frequently examined. Other organic cerebral lesions, which may cause optic neuritis, are, meningitis, abscess, or, it may be, thrombosis of sinuses, or hydrocephalus. It is very rarely present in cases of simple cerebral softening or cerebral hæmorrhage. One or two cases have been described of optic neuritis in nervous diseases which were not intra-cranial—myelitis, disseminated sclerosis, peripheral neuritis; but these are also rarities. But it may be produced by causes quite outside the nervous system, such as Bright's disease, or perhaps anæmia. Hypermetropia, again, may cause appearances exactly resembling optic neuritis.

Optic atrophy, we have said, may be primary or secondary to pre-existing neuritis. If secondary, it points to some of the conditions which we have just enumerated as causes of the neuritis, as either existing or having at one time existed. If primary, optic atrophy is usually connected with degenerative disease of the spinal cord; most commonly with tabes dorsalis (of which it may constitute a very early symptom), sometimes with the allied disease, general paralysis of the insane (spinal form), and also with disseminated sclerosis. Such atrophy may begin in one eye, but both are affected in time. There is a rare form of optic atrophy which seems to run in families.

Affections of the other parts of the eye, even of the choroid and retina, have not the same direct bearing upon central nervous disease as have optic neuritis and optic atrophy. Nevertheless, an inspection of these parts may throw great light upon the constitutional condition of the patient, and thereby upon his nervous symptoms. Tubercles in the choroid may indicate the nature of a doubtful meningitis; syphilis may be revealed by choroidal changes; Bright's disease by its characteristic retinitis.

Affections of vision.—The function of vision may be considered under the heads of central vision,

the field of vision, and colour-vision (central or peripheral).

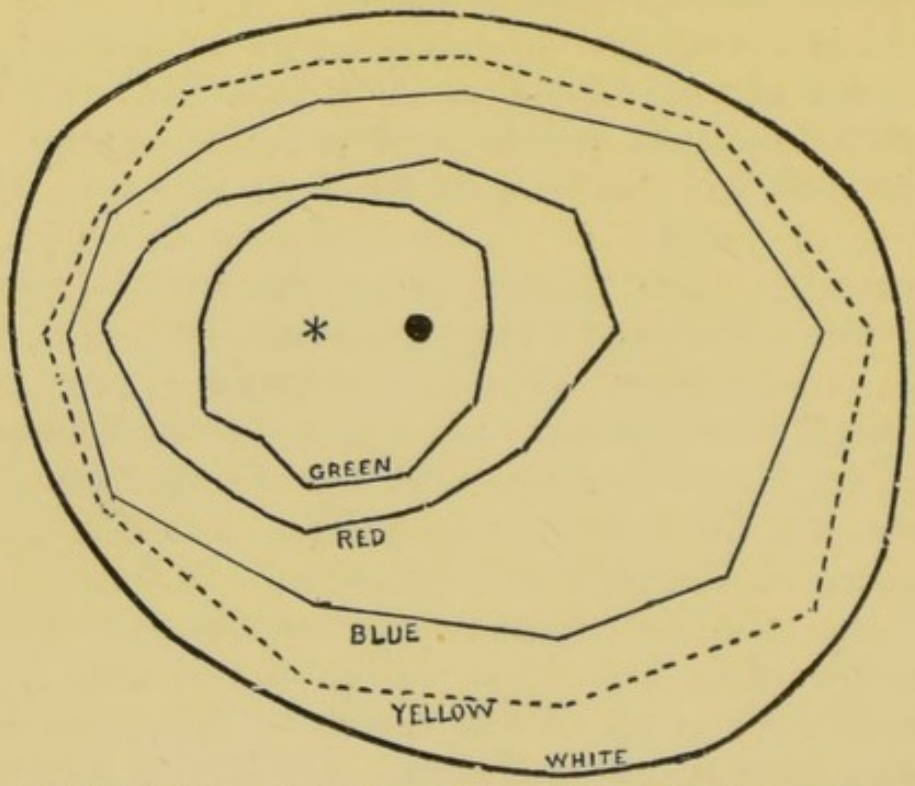
Of central vision.—When the eye is directed to any small object, which may be called the "fixing point," the rays from this object fall on the yellow spot, and since this is the most sensitive spot in the retina, the object is seen with the greatest attainable clearness. This is central vision. When "acuteness of vision" without qualification is spoken of, the acuteness of central vision is meant. It can be accurately measured (when sufficiently good) by test-types.* When too bad to allow of any type being read, it is judged of by the distance at which the patient can count fingers, or, in the worst cases, by his power of distinguishing light from darkness. But simple diminution of acuteness of vision is more often than not due to disease of the eye itself; and therefore, before we proceed to associate it with central nervous disease, we must satisfy ourselves that the media, and more particularly the refraction, are not at fault, and that the ophthalmoscopic appearances of the choroid, retina, and optic nerve are healthy. The following are some of the causes of defective vision without demonstrable disease of the eye, and with little or no changes in the disc: toxic agents, such as tobacco, carbon bisulphide, quinine, perhaps alcohol, producing probably some neuritis; retro-bulbar neuritis—*i.e.*, neuritis too far back to be visible ophthalmoscopically: central causes—uræmia, hysteria, migraine, epilepsy (temporarily), reflex irritation.

Of visual field.—But disease of parts behind the optic nerve usually causes symptoms which have

* Snellen's test-types are so constructed that each type can be read, when vision is normal, at a distance corresponding to the number of the types. Thus (if constructed for metres) when No. 6 is read at 6 metres, vision = $\frac{6}{6}$, *i.e.*, is normal; when No. 6 is read at 3 metres, vision = $\frac{3}{6}$, or half the normal, and so on. Small types, to be held by the patient, may be used, if we are satisfied that his accommodation is sufficiently powerful.

special relations to the fields of vision. The field of vision (Fig. 44) is the area around the fixing point within which objects can be perceived without moving the eye. Each retina has its own field of vision; in the centre is the fixing point which corresponds to the yellow spot of the retina, while the remainder corre-

FIG. 44.



Field of vision of right eye for white and colours on a dull day. (From Ross, after Gowers.)
The asterisk indicates the fixing point, the black dot the blind spot.

sponds to the more peripheral parts of the retina. Since the rays from objects in the field cross between the cornea and the fundus, the left half of the field will correspond to the right half of the retina, and *vice versâ*, the upper half of the field to the lower half of the retina, and *vice versâ*.* The fields of the

* *Examination of Fields of Vision.*—A rough examination of the field may be made thus:—The patient closes one eye, and with the other fixes the corresponding eye of the examiner (so that the latter can see that the patient does not move his eye, and can at the same time contrast the patient's field with his own). The fingers of the examiner are then brought gradually from various points of the peri-

two eyes are symmetrical, they do not coincide exactly, for it is evident from their shape that the right half of the right eye's field overlaps the right half of the left eye's (and reversely for the left halves), but they coincide in this sense, that rays from the same object

FIG. 45.

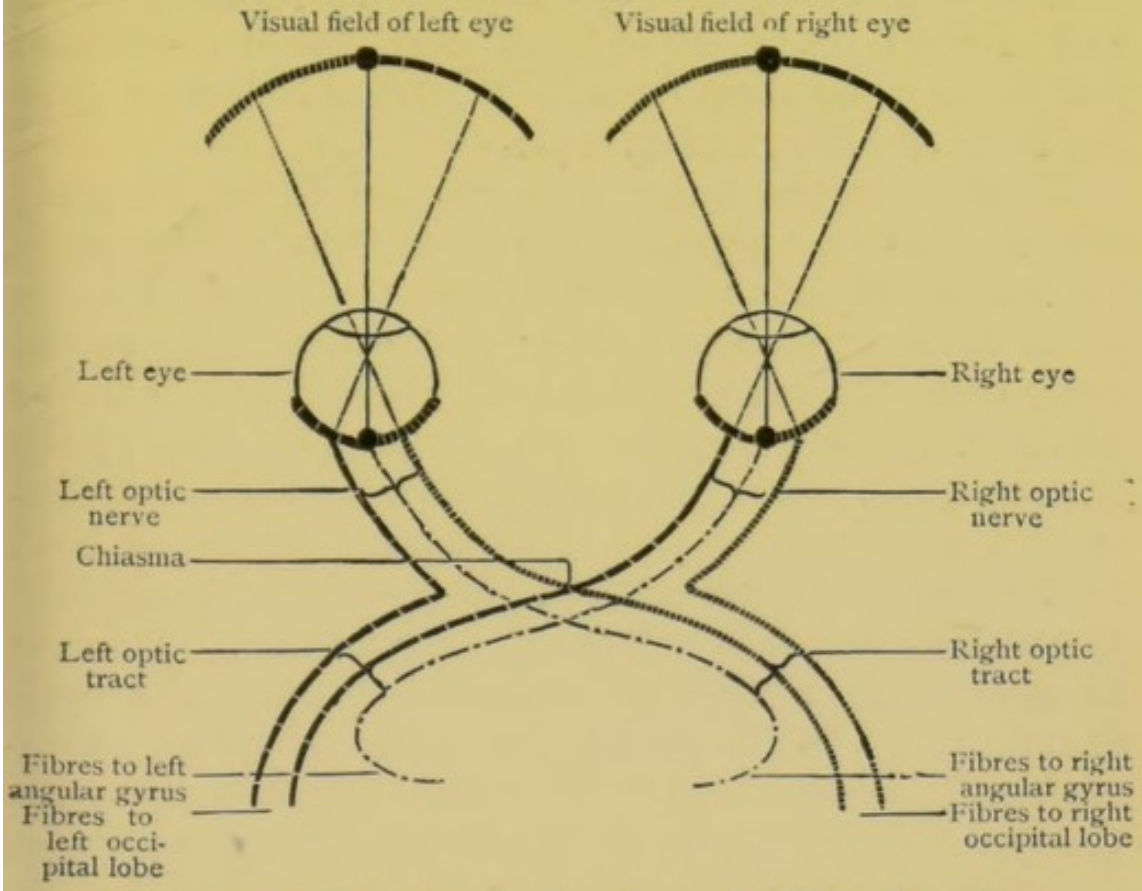


Diagram to illustrate the distribution of fibres from the occipital lobes to the lateral halves of the retinæ.

(the fixing point being the same for both eyes) fall on corresponding points of both retinæ—*i.e.*, on points

phery towards the fixing point, and the patient has to say when they come into sight. For more accurate examination and recording, a perimeter must be used. A movable arm is made in the shape of a quadrant of a circle, and divided into angles, the patient's eye is placed at the centre of the circle, and the fixing point is at one end of the quadrant: the object, usually a small square of paper, is made to travel along the quadrant, and the angle at which it becomes visible is noted, and marked off on a diagram; the quadrant can be rotated around the fixing point, and the process of estimating and recording the angle is thus repeated for as many meridians as is thought to be necessary.

symmetrically disposed with respect to the yellow spot, to right or left of it, above or below. Taking then the visual field of the two eyes thus combined, and supposing it divided into two lateral halves, the right half of it will be perceived by the left half of either retina (inner half of right retina, outer half of left), and the left half of it by the right half of each retina (outer half of right, inner half of left). Now, the disposition of the fibres proceeding from the lateral halves of each retina is as follows (Fig. 45):—

Those from the outer half proceed along the optic nerve-trunk in company with those from the inner as far as the chiasma; but here they part company, the fibres from the outer half of the retina going to the optic tract of the same side, the fibres from the inner half of the retina crossing over to the optic tract of the opposite side. Thus the optic tract of the right side contains the fibres from the right lateral halves of both retinæ, and the optic tract of the left side contains the fibres from the left halves of both retinæ. This arrangement is continued upwards through the optic radiations of Gratiolet into the occipital lobes. Consequently, a lesion of one occipital lobe or of the optic tract of one side produces blindness of the *same named halves of both retinæ*, and loss of the *opposite half of the field* of vision. This is called "lateral" or "homonymous" hemianopia. Disease of the chiasma itself will damage the decussating fibres, and since these come from the inner half of each retina, there will be loss of the outer or temporal half of the field of vision in each eye (not corresponding halves as in the last case). This is called "temporal hemianopia." Progress of disease at the chiasma may cause total blindness of both eyes.

So far as regards the occipital lobes and the optic tracts, the scheme just given is fairly satisfactory. But in lesions of the angular gyrus,* and still more

* It must be noted, however, that the whole question of the relation of the angular gyrus to vision is still unsettled.

frequently in cases of hysterical hemianæsthesia, a different state of things is observed. The central vision of the eye opposite the lesion, or on the side of the hemianæsthesia, is diminished in acuity, perhaps almost abolished, and the field of vision is concentrically narrowed, while a similar but much less marked affection may be discovered in the other side. It would seem that each angular gyrus stands in relation to the vision, and particularly the central vision, of the opposite eye, and in a less degree to that of the eye of the same side. Apparently each such centre (in the lower animals at any rate) can supplement the action of the corresponding centre on the other side.

Besides hemianopic defects and concentric narrowing, the fields of vision may be affected in other ways. A blind patch may appear in the centre of the field (central scotoma), or sector-like pieces of the field may be blotted out. Atrophy of the optic nerve is usually the cause of such affection.

Of colour-vision.—Colour-vision may be affected in connection with, or independently of, other defects of vision. Some people, whose sight is otherwise good, are unable from birth to perceive certain colours. This constitutes congenital colour-blindness; it is a defect that occasionally runs in families. Such a condition may be acquired; it is then generally due to optic atrophy, but may be also caused by hysteria or some cerebral lesion. Colour-vision may be tested with sets of coloured wools which are supplied for the purpose. In acquired colour-blindness it may be sufficient to ask the patient to name the colours shown to him. But it is better to make him sort them into colours which seem to him unlike and like, or to match different colours chosen for the purpose; being unable to perceive some one fundamental colour, he will class as like some wools which to a normal eye appear different.

There are visual fields for colours, as for white light (*vide* Fig. 44). These are less extensive than

the field for white. The fields for colours may be concentrically narrowed (just as the fields for white): in some rare cases there may be hemianopia for colours.

THE SENSE OF HEARING.

Probably affections of hearing have as close a bearing on nervous disease as those of vision. But the deeper parts of the ear are not, like those of the eye, open to direct inspection; hence the investigation of them is the less easy, and has been less fruitful of result.

The auditory nerve and its expansion in the labyrinth is devoted to two different functions: (1) that of hearing (cochlea); (2) that of receiving impressions necessary for equilibration (semicircular canals). Of disturbances of this second function we have already spoken under the head of "aural vertigo" (p. 106). It should be as natural to examine the ear in a case of vertigo as it is in a case of deafness.

A definite and general standard of hearing has not yet been framed. The hearing distance is usually estimated by a watch. If a given watch is audible by a normal ear, say at 24 inches, and the patient can only hear it at three inches, then the hearing distance of his ear may be expressed as $\frac{3}{24}$, and so on. Each ear must (it is needless to say) be tested separately, the watch must be held vertically, and in a straight line with the ear. The hearing *power* is said to vary not as the hearing distance, but as the square of the distance. The watch test is open to the objection that different kinds of sound may be heard with different degrees of distinctness; a patient who can hear the watch may be deaf to conversation, and so on. It is evident that in this method of examination the sound is conveyed from the watch by the air through the meatus to the membrana tympani, and thence *viâ* the ossicles to the labyrinth. This is called "aërial conduction," or it may be called "meatal hearing." But

if a sounding body such as the watch or a tuning-fork be placed on the bones of the head (the forehead, vertex, mastoid process, or teeth), it is still heard even though the meatus be closed. This is called "perosseous conduction or perosseous hearing." The distinction between meatal and perosseous hearing may be used as a means of localising the defect which causes deafness in a given case. If the deafness is due to disease of the external meatus, or of the tympanum, the perosseous hearing is not diminished, it may be even increased. (This fact may easily be verified thus: stop one external meatus with a moderate degree of pressure, and place a vibrating tuning-fork on the middle line of the forehead or vertex, the sound will be heard best in the ear which is closed.) But if the disease be in the labyrinth, auditory nerve, or nervous centres, both meatal and perosseous hearing are diminished *pari passu*. So that the tuning-fork placed on the middle line of the skull should be best heard with the diseased ear when the disease is peripheral (*i.e.*, in the external meatus or tympanum), best heard with the sound ear when the disease is central (in labyrinth or nervous apparatus). But this test is perhaps less valuable than it appears to be, because (1) the patient's answers may be misleading; (2) both ears may be diseased; (3) the disease may be both central and peripheral. Another test which may be useful is the following—it depends on the fact that the meatal hearing is normally more acute than the perosseous: place the tuning-fork on the mastoid process and let the patient say the moment when the sound ceases, then transfer the fork immediately and hold it just opposite the meatus. If the middle ear is sound, it should still be heard for a short time; but if it is not so heard, disease of the middle ear is indicated. But still the fact remains that it may be very difficult, even after careful inspection and careful testing, to eliminate the possibility of middle-ear disease, or to say, in the presence of some middle-ear disease, how

much of a given deafness is due to it, and how much to central causes.

There is a natural limit of the hearing power for notes that are very high pitched or very low pitched. This varies in different individuals, with age, and perhaps with disease. Some people cannot hear the squeak of a bat or chirp of a cricket. The range for high notes may be tested with Galton's whistle. The total range of hearing for different notes has been compared to the field of vision. Perhaps it might be more accurately compared with colour-vision. Abnormal limitation of this range, or a condition (sometimes observed) in which the patient is deaf to some intermediary notes, is usually held to be indicative of disease of the cochlea.

Defective hearing is not uncommon in tabes; but this may be often due to chronic middle-ear disease. Deafness may result from disease of the auditory nerve (it may be the seat of new growth, and atrophy of it has been observed in tabes), or from disease of the pons, or from cerebellar tumour which has spread to the fourth ventricle. In such instances there will probably be paralysis of other cranial nerve-trunks. Unilateral deafness occurs in hysterical hemianæsthesia, or in disease of the posterior extremity of the internal capsule. Deafness from disease of the cortical centres requires further clinical investigation; some cases, however, have been recorded. Deafness from nerve-disease affects, as we have said, the perosseous as well as the meatal hearing.

Tinnitus, or noises in the ear, is a symptom that may be purely nervous, or may arise from disease of the ear itself; this must be determined by the accompanying conditions.

SMELL AND TASTE.

We need say but little concerning the senses of smell and taste. In testing smell we must not

use such a substance as ammonia, the presence of which may be recognised by its irritating effects, apart from any smell proper; but rather such substances as musk, assafœtida, &c. The olfactory nerve is anatomically connected with the centre for smell (in the anterior part of the temporo-sphenoidal lobe) of the same side, but certain facts indicate that it is also connected with the centre of the opposite side. Disease of the anterior part of the temporo-sphenoidal lobe has in some cases been accompanied by fits, which began with the sensation of a smell (or taste).

In testing taste we must limit ourselves to the simple tastes of sweet, bitter, sour, salt (sugar, quinine, acids, salt). Elaborate flavours and aromas, which we are said to "taste," are really distinguished by the sense of smell. Taste may be examined electrically, the nerve-ends being readily excited by a mild constant current applied by bared wires to the moist tongue. The nerves of taste are distributed to the tongue, palate, and fauces, in two areas: (1) the sides and tip of the tongue are supplied from the lingual branch of the fifth; (2) the back of the tongue, palate, and fauces are supplied from the glosso-pharyngeal. But the course of these fibres as they run towards the mednlla is complicated. For (1) the lingual fibres pass up along the chorda tympani to the facial nerve, and run in it to the geniculate ganglion; thence along the Vidian nerve (large superficial petrosal) to the spheno-palatine ganglion, and so along the second division of the fifth to its root. Thus disease of the lingual nerve near its termination, of the chorda, or of the facial between the origin of the chorda and the geniculate ganglion, may cause loss of taste. (2) The fibres which appear to come from the glosso-pharyngeal do not accompany this nerve up to its root, for disease of the glosso-pharyngeal root does not affect taste; they may perhaps ascend *viâ* the tympanic nerve

and small superficial petrosal to the otic ganglion, and thence along the third division of the fifth nerve to its root (Gowers). It would thus seem that the whole of the fibres of taste are gathered together in the (sensory) root of the fifth nerve. The cortical centre for taste is in the same region as that for smell.

CHAPTER V.

SYMPTOMS REFERABLE TO SPECIAL DISTRICTS (FACE, TONGUE, LIMBS, &c).

ELECTRICAL EXAMINATION.

We have already had to allude to the electrical reactions of muscles; and to the present chapter, which will deal chiefly with the functions of particular muscles and nerves, we shall prefix a short account of the uses of electricity in diagnosis. **Electrical examination of muscles.**—We shall confine ourselves to its (commonest) use in testing muscles and motor nerves, the electrical examination of the special organs being a subject too difficult to deal with here. In testing either muscle or motor nerve, the effect to be observed, and from which conclusions are to be drawn, is the same for both—viz., a muscular contraction; for the condition of the motor nerve reveals itself only by its action on the muscle. This effect may be produced in two ways, either (1) by the direct application of the current to the muscle, or (2) indirectly by the application of it to the motor nerve of the muscle. In health the same result is obtained by both methods; but in disease, as we shall see, there may be modifications which show themselves only when galvanism is applied directly to the muscle.

The two kinds of current and their effects upon normal muscle.—Two kinds of current are used in testing: (1) the faradic, otherwise called the

induced or interrupted current; (2) the galvanic, also called the voltaic, or continuous, or constant current. The mere passage of a galvanic current through a normal nerve or muscle causes no stimulation, and therefore no contraction, so long as this current does not vary in strength; to elicit a muscular contraction its strength must be made to vary rapidly. This is most effectually done either by rapidly making the current (closing the circuit), or by rapidly breaking it (opening the circuit) which causes a sudden rise or fall of current strength from or to zero. There is in this respect an important difference between the two kinds of current. The faradic current is by its very nature variable in strength, consisting, as ordinarily applied, either of one rapidly appearing and disappearing current (single induction shock), or of a succession of such momentary currents. Its effect, in health, is to produce a single sharp muscular twitch when a single current is passed, a tetanic contraction of muscle when a series of such currents is passed. The galvanic current, on the other hand (viz., that obtained directly from the poles of a series of galvanic cells), when once established through the body, flows equably and permanently. During the flow of such an equable current effects indeed are produced, but no stimulation such as can cause muscular contraction. When, however, a current of sufficient strength is either made or broken, a sharp single muscular contraction follows, just as in the case of the single induction (faradic) current. And this contraction is, as we have said, the same, whether the application of the current be to the muscle directly or to its motor nerve-trunk. Stimulation even more effective than that caused by simply making or breaking such a current may be obtained by rapidly reversing its direction (voltaic alternative).*

Note on Apparatus.—A *faradic battery* consists of the following parts:—One or two good-sized cells, the current from which circulates through a coil of (comparatively) stout wire,

Localisation of current.—But before considering this contraction further, we must explain in what sense a current which in reality circulates through

called the primary coil. Encircling this coil, but not in connection with it, is placed another coil of finer wire, called the secondary coil. The circulation of the current in the primary coil, when flowing equably, produces no effect in the secondary; but when the primary current is rapidly made or broken, a current of considerable electro-motive force but of extremely short duration appears in the secondary coil. This is the current which supplies the "single induction shock" and causes a single muscle-twitch. An automatic hammer (which we need not describe fully) is attached to the apparatus, by which the current in the primary coil is continuously made and broken, and thus a series of induced currents (or as it is shortly called, a faradic current) is set up in the secondary coil. This series of shocks produces tetanus of the muscle. The strength of this current is graduated either by sliding the secondary coil more or less over the primary, or by sliding between the coils a metal tube which serves to shield one from the other. The distinction of poles, so important to observe in the galvanic apparatus, is of less consequence in the faradic. In most batteries provision is made for using either the currents in the secondary coil or those in the primary. The secondary currents are as a whole the strongest and the most acutely felt, and most suitable for stimulating the skin and its sensory nerves; the primary are milder, and perhaps the best fitted for eliciting contraction of deeper-seated muscle.

A *galvanic battery* consists of a number (about 40) of cells connected in series—*i.e.*, the negative pole (zinc) of one connected to the positive pole (carbon) of the next. The object of this is to provide a sufficient electro-motive force to overcome the resistance of the skin, which is very large. The current obtained will be directly proportional to the electro-motive force (*i.e.*, roughly speaking to the number of the cells used), and inversely proportional to the resistance. Since this resistance varies according as the skin is moist or dry, or according to the part of the body tested, it evidently contributes a very important factor towards the strength of the current, quite as important as the number of cells used. Thus if 20 cells, each giving $1\frac{1}{2}$ volt electro-motive force, are applied through a part where the resistance is comparatively high, say 3000 ohms, then the current strength will be $\frac{30}{3000}$ amperes or 10 mille-amperes, a strong but bearable current. The same cells applied through a part when the resistance is low, such as the face, would give, reckoning the resistance as

the whole body can be said to be applied to a particular part of it, such as a muscle or nerve. The explanation will apply to either current, faradic or galvanic. From the two poles of the battery the current is conducted by insulated wires, called rheophores, and thence into the body by means of metal plates or discs, termed electrodes, which are covered with wash-leather and well moistened with warm water, so that their moisture may reduce the resistance of the skin. The current enters the body at the point of application of one electrode, then spreads itself out over the internal tissues, and is again gathered up at the point of application of the other electrode, where it leaves the body (Fig. 46). The two electrodes therefore constitute narrow necks in the channel of the current, where it becomes concentrated. This concentration, or "density," depends on two things: first, the absolute strength of the current used, and, secondly, the smallness of its channel at the particular point in question (in other words, the

1000 ohms, a current of 30 mille-amperes, unbearable to most patients. The galvanic current is usually graduated by a dial or sledge which admits of any number of cells being thrown into circuit, and thus varying the *electro-motive force* used. (It may also be graduated by a contrivance called a rheostat for varying the *resistance* in the circuit.) The terminals of the battery (*i.e.*, the screws to which the rheophores are to be attached) should be marked so that it can be seen at once which corresponds to the negative and which to the positive pole. There should be also a handle (commutator) whereby the current can be made or broken, or its direction reversed (poles changed) without removing the electrodes from the patient. Further, there should be a galvanometer graduated in mille-amperes for measuring the current *during its passage;* and the needle thereof should come to rest quickly (dead-beat) in order to save the time of the operator and the skin of the patient. Without a galvanometer the current strength can only be guessed at, or measured by its effects, which is obviously illogical when its effects are what we wish to test. Rheophores and electrodes of proper sizes, and an interrupting handle by which the make and break of the current are under the control of one finger of the operator, complete the requisites for electro-diagnosis.

density is directly proportional to the current strength and inversely proportional to the area of its section). The effect of the current depends on its density; and

FIG. 46.

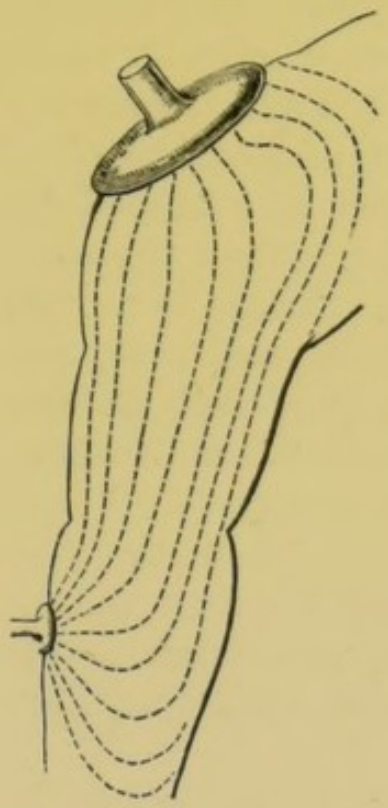


Diagram to illustrate the concentration of the current under the point of application of the smaller electrode.

it can thus for practical purposes be localised at any sufficiently superficial point by using a moderately small electrode.

This fact is utilised in what is called the "unipolar method" of testing as follows:—One of the electrodes, of large size, and therefore giving but a slight concentration of current, is applied at any point, such as the nape of the neck, sternum, buttocks, &c., sufficiently far from the part we wish to test. This is called the "indifferent" electrode. The other or "different" electrode is of small size, so as to concentrate the current, and is applied over the part to be tested. The current is thus concentrated to a maximum at this point, and is said to be localised there.

EFFECTS OF DIFFERENT POLES.

Normal polar formula.—The next question, that of the different action of the two poles, is of importance only with reference to the galvanic current, and may practically be neglected with respect to faradism. That electrode which is connected to the negative pole (or zinc element) of the battery is called the kathode (and designated by the symbol K); the other one which is attached to the positive pole (carbon element) is called the anode (and designated by the symbol A). There is a difference in the readiness of the muscular response, according as (1) the kathode or anode is placed on the part to be tested, (2) the current is made or broken. Thus, if we take a healthy nerve or muscle, and ascertain by gradually raising the current strength what are the minimum strengths at which we can obtain contractions with kathode or anode, and with make and break respectively, we shall find that they fall into the following order.

(1) To obtain a contraction with the least current strength, the kathode must be placed on the muscle or nerve, and the current made (circuit closed). This is designated in symbols as follows:

K.	C.	C.
(Kathode).	(Closure).	(Contraction).

or German—

K.	S.	Z.
(Kathode).	(Schliessung).	(Zückung).

(2) With a greater current strength, a contraction may be obtained when the anode is on the muscle or nerve, and the current is made (circuit closed) ACC or German ASZ. If the kathode be substituted for the anode with this current strength, its action is greater than that of the anode, KCC > ACC.

(3) With a still greater current strength, the anode causes a contraction when the current is broken

(circuit opened); AOC, German AOZ. (This is the general rule; but sometimes even with healthy tissues AOC occurs before ACC.)

(4) Lastly, with a current strength which is to most patients unbearable, the kathode causes contraction when the current is broken (circuit opened); KOC, German KOZ.

The usual "polar formula" therefore is—

KCC > ACC > AOC > KOC,

K or A indicating the character of the "different" electrode; and > that the contraction occurs at a lower current strength, or is greater when produced by the same strength. But there may be, as just stated, a little uncertainty as to the order in which the anodal closing and opening contractions follow each other. (A very slight contraction may be indicated by a small c or z.)

It should further be noted, that in health the character of the muscular contraction is a sharp, short twitch, quickly evoked and quickly subsiding.

Motor points.—If an electrode of moderate size be passed over a muscle, it will usually be found that there is one point in the skin at which the current most readily calls forth contraction. This is called the "motor point" of the muscle, and in testing the contractility of the muscles to weak currents (at any rate when they still respond to faradism), we should remember the existence of such points. Maps* of the motor points have been prepared, but unfortunately they differ considerably in different individuals.

Abnormalities in electrical reaction.—The deviations from the normal electrical reactions, which are observed in disease, are of two main kinds:

(1) Quantitative—*i.e.*, simple excess or defect in readiness of response to the electrical stimulus.

(2) Qualitative—*i.e.*, changes either in the character of the muscular contraction or in the normal polar formula.

* *Vide* figures at the end of the book.

Quantitative variations in electro-irritability are usually in the way of defect, but excess of electro-irritability is said to occur in tetany, and some other conditions. Diminution occurs in that form of muscular atrophy wherein the disease falls primarily on the muscular tissue, and not on the nerves or nerve-centres; in muscular atrophy that is secondary to joint-disease, and sometimes in peripheral neuritis. We should not too hastily conclude that the electro-contractility of a muscle is diminished because it appears to react badly. A certain allowance must be made for differences between different patients, and between muscles in different parts of the body. Therefore it is well, wherever practicable, to contrast the reaction of a muscle with that of its fellow on the opposite side. Again, the apparent defect in reaction may be really due to defect in the current used; either because the electro-motive force of the battery is insufficient, or because the resistance in the skin of the part is unusually high. This fallacy may be avoided by the use of the galvanometer.

Qualitative changes are, as a rule, elicited by galvanism only, and by the direct application of galvanism to the muscle. Qualitative changes are evidenced as follows:—**Reaction of degeneration.**—The character of the muscular twitch alters; it becomes sluggish, developing and subsiding slowly. The polar formula is modified, and usually in this sense, that the current strength required to produce contraction with anodal closure is not greater, or is even less, than that required with kathodal closure (ACC = or > KCC). Such changes constitute what is called the reaction of degeneration (RD, or German EaR). When they occur, we generally find that at the same time there is loss or great diminution of contractility to currents applied in the other way—namely, to faradism, whether applied to the nerve or muscle, or to galvanism applied to the nerve.* In this case, the reaction of degenera-

* The electro-contractility of muscle is probably of two kinds—the neuro-muscular, which is produced by the inter-

tion is said to be "complete." If, as occasionally happens, qualitative changes are present without loss of farado-contractility, the reaction of degeneration is "incomplete."

Meaning of the reaction of degeneration.—The main inference to be drawn from the presence of the reaction of degeneration is this: that a degenerative process has been set up in the muscle consequent on some interference with the trophic influences which are normally exercised upon it by the cells of the anterior grey cornua of the cord. Either the cells themselves have been injured, or the nerve has suffered in some part of its course between the cells and the muscle.* Reaction of degeneration is therefore the clinical concomitant of Wallerian degeneration (*vide* p. 66). Along with it are generally to be found paralysis and wasting of the muscle, but the three factors may be present in very unequal degrees.

The phenomenon is best watched where the lesion is acute, say in an ordinary case of facial paralysis (acute neuritis of the facial trunk). For the first few days there is no change in electro-irritability. Then the response to faradism, whether applied to the nerve-trunk or to the muscles, and the response to galvanism as applied to the nerve-trunk (*i.e.*, the neuro-muscular electro-contractility), begins to fail, and rapidly disappears altogether. *Per contra*, the response to galvanism, as applied to the muscle (idio-muscular electro-contractility) is increased, and with this increase appear the characteristic phenomena of RD, which

vention of nervous influence, this influence being evoked by the application of faradism to the nerve-trunk or nerve-ending, or of galvanism to the nerve-trunk; and the idio-muscular, produced by the application of galvanism to the muscle. When the neural influences are removed, the idio-muscular phenomena can be studied by themselves.

* As a rule, therefore, RD enables us to localise the lesion, though it is true that in some diseases, such as lead paralysis, wherein it occurs we cannot confidently assert this. Some recent German observations indicate that a modified RD may be present in cerebral lesions: but these observations require further confirmation.

we have already described. Roughly speaking, about a week is required for the development of these changes. After a week or two more, if the disease is mild and transitory, the increased galvano-contractility falls again, the motor paralysis begins to disappear, the farado-contractility reappears, and the qualitative changes return to normal. If the case is more severe, the galvano-contractility falls even below normal, but neither the motor power nor the farado-contractility return, and the qualitative changes persist. In the worst cases of all this state of affairs goes on till the galvano-contractility also disappears, so that the muscle is left paralysed, wasted, and without electro-contractility of either kind.

When the disease is not acute, but insidious in onset and spreads gradually, the phenomena are less regularly developed. The stage of increase in the galvano-contractility may be absent. The failure of farado-contractility in some fibres may be masked by reason of its retention in neighbouring fibres, and care may be required to demonstrate the qualitative changes. Probably these will become evident enough as the disease progresses. Just as in acute lesions the motor power returns before the electrical abnormalities disappear; so in chronic and widespread disorders we may find electrical changes in muscles which were not known to be paralysed; for the presence of RD indicates, as we have already said, a defect of nutrition rather than a paralysing lesion, though the latter is usually associated with it.

THE FACE.

Paralysis from disease of facial nerve-trunk.—Facial paralysis, when due to disease of the facial nerve-trunk, is almost always unilateral. It involves all the muscles of one side, those of the forehead and the orbicularis oculi, as well as the lower facial muscles; this distinguishes it from the facial paralysis caused by disease of one cerebral hemisphere, wherein the muscles of the forehead and

the orbicularis oculi for the most part escape. The affected side of the face is motionless and expressionless, the naso-labial fold is less marked than on the sound side, the angle of the mouth generally droops. The features may be distorted, being pulled over by the muscles of the sound side. The paralysis, if not obvious when the face is at rest, becomes so when the muscles are thrown into action. Thus, on showing the teeth, the mouth is drawn over to the sound side. On shutting the eyes, the lids of the affected side remain open, while the eyeball rolls upwards (concealing the cornea perhaps, but not the whole sclerotic). Owing to continual want of apposition of the lower lid, the tears are apt to run from the affected eye. On attempting to whistle, the air escapes from the paralysed corner of the mouth. The paralysed buccinator bulges when the mouth is inflated; and during mastication it lets the food slip between the gums and the cheek. If the muscles be tested electrically, reaction of degeneration will be found (in all but the mildest cases) after the first week of the disease.

There are two common causes for such a facial paralysis: (1) exposure to cold, as from sitting at an open railway-carriage window, sleeping in a draught, &c.; this is called "rheumatic neuritis."

(2) Suppurative disease of the middle ear, especially when accompanied with bone-disease. The course of the nerve through the inner wall of the tympanum renders it obvious to inflammatory change from this source. Further, syphilis is alleged to be a cause of facial neuritis, and facial paralysis may be associated with paralysis of other cranial nerves in tumours, injuries and disease of bones, &c., affecting the base of the skull.

The onset of a facial paralysis is generally acute. At the commencement of a "rheumatic" case there may be pain in the side of the face; this is said to be due to a coincident affection of the trigeminus. In a mild case the paralysis may subside in the course of a few weeks. Electrical examination may prove useful

in prognosis; for if (in a case known to be due to disease of the nerve-trunk) the electrical reactions are normal after a week or fortnight, the case is a mild one, and will probably recover soon. But severer cases may last for months, and some may prove quite irremediable. In long-standing cases the muscles degenerate and shorten, so that the face appears drawn to the *paralysed* side. But this is only when at rest; on making the patient shut his eyes, &c., it will be quite obvious which side is paralysed.

In the early stages, counter-irritation, in the shape of a blister, may be applied over the nerve-trunk. Iodide of potassium may be administered. The ear should always be examined, and treated if active disease exist in it. After the first ten days, electricity may be applied to the muscles; galvanism is usually required, because they no longer act to faradism. The eye, being unprotected by closure of the lids, is apt to become inflamed, and therefore it should be carefully protected from wind and dust.

In some cases of facial paralysis there is loss of taste on the corresponding side of the tongue. This means either (1) that the facial nerve-trunk is diseased in that part of its course where it contains the fibres from the chorda tympani—*i.e.*, between the geniculate ganglion above and the origin of the chorda (at the lower part of the aqueductus Fallopii) below); or (2) that the chorda tympani is involved simultaneously, as may well happen in disease of the ear.

Again, it has been stated that in disease of the facial nerve *above* the geniculate ganglion the corresponding half of the soft palate and uvula is paralysed, since the motor fibres to those parts run *viâ* the facial root and geniculate ganglion, through the large superficial petrosal nerve to Meckel's ganglion, which latter ganglion supplies the soft palate. But the clinical fact appears to be doubtful.

Double facial paralysis (from disease of the nerve-

trunks) is a rare occurrence. Some constitutional cause (such as syphilis) is here more likely than in unilateral disease. Bilateral ear-disease may also cause it. The whole face is like a mask; the patient may weep bitterly, or be convulsed with laughter, but his facial muscles betray no emotion.

Other causes of facial paralysis.—Facial paralysis may be caused not by disease of the nerve-trunk, but by a central lesion, affecting either (1) the nerve-nucleus in the pons, or (2) the parts above this —viz., the cerebral cortex or motor tract between the cortex and nerve-nucleus. In (1) "nuclear" paralysis, the facial palsy is probably more limited than in disease of the nerve-trunk.* And there may be further symptoms indicating disease of the pons, either paralysis of other cranial nerves or cross paralysis.

In (2) "supra-nuclear" paralysis we look for traces of hemiplegia on the same side; but even in the absence of this we may distinguish as follows. Facial paralysis, when the lesion is in the cerebral hemisphere, does not affect the orbicularis oculi, or occipito-frontalis, so that the patient can still frown and close his eye. Secondly, the electrical reactions in such a case are not altered.

Lastly, the facial muscles may be affected in some forms of progressive muscular atrophy (infantile myopathy), but such affection is bilateral.

Facial spasm.—Muscular spasm in the district of the facial nerve (motor tic) is far less common than paralysis. It mostly occurs as paroxysms of clonic spasm, affecting more particularly the orbicularis oculi and zygomatici. Unlike facial palsy, it begins gradually; it may last a very long time. It may be

* It seems probable, from the facts of glosso-labio-laryngeal palsy (*vide infra*, p. 155) that the motor supply of the lips comes from the hypoglossal nucleus, and not from the facial. It is also thought that the orbicularis oculi is innervated from the third nucleus. Thus the facial nucleus proper would innervate only the lower part of the face, and that with the exception of the orbicularis oris.

due to direct irritation of the facial trunk (from tumour, aneurysm, or the like), or reflexly to irritation of the trigeminus (decayed teeth, &c.), or to disease of the cerebral cortex. Such conditions as mental anxiety, grief, &c., often precede it.

Facial neuralgia.—The sensory nerve-supply of the head and face is represented in Fig. 47. The face, with the exception of the skin near the parotid and lower jaw, is supplied by the three branches of the fifth nerve; the back of the head by the great occi-

FIG. 47.

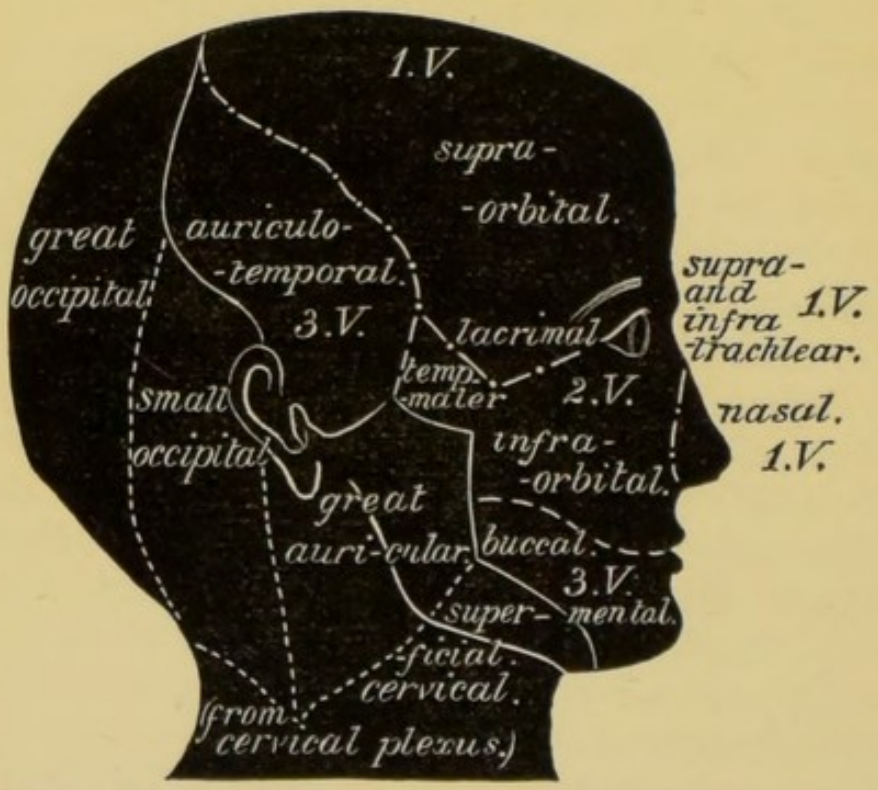


Diagram of the cutaneous nerve-supply of the face and head. (After Flower.)

pital (posterior division of second cervical nerve), the remaining parts from the cervical plexus (small occipital, great auricular, and superficial cervical nerves). The most important sensory affection of these parts is facial neuralgia. Evidently the fifth nerve is specially obnoxious to two common causes of neuralgia, (1) exposure to cold, (2) peripheral irritation, since it is the sensory nerve of the teeth. Again, since it passes through bony foramina, it may be easily affected by

swelling of its sheath, or of the periosteum in its neighbourhood. Malarial neuralgia is a form which specially affects the supra-orbital division; it is known as brow-ague. The so-called epileptiform neuralgia is characterised by the extreme suddenness and violence of the pain. The points of emergence of the fifth nerve upon the face should be noted. The pains are apt to radiate from them, and they are often specially tender to pressure.

"Cervico-occipital" neuralgia is less common than facial. Both this region and that of the face may be selected by the lightning pains of tabes dorsalis.

Hemiatrophy of face.—The curious disease known as "facial hemiatrophy" is possibly due to disease of the fifth nerve. In this disease the skin, subcutaneous tissue, hair, bones, in short all the tissues except the muscles, waste gradually, but only upon one side, so that the patient's face appears to be made up of two dissimilar halves, one smaller than the other and wizened-looking. Sometimes a thin furrow divides the two. There is, however, no affection of sensibility, and the electrical reactions of the muscles are normal. Sometimes there is wasting of the skin or bones of other parts of the body.

THE TONGUE.

Paralysis and atrophy of the tongue.—The motor nerve of the tongue is the hypoglossal; the lingual branch of the fifth supplies it with common sensibility; of the sense of taste we have already spoken.

Paralysis of the tongue, like that of the face, may result from disease of the motor nerve-trunk, of its nucleus, or of the parts above. Paralysis of one-half the tongue usually indicates disease of the nerve-trunk, or of the opposite cerebral hemisphere; in the first case the paralysis is accompanied by wasting, in the second case it is not. In either case the tongue, when protruded, deviates towards the *paralysed* side; since the tongue is under normal conditions pushed

out, and in unilateral paralysis the unbalanced action of the healthy muscles push it over. Such unilateral paralysis interferes little with the functions of the tongue. Bilateral paralysis is usually nuclear in origin. In such a case the tongue wastes, becoming thin, flabby, and wrinkled longitudinally. It can only be protruded imperfectly, or not at all. Difficulty in articulation and, still more, difficulty in swallowing are the chief symptoms. Such wasting palsy of the tongue commonly forms part of a more general muscular atrophy (progressive bulbar paralysis), but it may be the earliest symptom thereof. With it may be associated paralysis of the lips, palate, and larynx.

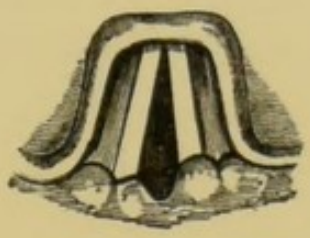
It would appear that in exceptional instances unilateral palsy of the tongue may be due to nuclear disease; such appears to be the case when hemiatrophy of the tongue occurs in tabes dorsalis, or in general paralysis. Conversely, bilateral palsy may be due to disease of the hemispheres, either when the cerebral disease is bilateral, or sometimes even when it is unilateral.

The palate.—The natural movement of the palate is best seen by making the patient open his mouth, and say "ah" or "eh," drawing in his breath the while. (The tongue is to be kept depressed by a spatula, if necessary.) The palate is then drawn upwards in the form of a symmetrical arch. If the whole palate is paralysed, the whole of it hangs motionless. If one side is paralysed, the palate moves, but not symmetrically. The upward traction takes place from the centre of the sound side, and therefore causes the sound side to rise highest, and at the same time to become "dimpled." Palatal paralysis is common as a sequela of diphtheria; the whole palate is affected, the voice becomes nasal, fluids while being swallowed are apt to regurgitate through the nose. It occurs also in progressive bulbar paralysis. In this disease, as above remarked, there is engrafted upon paralysis of the tongue paralysis of the lips, palate,

and larynx. That of the tongue, and probably of the lips, is caused by disease of the hypoglossal nuclei; that of the larynx, and probably of the palate, to disease of the adjacent spinal accessory nucleus. Similarly, unilateral paralysis of the tongue, palate, and larynx has been seen in disease involving the roots of these two nerves upon one side of the medulla.

Paralysis of the larynx.—The intrinsic muscles of the larynx are supplied by the recurrent laryngeal nerve. This nerve, though it branches from the vagus, is probably ultimately derived from the spinal accessory. For clinical purposes, we may divide the muscles of the larynx into two groups: (1) adductors (and tensors), which approximate the two vocal cords, and which are necessary for phonation; (2) abductors, which separate the cords, and therefore are necessary for respiration. When one vocal cord is paralysed completely (*i.e.*, both as regards abduction and adduction) (Fig. 48), it stands on a slightly different level to its comrade, and midway between the

FIG. 48.

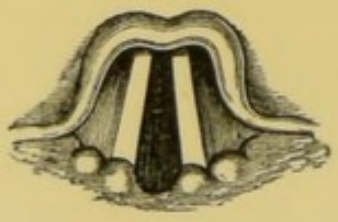


Complete paralysis of left vocal cord (paralysis of recurrent laryngeal nerve), position of cords during inspiration. (After Ziemmsen.)

positions of complete abduction and complete adduction. This is called the cadaveric position, since it is that which the cords assume post-mortem. During deep inspiration it is not abducted, nay, the entering current of air may even make it flap inwards slightly. During phonation it is not adducted, but the sound cord may cross the middle line to meet it, thereby rendering phonation just possible. But the voice is

apt to be hoarse, and the cough ineffective. Such a condition points to disease of one recurrent laryngeal

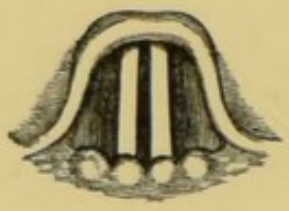
FIG. 49.



Complete paralysis of both vocal cords (cadaveric position of cords.) (After Ziemmsen.)

nerve, the ultimate source of which is often to be found within the chest—viz., pressure upon or

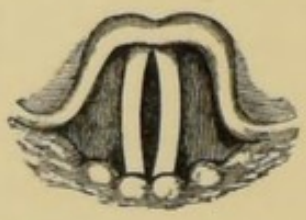
FIG. 50.



Bilateral paralysis of posterior crico-arytænoids (abductor paralysis); position of cords during inspiration. (After Ziemmsen.)

destruction of the nerve from a new growth, or aneurysm. An aneurysm is particularly probable if

FIG. 51.



Bilateral paralysis of internal thyro-arytænoids ; position of cords during phonation. (After Ziemmsen.) The usual condition in hysterical aphonia).

the paralysis is left-sided ; because the left recurrent nerve winds round the arch of the aorta.

Complete paralysis of both cords (Fig. 49) is a very rare condition: here the voice is whispering, coughing is impossible; respiration is normal, save that the patient may be unable to husband his breath sufficiently to say a long sentence or make a prolonged effort without taking breath.

Abductor paralysis (Fig. 50) is bilateral as a rule. The two cords stand near the middle line; during inspiration they do not move outwards (they may even flap inwards a little), but in phonation they act normally. The voice is unaffected, the cough is natural. As to respiration, in mild cases little more may be noticed than that inspiration is long and wheezy. But should any further source of obstruction arise, such as a laryngeal catarrh, or should the paralysis grow rapidly worse, the aperture of the glottis becomes inadequate, inspiratory dyspnœa arises, and tracheotomy is required. Such bilateral abductor paralysis may occur in various degenerative nervous diseases; particularly in tabes dorsalis, wherein it may be an early symptom. It is a remarkable fact that when any organic disease progressively invades the nervous apparatus of the larynx, whether in the nerve-trunks or the nerve-centres, the first result is *abductor* paralysis; the cords gradually approximate to the middle line, and not to the cadaveric position.

Adductor paralysis (Fig. 50) is also bilateral. The cords during attempts at phonation either meet imperfectly or are insufficiently tense, but in inspiration they move outwards normally. The voice is whispering, though the cough is (often) natural; the breathing is natural. This condition occurs without known organic cause, and constitutes what is known as "hysterical aphonia."

Laryngeal spasm is chiefly seen in the "laryngismus stridulus" of rickety children.

Laryngeal anæsthesia we shall not consider here. For its investigation much dexterity with the mirror and sound is necessary.

Anæsthesia of the fauces, or (more strictly) abolition of the reflex caused by irritation of the fauces, is usually hysterical.

Respiratory muscles.—Paralysis of the muscles of respiration may be considered here. The diaphragm is the principal inspiratory muscle. Its action, according to Duchenne, is twofold. *First*, it draws up the lower ribs to which it is attached, and thereby expands the lower part of the chest. This it can only do by acting on the convex and partially resisting surface supplied to it by the liver and spleen. (The resistance offered by these organs is conditional on the support which they receive from the muscles of the anterior abdominal wall. Hence, when the abdominal muscles are paralysed, the diaphragm can no longer raise the lower ribs.) *Secondly*, it depresses the floor of the thorax, thereby increasing the chest capacity. Its contraction makes the floor flat instead of dome-like, and drives before it the abdominal viscera. The visible effect of this is distension of the epigastrium during inspiration.

In paralysis of the diaphragm the expansion of the lower part of the chest is deficient, as compared with that of the upper, which is worked by the intercostals; and the epigastrium, instead of rising during inspiration, tends to be sucked in. Paralysis of the diaphragm may be caused by disease of the cord above the fourth cervical root, from which the phrenic nerve mainly originates (in this case the intercostals will also be paralysed); from disease of bone or meninges at the level of the fourth cervical root; from disease of the phrenic nerves themselves, which may occur in alcoholic neuritis, lead poisoning, diphtheritic paralysis, or from aneurysm or intra-thoracic growth; in muscular atrophy spreading to the diaphragm. Inaction of the diaphragm may be seen in some cases of pericarditis or pleurisy.

As to the intercostal muscles, the bulk at any rate of them serve as muscles of inspiration. The move-

ment of inspiration as performed by the intercostals only consists mainly of expansion of the upper part of the chest (superior costal type of respiration). The action of the intercostals without that of the diaphragm may suffice for quiet respiration, but should effort be required, or should respiration be embarrassed by the advent of any lung affection, dyspnœa sets in. Still more critical is the condition of the patient if both intercostals and diaphragm have failed, and respiration be left to the extraordinary muscles of inspiration, sterno-mastoids, trapezii, serrati, &c.

Expiration is usually performed by the elastic recoil of the lungs ; but for powerful expiratory efforts, as in cough, sneezing, &c., the muscles of the anterior abdominal wall are required, and their paralysis will render such actions impossible.

PERIPHERAL AFFECTIONS OF LIMBS AND TRUNK.

As the distribution of paralysis, anæsthesia, &c., in the body generally is of importance, so also is their grouping within particular parts. It is particularly important in the limbs. Say that a patient comes complaining of inability to perform certain movements ; we have to determine whether this indicates paralysis of a particular muscle or muscles ; whether the combination of muscles affected corresponds to any particular nerve-trunk, or whether it points to central disease.

To fix on the muscles at fault, we must evidently have some knowledge of the muscular mechanism of movements ; though of course we may be helped in many instances by the appearance of muscular wasting, and by the results of electrical examination.

We shall therefore consider here the method by which the various movements of the limbs are produced in health, taking as the basis of our account the writings of Duchenne ("Physiologie des Mouvements").

Muscular mechanism of upper limb.—The movements of the upper arm are effected partly at the shoulder-joint, partly by movements of the scapula.

Elevation of the arm, when hanging by the side, is effected by the deltoid. Contracting as a whole this muscle lifts the arm upwards and outwards to the horizontal position. But for this, support of the scapula is necessary; such support is mainly given by the serratus magnus, which begins to contract in association with the deltoid. Contraction of the anterior, middle, or posterior parts of the deltoid carry the raised arm forwards, straight outwards, or backwards respectively; and their successive action will give it a horizontal sweep as in swimming. In the forward movement, the clavicular part of the pectoralis major associates itself with the anterior part of the deltoid. The supra-spinatus co-operates, though feebly, with the deltoid in raising the arm. The posterior part of the deltoid can only lift the arm to an angle of 45° with the trunk, and no part of the muscle beyond an angle of 90° (horizontal position). All further elevation, as in the action of lifting the arm above the head, is effected by an upward tilting of the acromial end of the scapula. Continued action of the serratus magnus, assisted by the upper and outer fibres of the trapezius, produces this tilting movement. Paralysis of the deltoid is easily recognised: the patient, in endeavouring to raise his arm, lifts the point of his shoulder and inclines his trunk to the opposite side, but still he cannot separate the arm from the side, nor maintain it in the raised position when raised for him. Atrophy of the muscle produces flattening under the acromion instead of the rounded sweep of muscle normally seen there. Paralysis may attack the different parts of the muscle separately; that of the anterior fibres causes difficulty in bringing the hand to the mouth, taking the hat off, &c.; that of the posterior part, difficulty in buttoning the braces

behind, in putting the hands into the trouser-pockets, &c. Paralysis of the middle fibres may be imperfectly compensated for by combined action of the other two parts.

Depression of the raised arm may be effected by the weight of the limb alone, but when force is required the pectoralis major and latissimus dorsi are used. The pectoralis major acting as a whole brings the arm downwards and forwards; after the arm has reached the horizonital position the sternal fibres still continue to depress it, and finally bring it into apposition with the front of the chest. A forcible downward sweep, as in delivery of a sword-cut, is thus produced. But the clavicular fibres, after the arm has reached the horizontal, no longer depress it, but carry it forward to the middle line. The strength of these clavicular fibres may be tested by making the patient press together the hands when they are outstretched horizontally in front of him; that of the sternal fibres by making him press them together when they are at a lower level. When the arms hang by the sides, the clavicular fibres act as follows: they raise the points of the shoulders and bring them round towards the front of the chest, producing the position assumed in crouching, shivering, &c. The latissimus dorsi also depresses the arm, but simultaneously carries it backwards. In so doing it lowers the point of the shoulder and carries the whole scapula backwards towards the spine. Both muscles acting together from the small of the back extend the upper part of the spine upon the lower, and this with the backward and downward movement of the scapulæ produces a military carriage. Another muscle—viz., the teres major—depresses the arm and carries it backwards; but this it can only do when the scapula is fixed (by the rhomboids). When the arm is fixed (as by approximation to the side of the trunk) the teres major, using it as a fixed point, shrugs the shoulder by dragging up the scapula and tilting upwards its acromial angle.

All the depressors of the arm, when the raised arm is made their fixed point, tend to drag the body upwards; and they are thus the muscles used in climbing.

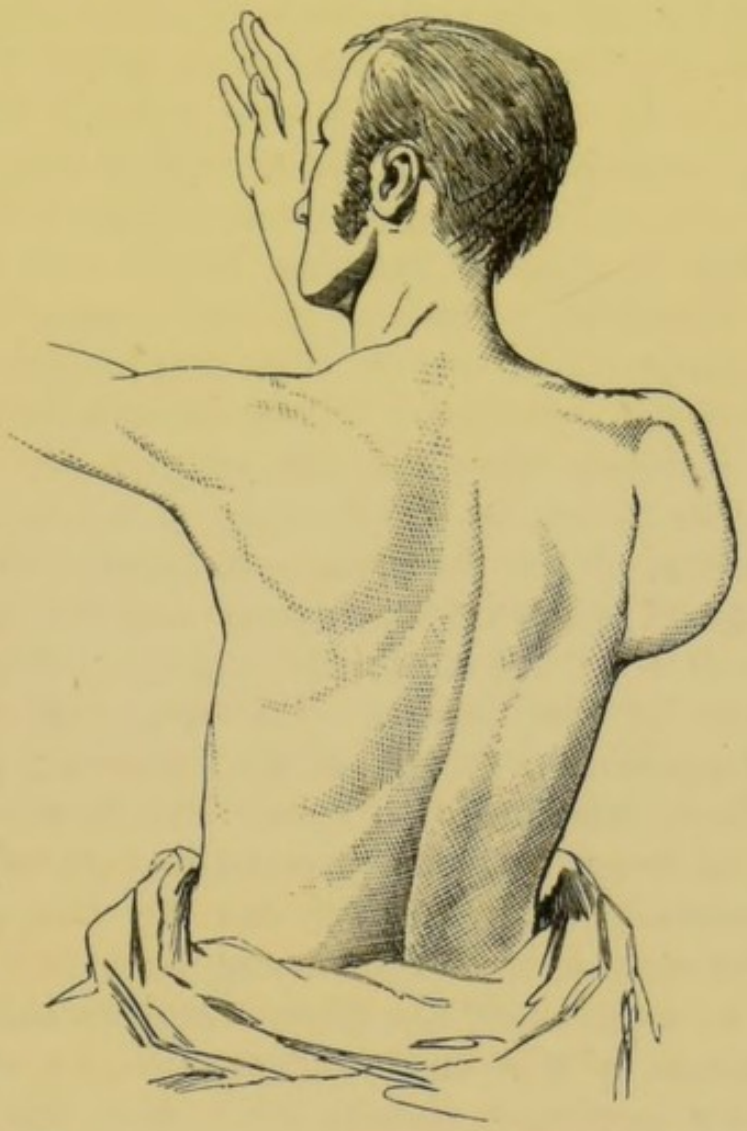
Rotation of the arm in an inward direction is effected by the subscapularis; in an outward, by the infra-spinatus and teres minor. These rotatory movements, according to Duchenne, may supplement the movements of pronation and supination in the fore-arm. The same author notes the curious fact that paralysis of the infra-spinatus causes difficulty in writing; the hand can no longer be carried, by external rotation of the shoulder-joint, horizontally along the paper from left to right.

Certain muscles appear to act as ligaments of the shoulder-joint; these are the supra-spinatus, long head of the biceps, of the triceps, and the coraco-brachialis. We may add the muscular fibres contained in the capsule itself.

Movements of the scapula, which, as we have said, are intimately associated with movements at the shoulder-joint, are of two kinds—viz., a sliding of the whole bone over the surface of the chest, or tilting (rotatory) movements which alter the relative level of its three angles. The forces which mainly serve to hold the bone in balance are, the trapezius, the serratus magnus, the rhomboids, and levator anguli, the weight of the upper limb, and the action of its depressor muscles. The trapezius, at any rate those fibres which run downwards and outwards to the acromial angle of the scapula, tilt this angle upwards, thereby giving a contemptuous shrug to the shoulder; the other fibres mainly draw the scapula backwards towards the spine. Forcible lifting of the shoulder, as in carrying a burden upon it, is effected, as we have already said, by the serratus magnus, trapezius, teres major, clavicular part of pectoralis major. Weakness of the trapezii allows the shoulder to droop downwards and forwards, giving the patient a long-

necked, round-shouldered appearance. When the trapezius and serratus are both paralysed, the point of the shoulder drops markedly, while the lower angle rises and approaches the spine; the bone appears as if it were suspended by its inner angle (to which the levator anguli is attached). The serratus magnus not

FIG. 52.



Paralysis of right serratus magnus. (From Duchenne.)

only lifts the acromial angle, but moves the whole scapula round the chest away from the spine towards the front, at the same time keeping the vertebral border closely applied to the chest. It is used in forcible forward extension of the arm, as in striking out from the shoulder, or pushing a heavy weight in

front of one. Paralysis of this muscle (at any rate when the deltoid is intact) is characterised thus (Fig. 52): If the patient extends his arm before him, the vertebral border of the scapula becomes separated from the chest, and projects like a wing. The apex of the wing is formed by the lower angle tilted upwards and away from the chest. The rhomboids draw the scapula towards the spine, lifting it upwards, but tilting it in the opposite way to the serratus and trapezius—viz., the acromion downwards, the lower angle upwards and towards the spine. Combined with the trapezius and levator anguli they lift the scapulæ upwards and carry them towards the spine, the position is that of tugging backwards against a weight. Since they tend to move the scapulæ in the opposite sense to the serrati, they are used to fix these bones when the serrati act on the chest in forced inspiration. Further, they fix the scapula for the teres major, when it acts as a depressor of the arm.

Movements of the forearm.—Extension is effected by the lateral heads of the triceps and the anconeus: simple flexion by the brachialis anticus; flexion with supination by the biceps. Further, the supinator longus is a powerful flexor of the forearm; its action as regards supination is to bring the forearm into a position midway between pronation and supination. Simple supination is carried out by the supinator brevis: pronation by the pronator teres and pronator quadratus, assisted by the flexor carpi radialis.

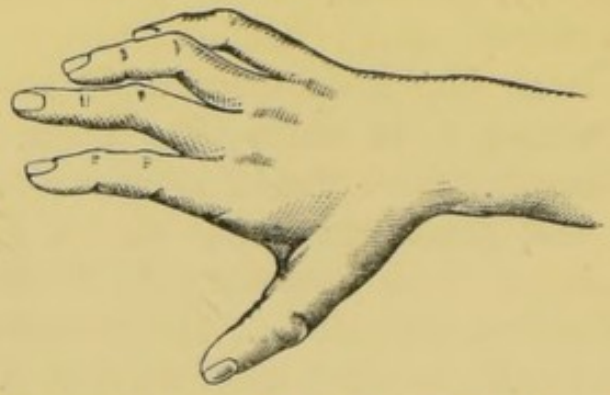
Movements of the wrist.—The extensor carpi radialis brevior produces simple extension, the extensor carpi radialis longior extension with abduction, the extensor carpi ulnaris extension with adduction. These lateral movements of abduction and adduction are most free when the wrist is extended, and they are produced by the two lateral extensors. As to flexion, the palmaris longus flexes the wrist simply; the flexor carpi radialis flexes with slight pronation; the flexor carpi ulnaris flexes the wrist, at the same time turning

the uluar border of the hand inwards towards the palm (as in the action of a violinist reaching round to touch the higher parts of the strings with the little and third fingers). These flexors do not, like the extensors, produce lateral movements of the carpus.

Movements of fingers.—The extensor communis digitorum extends first the metacarpo-phalangeal joints, and next the wrist-joint; it also gives the fingers a movement of abduction from the middle line of the hand. The flexor profundus flexes the middle and last phalanges; the flexor sublimis the middle phalanges; and this they do most powerfully when the wrist is in extension. The interossei, assisted by the lumbricals, abduct and adduct the fingers; but they have also a more important action, viz., to flex the metacarpo-phalangeal joints, and at the same time extend the phalangeal joints. They are required for many delicate movements of the fingers, and particularly that of writing. Like the long flexors, they require for their proper action a certain amount of extension at the wrist-joint. It may be noticed that between the opposed movements of extension and flexion in the wrist and finger joints there is a very close connection. Thus, when the fist is tightly clenched (flexion of fingers) the wrist instinctively starts into extension, so as to put the flexors of the fingers into a favourable position for acting. Conversely, in extension of the fingers the flexors are engaged to moderate and steady the movement. These relations between flexors and extensors are illustrated in lead paralysis. Here there is paralysis of the extensor longus digitorum, and the wrist and fingers fall into a position of flexion (wrist-drop). If the proper extensors of the wrist be sound, the patient can straighten out his wrist when told to do so; but if he then tries to straighten his phalanges the wrist becomes flexed again, because action of the flexors normally accompanies movements of extension, and therefore occurs during the attempt at exten-

sion, though the paralysed extensors remain inactive. Again, if the patient's grip be tested, it will be found weak, not because the paralysis extends to the flexors, but because these muscles are, through the wrist-

FIG. 53.

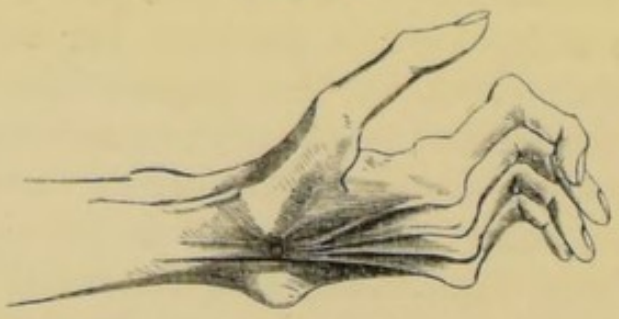


Incomplete paralysis of the interossei, after injury to the ulnar nerve. (After Duchenne.)

drop, unfavourably placed for action. Again, the patient cannot extend the two distal phalanges; this is not because the interossei are paralysed, but because they also cannot act well while the hand is in flexion.

Paralysis of the interossei causes a characteristic pose of the fingers; the last two joints are flexed by the unopposed action of the long flexors, the first joint

FIG. 54.



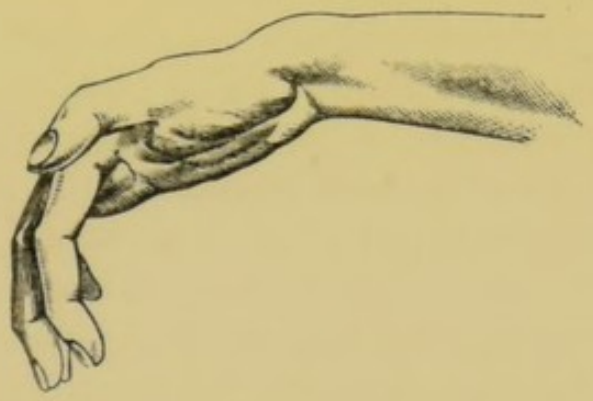
"Main en griffe," resulting from paralysis and wasting of the interossei, thenar, and hypothenar muscles. (After Duchenne.)

over-extended by that of the extensor communis digitorum, and probably the fingers will be awkwardly separated from each other. In its early stages (Fig. 53) this condition is most noticeable when the hands are

being used for such purposes as buttoning clothes, &c. In complete interosseal paralysis with atrophy, particularly when associated with atrophy of the thumb-muscles, the hand takes the shape of a claw, "main en griffe" (Fig. 54).

Movements of the thumb.—The movements of extension and flexion are arranged upon much the same plan as in the fingers. Thus, the abductor brevis and flexor brevis flex the first joint and straighten the second, therein corresponding to the interossei. The extensor primi internodii straightens the first joint only, corresponding to the extensor communis digitorum. The long flexor flexes the second joint. Movements of abduction

FIG. 55.



Atrophy of muscles of thumb-ball; the thumb is drawn backwards into the plane of the hand, and rotated outwards. (After Duchenne.)

and adduction are much more extensive than in the fingers. The extensor ossis metacarpi pollicis is primarily an abductor of this bone; secondarily, it abducts (draws over to the radial side) the whole carpus, this second action being checked by the extensor carpi ulnaris. Adduction of the thumb is of two kinds: the first kind is effected by the extensor secundi internodii; not only is the thumb approximated to the radial side of the forefinger, but it is brought back into the plane of the hand and rotated outwards, so that it looks in the same direction as the fingers instead of partially facing them. The thumb assumes this position when the muscles of the thumb-ball are atrophied (Fig. 55). The

resulting deformity has been compared to an ape's hand. The second kind of adduction is produced by the adductor and inner half of the flexor brevis; the thumb is approximated to the first finger, but remains in front of the plane of the hand. The important movements of opposition are performed by the opponens, abductor brevis, and outer head of flexor brevis. The opponens turns the metacarpal bone inwards towards the palm, the other two muscles rotate the first phalanx inwards, so that the thumb looks towards the fingers, and incline the first phalanx, so that the pulp of the thumb can touch any finger from the first to the little finger.

It is worth while to notice particularly the movements of the thumb and fingers in writing. The thumb is placed in partial opposition, and its metacarpal bone slightly abducted; the pen is thus held between it and the two first fingers; the position of the finger-joints being that given by the interossei—viz., flexion of the first phalanx, and extension of the distal phalanges. The down-stroke is then made by the action —upon the fingers, of their long extensors and flexors, which bend the distal phalanges and partially straighten the first phalanx—upon the thumb, of its long flexor, which bends the second phalanx, and of the extensor primi internodii which straightens the first. Next, the up-stroke is made by the interossei, which extend the last two phalanges and slightly flex the first, while the abductor brevis performs the same office for the thumb. The whole hand is carried along the line as it writes by an external rotation of the shoulder, effected by the infra-spinatus.

Defects due to paralysis, &c., of special nerves of upper arm.—When paralysis can be referred to particular muscles, the next step is to see whether the muscles affected can be grouped under any one nerve. This and the corresponding delimitation of sensory paralysis is evidently a matter to be determined from anatomical knowledge. For details we must refer the reader to anatomical text-books, but

we append some diagrams (Figs. 56 and 57) indicating the nerve-supply of the skin, and a table of the nerve-supply of muscles.

FIG. 56.

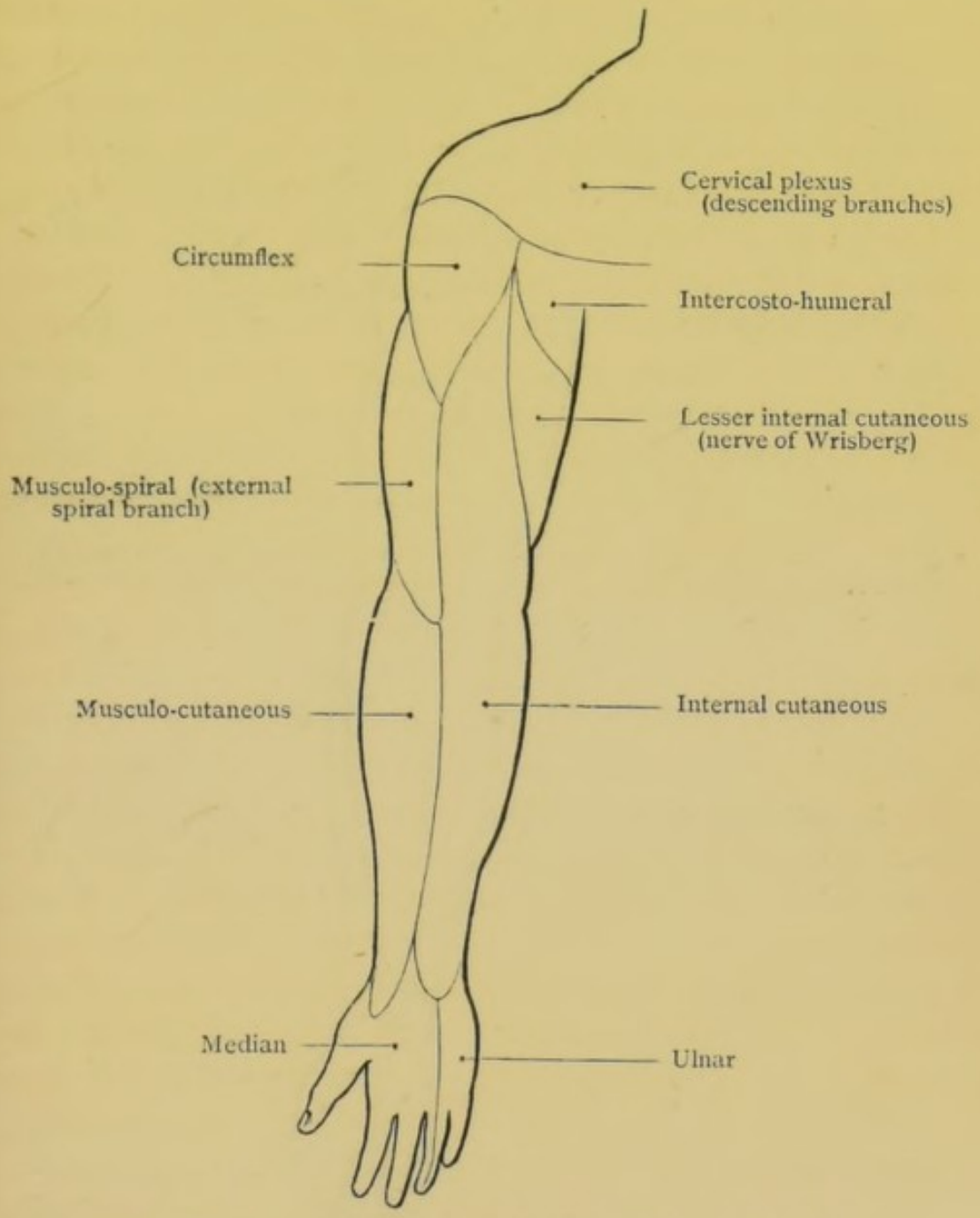


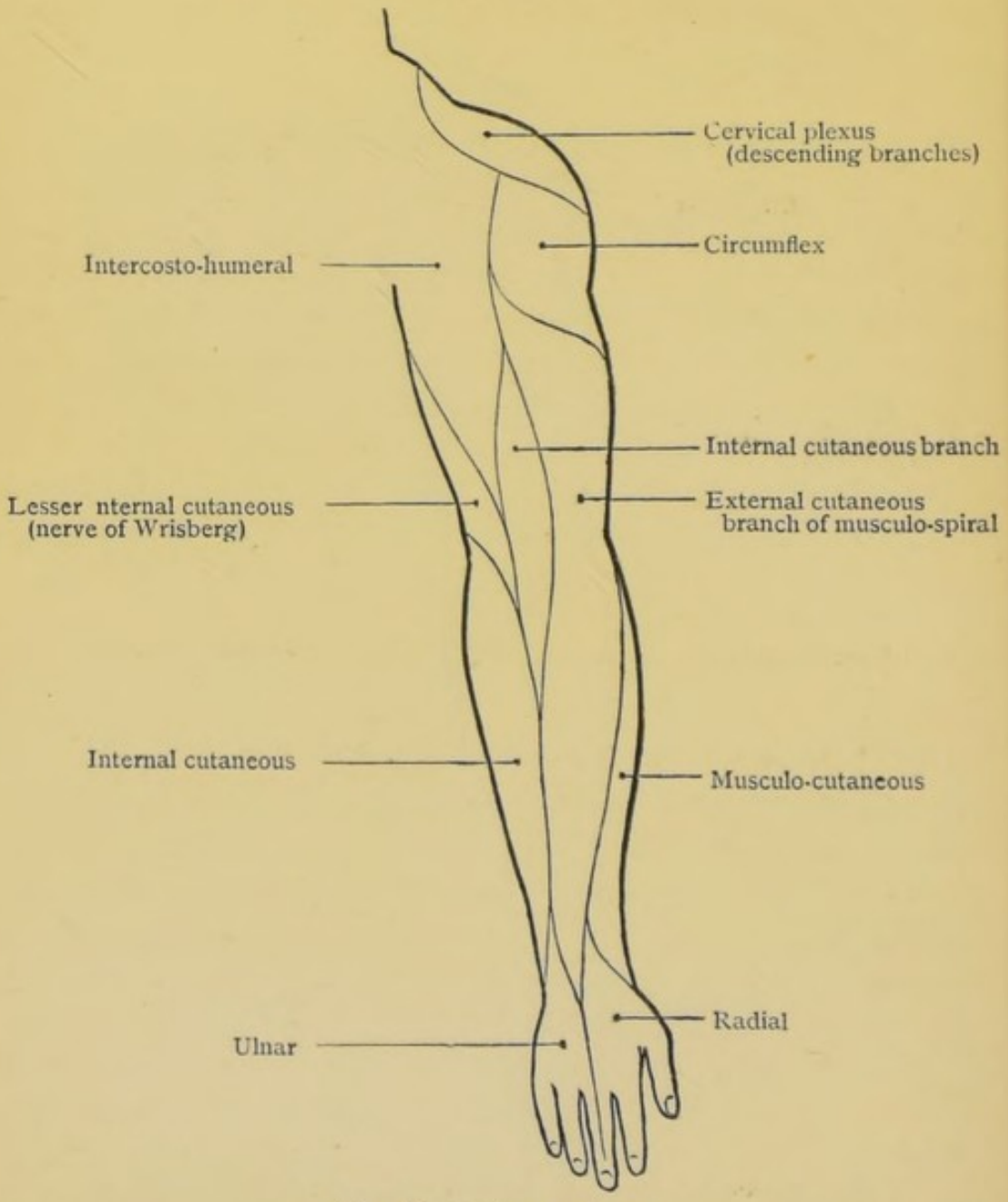
Diagram of cutaneous nerve-supply of upper limb.
(After Flower.)
A.—Anterior surface.

The sensory nerve-supply in the upper limb is, broadly speaking, as follows:—

In the hand.—The palm and palmar aspect of the fingers and thumb are shared between the median and

ulnar nerves, the median taking the radial portion, the ulnar nerve the ulnar portion, and the two districts joining along the middle line of the ring-finger. There is an anastomosis of several nerves (musculo-

FIG. 56.



B.—Posterior surface.

cutaneous, median, and radial) on the thumb-ball. The dorsum of the hand is similarly shared between the radial nerve (branch of the musculo-spiral) and the ulnar; the ulnar having the ulnar part and the radial the radial part, their districts meeting along the middle

line of either the ring or the middle finger. But the dorsum of the last phalanx, in those fingers to which the radial goes, is supplied not by this nerve but by the median.

In the forearm.—The radial part is taken by the musculo-cutaneous nerve, the ulnar part by the internal cutaneous. Along the front of the forearm these two nerve-districts meet, but behind they are separated by a broad stripe which the musculo-spiral supplies.

In the upper arm.—The inner aspect is supplied—above, by the intercosto-humeral and the lesser internal cutaneous—below, by the internal cutaneous. The internal cutaneous supplies also most of the front aspect. The external aspect is supplied—above (*i.e.*, over the deltoid), by the circumflex—below, by the musculo-spiral. The musculo-spiral supplies most of the posterior aspect.

TABLE OF MUSCULAR DISTRIBUTION OF NERVES, IN NECK AND UPPER LIMBS.

Spinal accessory	is distributed to	Sterno-mastoid. Trapezius.
2nd cervical nerve	,,	Sterno-mastoid.
3rd and 4th do.	,,	Trapezius. Levator anguli. Scalenus medius.
Phrenic (mainly from 4th cervical)	,,	Diaphragm.
Branch from 5th cervical	,,	Rhomboids, levator anguli.
Posterior thoracic (from 5th and 6th cervical)	,,	Serratus magnus.
Branch from trunk* formed by 5th and 6th cervical	,,	Subclavius.
Do. supra-scapular nerve	,,	Supra- and infra-spinatus.
Anterior thoracic nerves	,,	Pectoralis major and minor.
Musculo-cutaneous	,,	Biceps, brachialis anticus, coraco-brachialis.
Subscapular nerves	,,	Subscapularis, latissimus dorsi, teres major.

* For further remarks as to the trunk from 5th and 6th cervical roots, see p. 173, "Erb's paralysis."

TABLE OF MUSCULAR DISTRIBUTION OF NERVES—*continued.*

Circumflex	is distributed to	Deltoid, teres minor.
Musculo-spiral	,,	Triceps, anconeus.
		Supinator longus, extensor carpi radialis.
		(Brachialis anticus partly.)
		Supinator brevis, Extensor carpi ulnaris, All the extensors of thumb and fingers — by posterior interosseous branch.
Ulnar	,,	Flexor carpi ulnaris.
		Ulnar half of flexor profundus digitorum.
		Palmaris brevis.
		Interossei and two inner lumbricales.
		Short muscles of little finger.
		Adductor and inner head of flexor brevis pollicis.
Median	,,	Pronator radii teres, flexor carpi radialis.
		Palmaris longus, flexor sublimis digitorum.
		Pronator quadratus, radial half of flexor profundus digitorum.
		Opponens, abductor brevis, and outer head of flexor brevis pollicis.
		Two radial lumbricales.

The musculo-spiral trunk is accessible to pressure and to electrization in the musculo-spiral groove, between the biceps and the supinator longus. It may be difficult to pick out here by electrical currents, owing to the violent contraction of the neighbouring muscles. This nerve is frequently affected by pressure, as in the paralysis which results from sleeping on the arm, or from the use of a badly made crutch. Extensor paralysis results with wrist-drop, resembling in some degree that of lead palsy. But it is unilateral, and involves the supinator longus as well as the extensor of the fingers.

The ulnar nerve can be felt above and behind the

internal condyle. It can be stimulated electrically either here or at the wrist-joint, just outside the tendon of the flexor carpi ulnaris. Ulnar neuritis is by no means uncommon, and one striking result of it is paralysis of the interossei. (Since these muscles also fail at the commencement of progressive muscular atrophy, careful consideration must be paid to the mode of onset, the presence or absence of sensory symptoms, the condition of the muscles in the other hand, &c.) The median nerve can be felt in the upper arm, and stimulated there or at the wrist, inside the tendon of the flexor carpi radialis. When the median nerve is paralysed, pronation of the forearm is interfered with, and also flexion of the first and second fingers. The wrist when flexed deviates to the ulnar side. The movements of opposition in the thumb are lost, and the thumb-ball wastes.

Disease of the circumflex nerve causes paralysis of the deltoid, probably with some anæsthesia of the skin covering it. (The deltoid may become weak and wasted as a consequence of disease in the shoulder-joint. But there is then no reaction of degeneration, and no anæsthesia.)

Disease of the posterior thoracic nerve causes paralysis of the serratus magnus, the main characters of which we have already described.

Affections of brachial plexus—"Erb's paralysis."—It is obvious that the distribution of a paralysis will be much more complex when more than one nerve-trunk is affected, or a cord of the plexus prior to its breaking up into nerve-trunks. As an instance of the latter class, we may take the following group of symptoms. The deltoid, biceps, and brachialis anticus, the spinati muscles, and sometimes the supinator longus, are found to be paralysed. This is believed to be due to disease of the trunk formed by the fifth and sixth cervical nerve-roots, for, on the one hand, this paralysis has been observed to follow injury in the neighbourhood of this trunk; on the other, faradisation of it

will, in a healthy person, produce contraction of the above-mentioned muscles. The electrode should be applied just outside the sterno-mastoid, a little way above the clavicle. (This point is called Erb's point, and the group of symptoms Erb's paralysis, or sometimes combined upper-arm paralysis.)

THE TRUNK.

Sensory affections.—The sensory nerve-supply of the trunk is comparatively simple. Between the districts of the cervical plexus above and the lumbar plexus below the intercostal nerves run in regular zones. In the thorax each intercostal nerve is distributed in the corresponding intercostal space. In connection with this distribution we may make the following remarks :—

(1) In a transverse spinal lesion, where sensation is annulled in the parts below, we can localise the central lesion by observing the level to which the anæsthesia reaches.

(2) In localised disease of the bones, meninges, &c., of the dorsal region, the irritation of the nerve-roots involved produces neuralgic pains, hyperæsthesia, &c., in their distribution. Such sensory phenomena have been called "root-symptoms"; and they may be an early sign of such disease.

In disease of the cord itself there may be felt at a level corresponding to the upper limit of the disease a painful sense of constriction, as if from a band tightly tied round the body. This is called "girdle pain."

(3) Apart from organic disease, neuralgic pains, known as "intercostal neuralgia," may occur in the distribution of the intercostal nerves. Herpes zoster is a trophic affection of the skin also to be connected with them.

(4) Tender points are often found in neurotic subjects at various positions in the course of the intercostals—*e.g.*, above, or more frequently under, the mammæ, at a few inches distance from the spinal

column, over the spinal column itself, in the inguinal regions, &c.

The commonest painful affection of the trunk, lumbago, can scarcely be considered in connection with nervous anatomy.

THE NECK.

Contractions of muscles of neck, or wryneck.—The muscles which balance the head are liable to fall into a state of continuous spasm, known as contracture. Contracture of these muscles forms the disease known as torticollis or wryneck. Sometimes the deformity can be traced home to one muscle. In spasm of one sterno-mastoid, the head is inclined forwards and towards the affected side, rotated towards the opposite side, and the chin at the same time tilted upwards. In spasm of the trapezius (clavicular portion) the inclination is backwards and to the affected side, with rotation towards the opposite side. Contracture of trapezius and sterno-mastoid together is common. Spasm of one splenius inclines the head backward and towards the affected side. Spasm of the deeper muscles may also occur (complexus, recti capitis, &c.), and it may then be impossible to say which are the muscles engaged.

Torticollis, as an independent disease, mostly occurs in patients of middle age or past it. Often there is a history of anxiety or mental trouble, business failures and the like. At first the spasms may be slight and intermittent, producing troublesome jerking movements of the head to one side. Then permanent contracture comes on, and the head is fixed in the faulty position. Usually the affection is unilateral, so as to produce some sort of rotation, but bilateral spasm, producing for instance constant over-extension of the neck, may be seen.

Paralysis of muscles of neck.—Paralysis of these muscles is mostly seen in progressive muscular atrophy. Paralysis of one sterno-mastoid produces

little effect: when both are paralysed, the head cannot be brought forward from the position of over-extension. Paralysis of the clavicular part of the trapezius has also little effect on movement, but when it is atrophied the shape of the neck is considerably altered. Regarding the position assumed in paralysis of the muscles which balance the head, we must say the same as we shall say about the trunk—viz., that in paralysis of one set of muscles, say the flexors, the head does not, as might be expected, fall back into extreme extension; but the patient keeps it carefully bent forward so that its weight can be kept constantly in equipoise by the non-paralysed extensors.

Muscles and the erect posture.—The muscles of the trunk are important agents in maintaining its natural upright posture. Since the weight of the body naturally tends to bend the spinal column forwards, the erectores spinæ of the two sides are those upon which most work is thrown. Paralysis of these muscles is seen in the disease known as pseudo-hypertrophic paralysis. When the extensors of the lumbar spine are failing, the patient instinctively assumes an attitude calculated to carry the centre of gravity backwards, so that if a tendency to fall arise, such tendency may be in the backward direction, and consequently under the control of the non-paralysed flexors. His attitude is that of a man carrying a heavy tray, the upper part of the trunk is bent backwards, forming an obtuse angle with the lower part; the hips are in extension, neither the abdomen nor the buttocks are particularly prominent. A kind of spurious lordosis is thus produced. When the erector muscles are weaker on one side than on the other, the spine is instinctively bent towards the weak side, with the similar object of throwing the weight on the sound muscles. A lateral curvature may be thus produced. When the abdominal muscles (*i.e.*, the flexors of the spine) are paralysed, a different type of lordosis results, according to Duchenne. The centre

of gravity has now to be shifted forwards. This is done by flexing the pelvis on the hips, but at the same time, since a certain counteraction appears to be necessary, the lumbar spine is arched backwards so that a saddle-like depression is produced in the loins; the buttocks are prominent, and the abdomen protuberant.

In standing, various muscles of the lower limbs are called into action, besides those of the trunk. The glutæi medii prevent sideward movements of the pelvis; the hamstring muscles, together with the posterior and crucial ligaments of the knee-joint, prevent that joint from bending forwards: the muscles which pass round the ankle, acting as we shall see at very various cross directions, brace up the foot and ankle-joint. Not only is the action of such muscles necessary, but harmonious action. Hence a patient may be unable to stand, not only from muscular paralysis, but also from want of muscular co-ordination, as in tabes dorsalis.

MUSCULAR MECHANISM OF LOWER LIMB.

We will now consider the muscular mechanism of the lower limb.

Movements at the hip-joint. — Extension is performed by the hamstring muscles and the glutæus maximus. In standing and walking on the level the action of the hamstrings is sufficient to prevent the pelvis inclining forward; but when, in consequence of previous flexion of the hip-joint, stronger extension is needed, as in rising from the sitting or kneeling posture, walking upstairs, jumping, running, dancing, &c., the glutæi maximi are called into action. This can be appreciated by palpating the muscles (α) while walking on the level, (β) while walking upstairs. The flexors of the hip are (1) the iliaco-psoas, which flexes powerfully and at the same time rotates the thigh outwards, (2) the tensor fasciæ femoris, which flexes feebly and rotates it inwards. Their combined action

produces simple flexion, but, unless the tensor fasciæ co-operates, the foot is apt to turn out awkwardly. Complete paralysis of the flexors of the thigh destroys all power of walking, the limb cannot be swung forward nor raised from the ground; mere weakness of them interferes with such actions as walking upstairs where extra flexion is required. There are many external rotators (viz., pyramidalis, gemelli, obturators, quadratus femoris,* most of the adductor muscles, ilio-psoas), as against few internal rotators—viz., the anterior part of the glutæus medius, the tensor fasciæ femoris, lower part of adductor magnus.

The adductor group is numerous and powerful; one of them—viz., the pectineus—is also a flexor of the thigh on the pelvis, so that its action (assisted by the ilio-psoas) is represented by the movement of crossing one knee over the other while sitting. Most of the adductors are also external rotators, but the lower part of the adductor magnus rotates the thigh inwards as it adducts it. This is the movement by which the rider grips his horse's flank.

The glutæus medius acts differently according to the different part of the muscle engaged. The anterior part carries the thigh forwards and outwards, at the same time giving it an internal rotation; the middle part simply carries it outwards (abducts); the posterior part carries it backwards and outwards, rotating it outwards; broadly speaking, it is an abductor of the thigh. But the main use of the muscle is not so much to abduct the thigh, as to act from the thigh as a fixed point upon the pelvis. The muscle of one side acting inclines the pelvis to that side; the muscles of both sides acting, prevent inclination of the pelvis. When the glutæus medius of one side is paralysed, the patient keeps his trunk bent towards that side in walking; when both sides are affected, he inclines alternately from one side to the other. The foot, too, is apt to

* In the semiflexed position of the thigh (as in sitting) this group of muscles act as abductors.

turn outward, the glutæus medius being an internal rotator of the thigh.

Movements at the knee-joint.—The quadriceps femoris is the sole extensor. It is not used in standing (the patella can be felt to be loose then), but in walking it straightens out the advanced leg just as this receives the weight of the body. When these muscles are paralysed, the patient in walking has to avoid flexing his knees, lest he should be unable to straighten them again or unable to prevent further flexion of them by the body-weight, consequently he walks with straight knees, short steps, and with the body leant forward. He cannot rise from the kneeling posture, nor walk upstairs. Flexion of the knee is performed chiefly by the hamstrings; of these, the biceps acts also as a powerful external rotator of the leg, the semi-tendinosus as a feeble internal rotator. To make up for this disparity, the anconeus, a feeble flexor, is a powerful internal rotator. The hamstrings acting from below extend the pelvis on the thigh both in walking and in standing. When they are paralysed, the patient stands and walks with the trunk thrown back (trying to counterbalance by the body-weight the loss of extensor power at the hip). Moreover, being unable to flex his knees sufficiently to clear his toes of the ground, he turns up his feet and toes excessively. The sartorius simultaneously flexes the hip and knee-joint. In walking this double flexion takes place in the backward leg just as it leaves the ground to be swung forward. The gracilis adducts the thigh, flexes the leg, and rotates it inward.

Movements of ankle and toes.—Confusion is liable to arise from the fact that "extension" of the ankle-joint denotes a movement in just the opposite direction to "extension" of the toes. To avoid this, it has been proposed to call the upward movement, whether of foot or toes, "dorsiflexion," the opposite movement "plantar flexion."

In plantar flexion of the foot the main agent is the

muscular mass which terminates in the tendo-Achillis —viz., the gastrocnemius and soleus. It acts chiefly on the back part of the foot, and has besides a tendency to adduct and invert the foot. These muscles bear the greater part of the body-weight as the heel is raised, and paralysis of them will render walking impossible. The other agent is the peroneus longus. The plantar flexion produced by it is less powerful, but it draws downward strongly the inner and anterior part of the foot, planting this part firmly against the ground, while the gastrocnemius is raising the heel. Its action on the unsupported foot is to (plantar) flex, abduct, and evert it; and its tendon running obliquely across the sole braces up the plantar arch. Paralysis of this muscle gives rise to flat-foot, with pain in the sole from pressure on the nerves. The foot becomes inverted during walking, and thus corns arise on its outer side.

Dorsiflexion of foot.—The tibialis anticus dorsiflexes the foot powerfully; at the same time it draws the inner border of the foot upwards and inwards (in exactly the opposite direction to the peroneus longus). It thus dorsiflexes, adducts, and inverts the foot. The extensor communis digitorum dorsiflexes (extends) the first joint of the toes and the foot; it also abducts and everts the foot. The extensor proprius pollicis dorsiflexes the first joint of the great toe, and feebly dorsiflexes and adducts the foot. Paralysis of this group of muscles as a whole causes "foot-drop or ankle-drop" (so commonly seen in peripheral neuritis). In walking, in order to clear the toes from the ground, the patient has to resort to exaggerated flexion of the knee, which gives him a kind of high-stepping action. Paralysis of the tibialis anticus alone causes feebleness in dorsiflexing the foot, which is also abducted by the extensor communis digitorum.

Simple abduction of the foot, without flexion in either direction, is produced by the peroneus brevis; simple adduction by the tibialis posticus.

The manner in which toe movements are produced is analogous to that of finger movements. Thus, while the long extensor muscles extend (dorsiflex) the first phalanges and the long and short flexors flex the second and third phalanges, it is the office of the interossei and short muscles of the little and great toes to flex the first phalanges, keeping the second and third phalanges straight. This particular action of flexion at the first joints with extension of the other joints, takes place whenever the weight of the body is maintained on the toes, and it enables the whole length of the toes, and not merely their last phalanges, to remain in contact with the ground. For the great toe this is of special importance, and consequently most of the muscles of the great toe-ball have this action. The transverse muscles of the foot act as a muscular ligament to the lateral arch of the metatarsals, which the body-weight tends to flatten out. Paralysis of the interossei of the foot causes a distortion somewhat resembling the "main en griffe."

Walking.—It is worth while to recapitulate in order the movements of the lower limbs in walking. Let us suppose that the right foot has been advanced and is about to receive the weight of the body from the left foot which is behind. The transfer necessitates a movement of the trunk forward and to the right. This is effected partly by the extension movement of the left leg (presently to be noticed), partly by the contraction of the right glutæus medius, which draws the pelvis over to the right: simultaneously the left erector spinæ contracts, producing a slight compensatory movement of the vertebræ, in order that the centre of gravity may not travel too fast or too far. As the right leg receives the body-weight, the right knee, at first slightly flexed, is forcibly straightened out by the quadriceps femoris. The hamstring muscles moderate this action, and also prevent the pelvis falling forward at the hip-joint. Meanwhile, in the hindmost (left) leg, the following

processes have been going on. As the body comes forward the left hip becomes mechanically over-extended, the knee slightly flexed, and the foot somewhat dorsiflexed; then comes a muscular movement of extension of the knee (quadriceps, and especially rectus cruris), of plantar flexion of the foot (calf muscles, peroneus longus), and of the first phalangeal joint of the toes (interossei, and short muscles of the toes). Thus, the joints of the left (hindmost) limb are straightened out all along the line, and the necessary forward impulse is given to the body. The weight thus transferred to the right leg, the next action is to bring the left (hindmost) leg to the front, for this the thigh is swung forward and then flexed on the pelvis (sartorius, ilio-psoas, tensor fasciæ femoris); and in order that the toes may clear the ground, a series of movements in the opposite direction to those just described are made—viz., the knee-joint is flexed (sartorius, hamstrings); the foot and toes are dorsiflexed (tibialis anticus, extensor digitorum, extensor longus pollicis). After the foot has cleared the ground, the knee-joint is straightened again, till the foot is placed on the ground in front of the other.

Distribution of sensory nerves in lower limb.—For the sensory nerve-supply of the lower limb we must refer to Fig. 57. It will be seen that—of the *thigh*—the external aspect is supplied by the external cutaneous; the front aspect by the genito-crural above, the anterior crural (middle cutaneous branch) below, and towards the inner side by the anterior crural (internal cutaneous branch), or by the obturator; the posterior aspect chiefly by the small sciatic; the *buttocks* by the small sciatic below, by the lumbar and sacral nerves above; the *perineum* by the pudic, associated with the inferior pudendal branch of the small sciatic on the scrotum or labia; the *front of the knee*, by the patellar plexus from the anterior crural, external cutaneous, and obturator nerves.

Of the *leg*—the inner aspect is supplied by the long

FIG. 57.

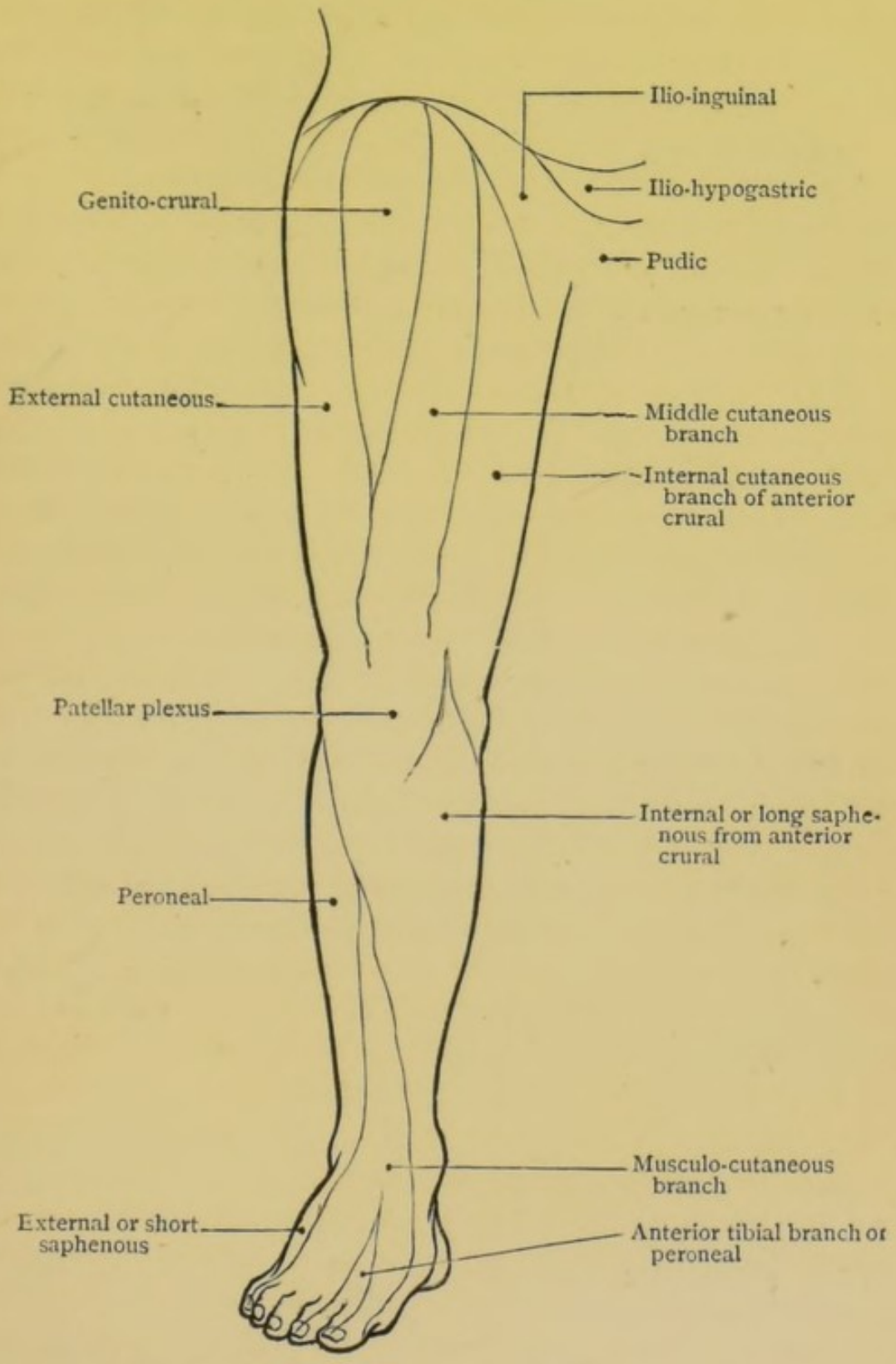


Diagram of the cutaneous nerve-supply of the lower limb.
(After Flower.)
A.—Anterior surface.

FIG. 57.

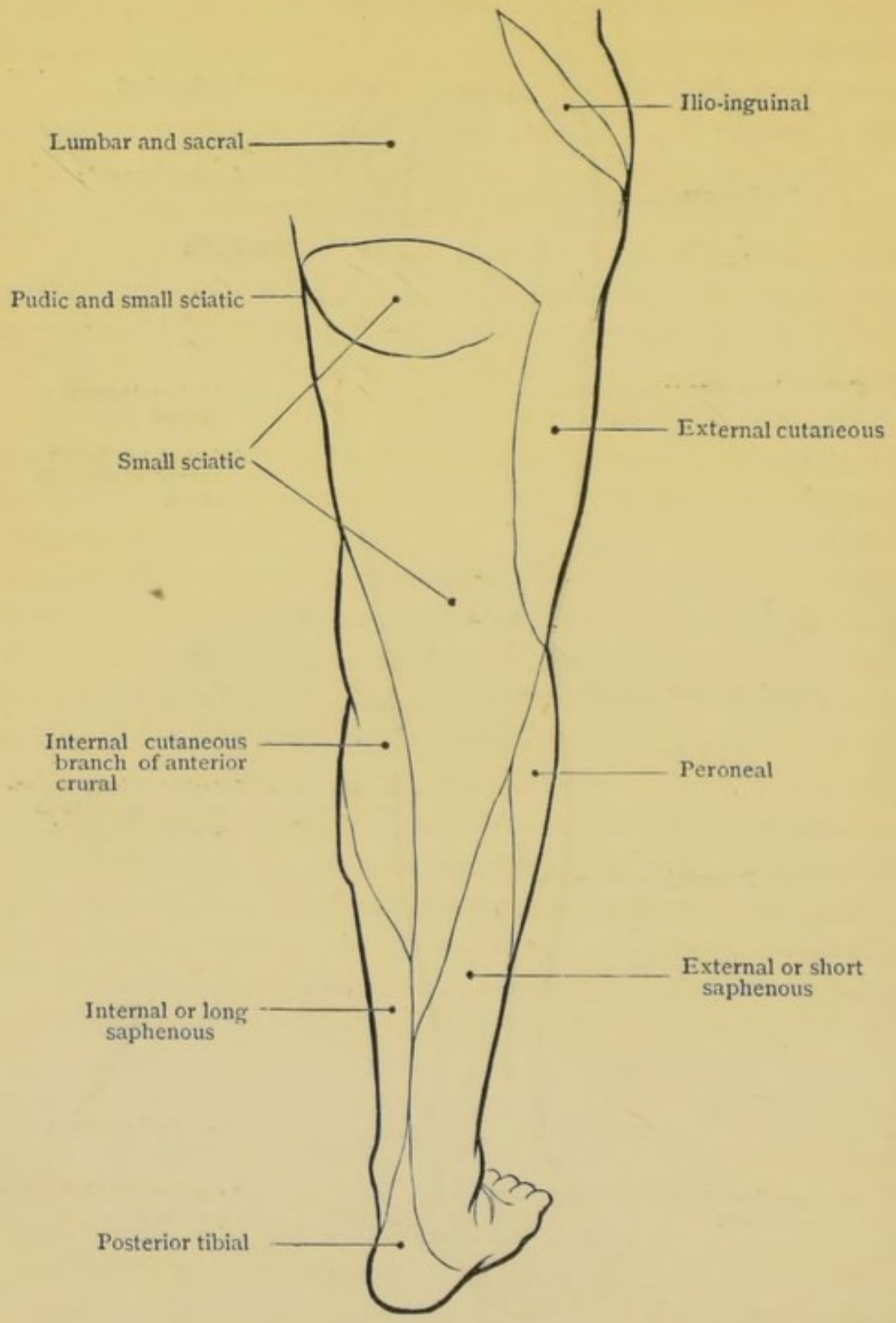


Diagram of the cutaneous nerve-supply of the lower limb. (After Flower.)

B.—Posterior surface.

saphenous branch of the anterior crural, the outer aspect by the peroneal (external popliteal); upon the front aspect these two nerve-districts meet in the middle line, but upon the posterior aspect they are separated near the back of the knee by the district of the small sciatic.

The *inner ankle* is supplied by the long saphenous prolonged downwards from the inner side of the leg; the *outer ankle* (and outer side of foot and of little toe) by the short saphenous (a nerve which is made by the junction of branches from the internal and external popliteal nerves).

In the *foot* the dorsum and dorsal aspect of the toes are supplied by the peroneal (external popliteal) nerve, the adjacent sides of the great and second toes *viâ* its anterior tibial branch, the remainder *viâ* its musculo-cutaneous branch; the sole and plantar aspect of toes are supplied by the internal popliteal nerve *viâ* its external and internal plantar branches, and then two branches divide this district after the fashion of the median and ulnar in the hand, the external plantar taking the outer part of the sole, the little toe and adjacent side of fourth toe (*cf.* ulnar in hand), the internal plantar the remainder (*cf.* median).

The **muscular nerve-supply** is given in the following table :—

TABLE OF MUSCULAR DISTRIBUTION OF NERVES IN LOWER LIMB.

Separate twigs from lumbar nerves	are distributed to	Psoas.
Obturator	,,	Muscles of adductor group, including pectineus and gracilis, obturator externus.
Anterior crural	,,	Iliacus, sartorius, pectineus. Quadriceps extensor cruris.
Separate twigs from sacral nerves	,,	External rotators—viz., pyriformis, gemelli, quadratus femoris, obturator internus.
Superior gluteal	,,	Glutæus medius and minimus. Tensor fasciæ femoris.
Inferior gluteal	,,	Glutæus maximus.

TABLE OF MUSCULAR DISTRIBUTION OF NERVES—*continued.*

Sciatic nerve (main trunk)	is distributed to	Hamstring muscles. Adductor magnus.
Internal popliteal division of sciatic	„	Calf muscles—viz., gastrocnemius, soleus, plantaris.
viâ its posterior tibial branch	„	Tibialis posticus, flexor longus digitorum, flexor longus hallucis, soleus.
viâ internal plantar	„	Abductor hallucis and flexor brevis hallucis, flexor brevis digitorum, and inner two lumbricales.
viâ external plantar	„	The remaining plantar muscles.
External popliteal division of sciatic or peroneal	„	Tibialis anticus (per recurrent articular branch).
viâ anterior tibial branch	„	Tibialis anticus, extensor longus digitorum, extensor proprius pollicis, peroneus tertius, and extensor brevis digitorum.
viâ musculo-cutaneous branch	„	Peroneus longus, peroneus brevis.

Disease limited to the individual nerve-trunks of the lower limb is less common than such disease in the upper limb; and we shall not comment on each separately.

Sciatica.—The common affection called sciatica calls for special notice. This is characterised by neuralgic pains extending along the course of the sciatic nerve and its branches. The pain may be dull and aching, or sharp and lancinating; it is increased by pressure or tension on the nerve-trunk. This fact may be utilised for diagnosis as follows. Simple pressure upon the nerve where it leaves the pelvis between the tuber ischii and great trochanter evokes pain in a case of sciatica. But to be sure that such tenderness is due to disease of the nerve and not of the hip-joint or neighbouring parts, we may try the effect of tension thus. Put the patient on his back and flex the hip,

first with the knee flexed, and secondly with the knee kept straight. The tension on the nerve, and consequently the pain, will be much greater when the hip is flexed with the knee straight.

The movement of walking also stretches the nerve, and consequently the patient walks very carefully, trying to keep his hip-joint straight and his knee slightly flexed. Usually there is no paralysis, but cases do occur in which actual muscular weakness and even wasting supervene. Probably there is then not a mere neuralgia, but neuritis with destruction of nerve-fibres.

The commonest causes of sciatica are exposure to cold, gout, rheumatism, nervous exhaustion due to overwork, to anxiety, to want of fresh air, and of proper food. The possibility of diabetes should be thought of. Syphilis has been alleged as a cause. Neither should we forget that sciatic pains (especially if accompanied by symptoms of true paralysis) may be symptomatic of new growth or inflammatory disease within the pelvis. Double sciatica, as a purely neuralgic affection, is rare; and should lead us to look, not only for intra-pelvic disease, but for disease of the lower part of the cord or its coverings.

The treatment consists in combating the constitutional cause where such can be found—thus, iodide of potassium, alkalis, salicylate of soda, saline purges, &c., may be given in rheumatic or gouty cases; quinine, iron and other tonics, with change of air, in other cases: and in local measures—rest to the limb; counter-irritants, such as blisters and mustard plasters; anodynes, whether local, as morphia, cocaine, belladonna, or by the mouth, as morphia, antipyrin, gelsemin. The constant current applied down the course of the nerve is sometimes very valuable. Yet many cases persist a long time, refractory to all treatment.

CHAPTER VI.

NEURITIS.

ORGANIC SPINAL DISEASES.

THE diagnosis of disease of a nerve-trunk must rest largely on anatomical considerations. We conclude that a given nerve is diseased because the area of paralysis, &c., corresponds with the known distribution of that nerve. But in practice this is not always so simply done, for the following reasons:—(1) Disease of single nerve-trunks is less common than might be imagined; for constitutional causes may affect more than one nerve at a time, or may affect the trunks higher up when they form the plexuses. (2) Sensory symptoms, notably anæsthesia, do not always give us the assistance which we might expect from them. For some unexplained reason, anæsthesia is often less marked and more fugitive than muscular paralysis. Neither does its grouping always correspond accurately with anatomical teaching; perhaps because the distribution of sensory nerves may vary in individual cases, or perhaps because the functions of a disabled nerve may be taken on by its neighbours. (3) Even muscular paralysis may not extend fully over the distribution of the nerve in cases where this is not completely severed. It behoves us therefore to consider, as well as its distribution, the other clinical phenomena of nerve-trunk disease.

Acute neuritis.—An acute neuritis, or inflam-

mation of a nerve-trunk, affects primarily its sheath or interstitial tissue. These parts become congested, swollen, and infiltrated with cells. Hence pain at the seat of the disease (from the implication of the nervi nervorum), and the presence, sometimes ascertainable by touch, of an elongated tender swelling of the nerve. As the nerve-fibres themselves become irritated, pains begin which are felt in the distribution of the nerve, are often very severe, and may have, like the pains of neuralgia, a periodic type. There may be extreme tenderness (hyperæsthesia or hyperalgesia) not only of the nerve, but of the parts supplied by it. To these symptoms of irritation succeed those of destruction of the nerve-fibres—viz., anæsthesia and paralysis in the district supplied by them; then those of neuro-muscular degeneration—viz., wasting of muscle and reaction of degeneration. Pain, and particularly the burning pain known as "causalgia," may persist even in anæsthetic parts. Trophic changes may show themselves in other parts than the muscles, chiefly in the skin and nails. Thus, the nails become thickened, incurved, and talon-like; the finger-ends tapering, and their skin thin, tense, smooth, and shining red ("glossy skin"); blebs may appear containing serous or sanious fluid which break and leave a deep ulcer. These latter symptoms probably denote a continued irritation of the nerve. Neuritis may be connected with the following causes: injury; exposure to cold; constitutional causes, such as rheumatism, gout, syphilis, diabetes, and perhaps alcoholism. Leprosy localises itself in the nerve-trunks; new growths may also affect them. The treatment of neuritis will run on the same lines as that of neuralgia—viz., to attack the constitutional cause; to secure perfect rest and protection from jars and blows to the affected limb, and to relieve pain during the acute stage; to apply blisters and counter-irritants. Mercury is recommended internally, even for simple cases; and there can be little doubt that the proper application of the

constant current both to the nerve and to the muscles is of great service.

Peripheral neuritis.—By "peripheral" neuritis we mean disease which affects the nerves near their termination. It may ascend so as to affect the trunks or even the roots, but its primary and principal seat is in the finer branches. Its nature may be inflammatory or degenerative, or both. It affects the hands, feet, or limbs, without limitation to any one nerve-district; is almost always bilateral, and sometimes spreads over the whole body. Therefore it is also called "multiple" peripheral neuritis.

Typical form (alcoholic).—Since this subject, when treated fully, is one of great complexity, we shall describe principally the commonest type of it. In this the disease is due to prolonged excess in alcohol, usually taken as spirits. The patient is often a woman of middle age or past, who has been drinking in secret. Probably before the outbreak of paralysis she will have had such premonitory symptoms as cramps in the legs, tingling, tenderness, and pains which she will call rheumatism. Loss of power in the legs is the complaint for which she generally is brought to the doctor. As a rule this has developed gradually, but sometimes very rapidly, so as to suggest an acute myelitis. It is often ascribed to a "chill," indeed there seems reason to think that this is the determining cause sometimes. On examination we find a paralysis of the legs which has the following characters. The paralysed muscles are flaccid,* and (if the disease be advanced) are wasted, and usually show changes in their electrical reactions.†

* There may be spasm of some muscles, notably the hamstring muscles. But there is no general rigidity as in descending sclerosis after transverse dorsal myelitis.

† Alterations of polar formula, and sometimes complete RD, are to be found; but less generally than in disease of a nerve-trunk. There is nearly always loss of faradic contractility and diminished reaction to galvanism.

The tendon reactions are absent. Further, the paralysis specially affects the anterior tibial group of muscles, so that the patient cannot dorsiflex the foot and toes, and there is a characteristic dropping of the foot. The bladder and rectum act normally as a rule. Sensory symptoms are usually best seen in the early stage. Besides the spontaneous pains and cramps already mentioned, the skin may be hyperæsthetic, so that the patient complains of the least touch. This may pass off and leave anæsthesia. But a still more constant feature is tenderness to deep pressure of the nerve-trunks and of the muscles, particularly the calf-muscles. The anæsthesia when it exists is limited to the distribution of no one nerve, but envelops the foot and lower leg like a stocking. As to the upper limbs, even if no complaint is made about them, careful examination may show that they are affected in a minor degree. Or the paralysis may obviously involve them also; and in bad cases even the trunk, face, cardiac, and respiratory nerves. Bedsores are exceptional. The ocular movements, pupillary reactions, and fundus oculi are generally normal; but squint may occur, and sometimes nystagmus. The pulse is often frequent, with a tendency to irregularity. The mental condition is remarkable. The memory fails, particularly for recent events; the patient will write letters and sign cheques twice over. She may give a connected history of her illness, but on further inquiry it proves quite erroneous. She seldom appreciates her real condition, for she will describe how she went walking, shopping, &c., yesterday, when she was actually unable to move. She may not recognise the place where she is. Sometimes, however, there is marked hebetude, and in rarer cases delusions and delirium.

The essential point in treatment is to enforce abstinence from alcohol. For this it will be advisable, as a rule, to remove the patient from home, and place her in a hospital or similar institution. Rest in bed,

wholesome food, warmth, and anodyne applications for the painful limbs, will be requisite at first, with the treatment of such gastric or other complications as may be present; and later, tonics, iodide of potassium, galvanism, and massage. If the capital point of abstinence from alcohol can be secured, even severe and long-standing cases may in course of time recover. But nevertheless there are dangers to life, which we shall rank as follows: (1) The whole nervous system may, from chronic poisoning, be so degenerate, that the patient dies, we hardly know why. (2) The neuritis spreads to vital nerves, pneumogastric or respiratory. (3) Causes outside the nervous system produce death, notably (*a*) phthisis, a possible accompaniment of peripheral neuritis, which should be always borne in mind: (*β*) dilatation of the heart; (*γ*) cirrhosis of the liver.

As to the morbid anatomy of this disease: the changes in the central nervous system (such, that is, as might be held to stand in direct connection with the neuritis) are slight, if any; the nerves to the naked eye may be normal, or may look swollen and reddened, or, in very chronic cases, grey and small. The microscope shows in them two sets of changes—(*a*) in the connective-tissue sheaths and endoneurium, infiltration with cells or connective-tissue overgrowth—that is to say, the results of inflammation; (*β*) in the nerve-fibres, breaking up of the myeline into blocks and globules, with final absorption of it, and disappearance (at any rate, in the advanced stages) of the axis cylinders—that is to say, degenerative changes. As yet we hardly know whether the inflammatory or the degenerative process be the primary, or whether they both go on side by side. We shall note two further points: (1) that infiltration with inflammatory cells has been seen in the muscles, as well as in the nerves. This may perhaps explain the tenderness of the muscles. (2) That if the axis cylinders of a nerve be destroyed by the disease, the nerve and muscle-fibres

below that point will fall into a state of secondary (Wallerian) degeneration (*vide* p. 66).

Other forms.—Peripheral neuritis assumes other forms, and may be due to other causes. Here we can only briefly allude to them. As to their morbid anatomy, it seems that in some forms this may be more strictly degenerative and limited to the parenchyma of the fibres than in that we have described. Further, sometimes the motor fibres are affected, to the exclusion of the sensory; and this may make the clinical diagnosis of neuritis very difficult. Two groups may be made, if we divide these forms according to symptoms: (1) Ataxic. There is little muscular weakness; the prominent features are unsteadiness of gait, and awkwardness of the hands. The tendon-reactions are absent, there may be little or no wasting of muscle or change in electrical reaction. The diagnosis has to be made from tabes dorsalis (progressive locomotor ataxy). Post-diphtheritic paralysis, and diabetic neuritis, not unfrequently take this form. (2) Paralytic. There is obvious paralysis, the muscles become flaccid, may waste and show reaction of degeneration. The onset may be acute, sometimes with high fever, the paralysis reaching its maximum within a few days, or the disease may be less rapid and more progressive. The diagnosis has to be made from a general myelitis, from the obscure disease known as acute ascending paralysis (Landry's disease), or from affection of the anterior cornual cells of the cord, acute or chronic, and from such a polio-myelitis in the absence of, or after the disappearance of, distinct sensory symptoms, the diagnosis may be well-nigh impossible.

And other modes of causation.—Many and various causes are assigned for peripheral neuritis. Some of these, like alcohol, are toxic in nature—viz., arsenic, carbon-bisulphide (used by india-rubber workers), salts of copper, silver, &c. Lead-palsy would be reckoned by some authorities under peri-

pheral neuritis. Other causes consist of constitutional diseases, either chronic, as diabetes, syphilis, tubercle, the disease known as beriberi; or febrile diseases, as diphtheria, typhoid, mumps, measles, &c. The neuritis may arise during or more commonly after the fever. Thirdly, peripheral neuritis may occur as part of a more general nervous degeneration, notably in tabes dorsalis. Fourthly, it may be connected with exposure to cold; and, lastly, there may be idiopathic cases—*i.e.*, those whose origin is as yet undiscovered.

Diphtheritic paralysis. — Of the paralyses which form the sequelæ of acute disease the principal is post-diphtheritic paralysis. It is probable that this is in most instances a degeneration of the peripheral nerves, though not always and entirely so, for in some cases disease of the spinal motor cells has been demonstrated. One of its commonest and earliest manifestations is paralysis of the soft palate, causing the voice to become nasal, and fluids, when swallowed, to regurgitate through the nose. The natural movement of the palate, seen on pronouncing "eh" with an in-drawn breath, is lost. Such palsy of the palate may occur so soon after the primary disease as to suggest that a local damage has been wrought by the inflammation. But that the damage is more than local is shown by the affection of other parts. Ocular paralyses are seen; most commonly a paralysis of the accommodation, so that the patient has difficulty in reading small type, less frequently palsy of the external ocular muscles, giving rise to squint; or perhaps a paralysis of associated movements, so that the eyes cannot be converged, moved upwards, &c.; or perhaps there may be total ophthalmoplegia externa. Further, there is affection of the limbs. Although a well-defined anæsthesia is not present, there is often numbness, tingling, and perhaps blunting of sensation; there may even be tenderness of the nerve-trunks to pressure. And there is either (as we have said) a definite loss of power, or

else an ataxic condition of the limbs. In either case the patellar tendon-reaction disappears. Diphtheritic paralysis varies considerably in severity; there may be little more than palsy of the palate, coupled with loss of knee-jerk, or the patient may be quite unable to stand. The usual course is favourable; the disease clears up gradually, and disappears without sequelæ. But sometimes, particularly in children, death may occur. This mostly results, it would seem, from implication of the respiratory and cardiac nerves or centres. Hence, we must consider the following signs to be of serious import: paralysis of the diaphragm, vomiting, frequent feeble irregular pulse, dyspnœa without apparent cause, or morbid modifications of respiration. Iron, strychnine, massage, and electricity to the limbs are the means usually employed in treating diphtheritic paralysis.

Lead paralysis.—The morbid anatomy of lead paralysis is still somewhat uncertain. That there is some organic change, and that this lies either in the nerves or in the spinal motor cells, we should infer from the wasting and electrical changes in the muscles. The paralysis may in some instances, like alcoholic paralysis, manifest itself suddenly, but, as a rule, the onset is gradual. It has, in typical cases, a characteristic localisation—viz., in the extensor muscles of the wrists and fingers, more particularly the extensor communis digitorum. If this muscle be paralysed on both sides, without affection of sensation, and the adjacent supinator longus be at the same time sound, the probability of lead paralysis is very great, even apart from a corroborative blue line on the gums and a history of lead poisoning. For wrist-drop suggests lead even more surely than foot-drop suggests alcohol. The affected muscles waste, and show electrically reaction of degeneration, and that usually in a very typical fashion. There is no anæsthesia. The paralysis may spread further than this, to the hand-muscles, those of the upper arm, or it may, in severe

cases, be more or less general. Sometimes it may be abnormal in distribution, affecting, for instance, the upper arm, while it spares the forearm. There is, for the most part, little danger to life; on the other hand, the paralysis is often very chronic and intractable, and after apparent cure, even at a long interval, it may break out again without obvious cause. Neither must it be forgotten that the lead may affect the nervous system in other ways, producing sometimes tremors, sometimes epileptic fits, sometimes a condition resembling general paralysis of the insane. We must remember, too, the predisposition of patients suffering from chronic lead poisoning to renal disease, and examine the urine, the heart, and the vascular system. For the treatment of the paralysis itself galvanism is generally used, while iodide of potassium and a daily dose of sulphate of magnesia are given to remove the lead from the system.

DISEASE OF THE ENVELOPES OF THE CORD.

Acute spinal meningitis.—An acute general spinal meningitis is described as taking place either from exposure to cold ("rheumatic"), or from unknown causes ("idiopathic"). This is certainly uncommon; more usually it results from some specific disease, as, for instance, tubercle, syphilis, pyæmia, fevers such as small-pox, typhus, &c. &c., or the disease known as epidemic cerebro-spinal meningitis. But in such cases a cerebral meningitis may co-exist and obscure the symptoms of the spinal disorder. Disease and injury of the neighbouring parts, such as the vertebræ, may also set up meningitis.

An acute spinal meningitis affects the pia and arachnoid; and in a well-marked example the surface of the cord (after opening the dural sheath) is found lined with inflammatory lymph, or even pus, held mostly in the meshes of the arachnoid. The inflammation may penetrate the surface of the cord, causing softening of its periphery (meningo-myelitis), and possibly of the

deeper parts. The nerve-roots may be similarly affected. The symptoms are as follows:—

Rigors, pyrexia, and other symptoms of severe internal inflammation.

Pain in the back, often very severe; lancinating pains, tenderness, and paræsthesiæ in the limbs. Movement, and particularly the movement of rotating the vertebræ, makes the pain much worse.

The muscles of the back are rigid, and when the upper parts of the cord are affected there is retraction of the head.

There may be at first no actual paralysis, but movement is difficult on account of the pain and stiffness. Later, if the nerve-roots or cord be damaged, paralysis, with muscular wasting and reaction of degeneration, will supervene.

For a like reason the tendon-reactions are generally annulled.

There may be retention of urine and fæces.

The patient may die during the height of the disease; or he may, in a favourable case, entirely recover; or he may die at a later stage from the effects of softening of the cord (bedsores, cystitis, and the like); or he may recover with paralysed and wasted limbs. In this last case, should the wasting be due to nerve-root lesion rather than to cord lesion, there is hope that it may eventually disappear.

For treatment, in addition to general measures (such as are calculated to afford to the patient perfect rest, relief of pain, and assuagement of fever), mercury has been recommended even in non-syphilitic cases; also iodide of potassium, spinal ice-bags, counter-irritants to the spine (applied with caution). After recovery, galvanism and massage will probably be of much use for the relief of paralysis.

Tubercular spinal meningitis usually occurs as an adjunct and subsidiary to the cerebral disease; but sometimes the conditions are reversed, the main symptom being a paraplegia which spreads upwards and proves fatal.

Syphilitic meningitis may be acute or subacute; it is more likely to be localised than the other forms. Nevertheless, in syphilitic subjects a meningitis occurs which spreads over the surface of the whole cord, and possibly of the brain also, with great rapidity. Mercurials and iodide must be pushed vigorously, and are here of undoubted value.

Chronic spinal meningitis.—A chronic meningitis affecting the pia mater and arachnoid may be the result of chronic alcoholism. The membranes become thickened and matted, the cord is liable to be damaged from injury to its vessels. The symptoms are spread over a longer period, and the true cause of the pains, &c., may be only apparent after paralysis has begun to set in.

Affections of spinal dura mater.—As to diseases originating from the spinal dura mater, they are not common. Gummata may grow from its internal surface. A peculiar disease, consisting of (inflammatory ?) thickening, which spreads inwards from the dura, and usually affects the cervical enlargement, causing a fusiform swelling in this region, is called by Charcot "cervical pachymeningitis." It develops gradually, causing at first pains and paræsthesiæ in the hands and arms, then, as the nerve-roots begin to perish, paralysis with wasting and anæsthesia. The paralysis usually has this peculiarity, that it affects the district of the ulnar and median nerves, but not the musculo-spiral, so that the hand and first joint of fingers are hyper-extended while the small hand-muscles are wasted. As the cord becomes pressed upon, paraplegia develops. This spinal pachymeningitis is certainly uncommon.

Effects of vertebral caries (Pott's disease).—Much more important, because much more common, is the paralysis which may accompany caries of the vertebræ. Caries of the vertebræ may be accompanied by two sets of symptoms: (1) those which appeal chiefly to the surgeon—viz., pain and tenderness to handling of the affected vertebræ, and, in addition, the

well-known angular curvature; (2) symptoms indicating disease of the cord, and possibly nerve-roots, at the level of the vertebral disease. It is natural, at first sight, to suppose that these latter symptoms are due to mechanical distortion of the cord from the curvature. This is not so, or not wholly so, for paralysis may be present with very slight curvature, and absent when the curvature is great. The paralysis is generally due to a "compression myelitis" (*vide* p. 75), *i.e.*, upon the diseased bones, within the spinal canal, inflammatory and caseating material is heaped up, which gradually compresses and invades the membranes and the cord. The nerve symptoms consist in paraplegia, gradual in onset, of the parts below, affecting first and principally the motor functions, and next the sensory. The bladder and rectum are paralysed in bad cases. There may or may not be at the onset symptoms indicative of interference with the nerve-roots at the level of the caries—viz., pain and hyperæsthesia with paralysis in their distribution. Usually such "root-symptoms" are not marked, unless the disease happens to affect the nerve-roots of the brachial plexus, but more commonly the caries is in the dorsal region. Thus the nervous symptoms indicate only transverse disease of the cord at a particular level, usually the dorsal region; they do not tell us the nature of the disease. This is inferred from the vertebral symptoms, principally from the curvature. Hence the importance of examining the spinal column in all cases of paraplegia.

Some remarks may be made, however, on the characters of the paraplegia. (1) It is, like all paraplegia from disease of the dorsal cord, "spastic" in type. The legs are dragged if the patient can still walk, and they become rigid. The reflex actions are exaggerated, and perhaps more so in this paralysis from compression than in any form of spastic paraplegia. Very excessive knee-jerk, very marked ankle-clonus, and excessive plantar reflex are seen even with moderate paralysis. (2) The degree of sensory paralysis may serve more

or less as an index of the damage to the cord. Complete anæsthesia indicates grave mischief, and with it usually come the serious complications of cystitis, bedsores, &c. Even bad cases, however, are more hopeful than paraplegiæ from disease originating in the cord; for the mischief may arise from compression rather than destruction, and then recovery is still possible. It need hardly be added that the symptoms will vary according to the part of the cord which is affected (*vide* p. 203, transverse myelitis). Should the upper cervical vertebræ be carious, there is the danger of sudden death from dislocation caused by the weight of the head. The treatment indicated for paralysis associated with vertebral caries is absolute and prolonged rest in bed, to allow the caries to subside and the vertebræ to consolidate themselves, for usually in this case the secondary affection will subside also; cod-liver oil, tonics, and feeding suitable for strumous patients; then surgical measures, such as spinal jackets and supports, for the relief of the bone disease. By some, the application of the actual cautery to the skin of the back is recommended while the disease is still in progress. In old-standing, obstinate cases it may be considered whether opening the spinal column, with the design of removing material which may be pressing on the cord, will offer prospect of relief.

Effects of vertebral cancer.—We shall here only mention a second form of vertebral disease, luckily less common than the preceding. This is cancer of the vertebræ. Like caries, it causes paralysis of the parts below by invasion of the cord; but it is distinguished by the extreme severity of the pain which accompanies its progress. This has earned for the disease the name of "paraplegia dolorosa."

DISEASES OF THE SPINAL CORD.

Classification.—Diseases of the spinal cord itself may be classified in various ways. They may depend

upon an acute softening (inflammatory or necrotic), or upon a more chronic inflammation. This group falls under the head of "myelitis." They may, on the other hand, depend on a degenerative process, this may be secondary or primary, as we have already said (*vide* pp. 67, 71). But between chronic inflammation and degeneration it is difficult to draw a line.

Again, spinal disease may be selective, discriminative, or "systematic"—*i.e.*, limiting itself to those tracts or areas of the cord which (we have reason to think) constitute definite physiological or embryological systems. On the other hand, it may be non-selective, indiscriminate, or "diffuse," attacking the tissues and spreading in them without regard for such boundaries. Most "system" diseases are degenerative, and most diffuse diseases inflammatory; but there may be exceptions. Lastly (turning to their mode of onset and cause), they may be, like all other diseases, acute, subacute, or chronic. (We omit for the present the subjects of syphilis and new growths affecting the cord.)

It is useful to bear these distinctions in mind, but convenience of description, as well as the limited state of our knowledge concerning some diseases, forbids us to follow out such a classification too strictly.

Under the term acute myelitis we shall here rank acute softening of the cord, whether from inflammation or from the necrosis which follows vascular obstruction. The processes are doubtless different, but it may be impossible to say which has been at work in a given case. For the net result in either case is softening, and the minute examination of softened nerve-tissue is somewhat unsatisfactory. An acute myelitis may be general—*i.e.*, affecting a long length of cord; or local, affecting a short length only. Or there may be numerous small patches of myelitis scattered through the cord. This is called "disseminated myelitis."

Acute general myelitis is not common; and fortunately so, for it is fraught with great danger to life. Exposure to cold, especially under circumstances of privation or overwork, is alleged to be the common cause. Other causes are—the infection of acute specific diseases, of pyæmia, and perhaps syphilis. It may also be secondary to injury, or disease of the neighbouring bones or membranes. Pain in the back and paralysis are the chief symptoms. The pain is much less severe than in meningitis. But the paralysis is an earlier and much more pronounced symptom than in meningitis. The muscles are flaccid, they soon show electrical reaction of degeneration, and begin to waste. The tendon-reactions are abolished. The bladder and rectum are paralysed. There is paralysis of sensation; absence of skin reflexes. Bedsores and cystitis are apt to appear. Delirium and coma may supervene, even apart from cerebral disease. In some exceptional cases optic neuritis has been seen. In the acute stage little more can be done than by careful nursing and feeding to keep the patient alive; a water-bed should be used; particular attention must be given to the skin of the back, and the state of the bladder. Should the patient recover, the paralysis that is left must be treated like the similar sequelæ of meningitis; but the prospect of cure is not so good, since there has been destruction of nerve-centres, and not of nerve-roots only.

Acute ascending paralysis (Landry's disease).—We may here mention the disease known as acute ascending paralysis, or "Landry's disease." The symptoms suggest an acute, rapidly spreading myelitis, but no constant lesion has been found in the cord. Some say that the lesion is a multiple neuritis. The spleen is found to be enlarged; hence it has been argued to be due to some acute specific disease; nay, it is even said that micro-organisms have been detected in the peripheral nerves. Dr. Bristowe mentions a case which he thinks was a

manifestation of hydrophobia. It manifests itself as a paralysis, beginning in the lower limbs and spreading rapidly upwards. The precise order of its spread is said to be—muscles of toes and feet, posterior muscles of thigh and pelvis, anterior muscles of thigh; muscles of fingers, scapular muscles, upper arm muscles; trunk; respiratory muscles; muscles innervated from the medulla. The limbs are flaccid and the tendon-reactions absent; but the electrical reactions are normal. Again, though there be numbness and paræsthesiæ, there is no definite sensory paralysis. These features are said to distinguish it from acute general myelitis. It may terminate fatally in a few days, or within a week or two; some few cases recover. Large and frequent doses of ergotin are credited with having saved life in one case.

Localised or transverse myelitis. — Local myelitis (*i.e.*, that which involves only a small length of cord), when it is acute will probably affect the whole thickness of the organ; for this reason it is called "transverse myelitis." (We must except, however, the acute affection of the anterior cornua, which we shall describe presently.) There is little to add concerning its causation, save that any deficiency of arterial blood-supply is likely to cause a local softening. Syphilis, a tolerably definite cause, may probably act in this way. The symptoms vary according to the level of the lesion, and according to its completeness.

Of dorsal region.—By far the commonest level is the dorsal region. A complete, or as it is sometimes called a "total," transverse dorsal myelitis produces motor paralysis of the lower limbs, paralysis of the bladder and rectum, and sensory palsy of all parts up to the level of the lesion; at the upper level of the anæsthesia there may be a zone of hyperæsthesia, or perhaps a "girdle pain." Acute bedsores are liable soon to form upon the sacrum. The condition of the lower limbs at first is this: they are flaccid, and

the skin reflexes and tendon-reactions are abolished. According to most accounts this is due to "shock," but as time goes on, and more particularly as secondary lateral sclerosis develops, the tendon-reactions return and become exaggerated, and rigidity sets in. This is certainly true for cases where the severance of the cord has not been quite complete. Where it has been complete, it would appear that though the skin reflexes may return, the tendon-reactions remain absent, and the legs do not become rigid. But their electrical reactions are not (as in lumbar myelitis) abnormal. A complete transverse lesion will probably never recover: in the milder cases sensation may return, the bladder may recover itself, often imperfectly, and motor power be more or less regained in the legs. The patient walks with his legs held stiffly, dragging his feet after him instead of lifting them from the ground; it is the gait of "spastic paraplegia."

Of lumbar or cervical enlargements.—Myelitis of the lumbar and cervical enlargements is characterised by the symptoms proper to destruction of the nerve-cells of these parts. Suppose the lumbo-sacral region of the cord destroyed, then not only are the legs, bladder, rectum, and nervi erigentes paralysed, but the muscles, instead of becoming rigid, are at first flaccid; then show degenerative changes to electrical tests; then waste; the skin reflexes and tendon-reactions are alike permanently abolished. The anæsthesia will correspond to the distribution of the lumbar and sacral nerve-roots (*vide* p. 11). Similarly an affection of the cervical enlargement will cause a wasting paralysis of the arms, with a spastic paraplegia of the legs.

Chronic myelitis.—In chronic myelitis the morbid process falls principally on the connective tissue and neuroglia. By the overgrowth of these tissues the nerve elements are, so to speak, choked. The general result is not softening, but induration; and except by the distribution of the lesions, it may

be difficult to tell whether we have to do with a chronic inflammation or a chronic degeneration. Chronic alcoholism, vascular disease—whether from atheroma, from renal disease, or other source—and syphilis rank among the causes. The symptoms will be regulated, as in acute myelitis, principally by the position of the lesion. They will differ from those of acute myelitis chiefly in their gradual onset, and in the fact that they are more apt to be heralded by symptoms of irritation, such as pains, paræsthesiæ, cramps, and twitchings. In the early stages it may be difficult to say with what complaint we have to deal.

Acute anterior cornual myelitis or infantile spinal paralysis.—Acute disease of the anterior grey cornua is known as acute anterior polio-myelitis, or anterior cornual myelitis, or acute atrophic paralysis, or sometimes simply as infantile paralysis (spinal form). It differs from the forms of myelitis we have just considered in that it is selective, limiting itself to the anterior horns of the grey matter. But probably this limitation is due to the fact that these parts have a particularly free blood-supply; for there is reason to think (from the few examinations that have been made at an early stage of the disease) that the process is one of acute inflammation. It is common in early childhood; it occurs, but with far less frequency, in adults. Its causes are practically unknown. Generally the child is taken suddenly ill, with more or less fever, vomiting, and perhaps convulsions. It is put to bed, and perhaps no diagnosis can be made at first. In a day or two probably the paralysis will be noticed. The extent of it varies in different cases. One or more limbs may be involved, or even the trunk muscles, so that the child cannot sit up or hold its head up. The face and tongue are scarcely ever affected. But the maximum is reached either at once or within a very short time. Then begins what may be called the "regressive stage." Motor power gradually returns in one part after another till the

paralysis is narrowed down, it may be, to one or more limbs, or even to a part of one limb. The distribution *quoad* the limbs may vary. One leg, one arm, both legs, arm and leg of same side, may be affected. Within the limbs certain groups of muscles may be picked out in a manner that is suggestive of spinal cord disease; for instance, in the upper limb the muscles of hand and forearm, or the deltoid spinati and flexors of elbow-joint; in the lower limb, the anterior tibial group, or the calf muscles, or the thigh muscles. Within a fortnight, or earlier, the electro-contractility begins to alter,* and next the muscles waste. This wasting marks out to the eye the localised character of the disease. The bladder and rectum usually escape. The tendon-reactions and the skin reflexes (so far as they require the co-operation of the paralysed muscles) are abolished. But sensation remains perfectly normal.

There comes a time when the "regression" of the paralysis ceases. Even in mild cases it is probable that some permanent disability will be left. In severer cases, as time goes on, not only do the muscles waste, but the bones of the affected limb are checked in their development, so that it becomes shorter than its fellow. Vaso-motor paralysis is seen, the skin becomes cold, purplish, and subject to chilblains. Deformities of the foot arise, mainly from the unbalanced action of non-paralysed muscles; this disease is a fruitful source of club-foot.

As to treatment, the damage to the cord will have occurred, most likely, before the diagnostic symptom of paralysis has shown itself. We cannot repair the destruction of cells; but we try to facilitate recovery by maintaining the nutrition of the paralysed parts. The limb must be kept warm from the first, and after three or four weeks massage and electricity should be

* This is a point useful in prognosis. If after a fortnight a muscle retains its farado-contractility, its spinal centre is not destroyed, and we may count on its recovery.

regularly applied. The constant current should be used; both because it dilates the blood-vessels of the part, and because the muscles will contract under it and not under faradism. Even at a late stage such measures will do some good. The child should be encouraged to use the limb; and if deformities have arisen, tenotomies may be practised, and surgical boots applied, to enable him to use it the better.

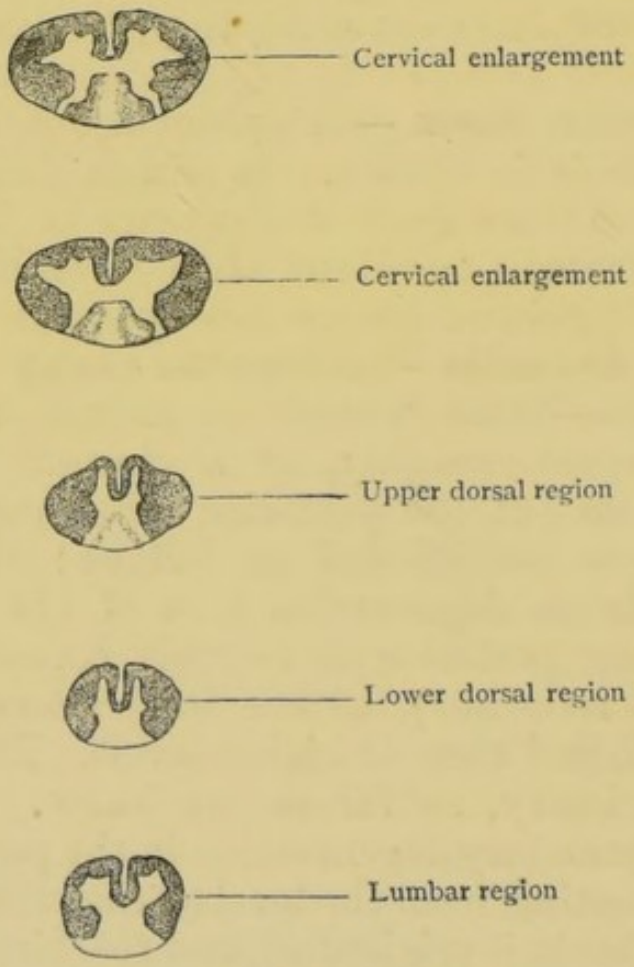
The same disease occurs, though exceptionally, in adults.

Subacute form.—A subacute form of anterior polio-myelitis is known. It differs from the acute form in the more gradual character of its onset. It is more common in adults than in children. It has to be distinguished from a multiple neuritis.

Tabes dorsalis—locomotor ataxy—posterior sclerosis.—Tabes dorsalis or locomotor ataxy consists, to speak roughly, of a chronic degeneration or "sclerosis" of the posterior columns of the cord. These parts are affected as follows:—The postero-lateral column degenerates first at the point where the entering posterior nerve-roots course through it (posterior root-zone), in the lumbo-dorsal region to begin with, and then at higher levels. This degeneration is primary, so far as we know. Next there occurs a secondary degeneration in the postero-median column, starting from the level of the primary degeneration (wherein it originates), and reaching up through the whole length of the cord. Thus, in the lower dorsal region the whole sectional area of the posterior columns may be involved; in the cervical region only the postero-median part of it (columns of Goll). In rare instances the primary degeneration begins in the cervical region of the cord, and not in the lower dorsal region. The posterior nerve-roots are often diseased as well; indeed, it may be argued that the primary disease begins here. Other parts of the cord may be affected: Lissauer's columns frequently, and at an early stage, it is said; the vesicular columns of Clarke,

and (probably in connection with them) the direct cerebellar tract; also the antero-lateral ascending tract. All these parts, it will be noted, consist of centripetal fibres. The degeneration may spread from the posterior columns to the posterior horns of grey matter. In some rare cases the anterior cornual

FIG, 58.



Tabes dorsalis.

In the two lowest sections the whole posterior columns are diseased; in the centre section nearly the whole of them; in the two upper sections the disease lies chiefly or solely in the postero-median columns.

cells also degenerate. Lastly, recent researches show that a degeneration of peripheral nerve-fibres is by no means uncommon in tabes.

There are few diseases in which such a variety of seemingly disconnected symptoms may arise. We will first describe an orthodox type of case, and then

give an account of the accessory (though often more prominent) symptoms.

Typical form.—The patient is usually a man of middle age, or past it; and he will complain of difficulty in walking. His gait is unsteady, especially as he turns; he looks at his feet as he walks to prevent falling. In advanced cases (chiefly where sensation is much impaired) there is a peculiar flourishing of the feet; they are lifted higher than necessary, thrown forwards and outwards, and brought down on the heel with a stamp. (The patient has been said to "walk like a cock.") When made to stand with his feet together, he is unsteady; and when in addition he shuts his eyes, he will fall (Romberg's symptom). These symptoms are due not so much to loss of power, as to want of harmony in the muscular actions necessary for walking, and hence the name "locomotor ataxia." Nevertheless, in many cases there is actual loss of power, and in some this becomes a prominent symptom. The inco-ordination may affect the hands, and then there is an awkwardness in fine movements, such as picking up a pin, fastening buttons, &c., and he cannot guide his fingers without looking at them. If told to touch his nose with his finger quickly, he may do so; but if he repeats the attempt with his eyes shut, he goes astray. The patient has, or has had at a previous date, severe pains. These may be of an aching, boring kind, or (what is more characteristic) of the kind known as "lightning pains"—sharp lancinating pains, of momentary duration, so far as each pain is concerned, but occurring in bouts that may last for a day or several days. Their usual seat is the legs, but they may appear in very various parts. They are not, like the pains of neuralgia or neuritis, limited to the course of any one nerve. They are ascribed to irritation of the posterior nerve-roots as they pass into the cord. In addition, there is anæsthesia, or more commonly a blunting of sensation in the legs, in consequence of which the patient feels as if he were walking on wool

or on a thick carpet. One or all of the modes of sensation (enumerated on p. 88) may be affected. It is common to find loss of muscular sense; the patient does not know how his legs are placed unless he looks at them. It is also common to find delay in the transmission of painful sensations. Often there is a girdle-pain. Examination will show the presence of two very important symptoms, not known to the patient himself:

(1) That the patellar tendon-reaction is completely absent, although the nutrition of the quadriceps cruris and its electro-contractility is normal (Westphal's sign).

(2) That the pupils, which probably are small in size, fail to contract under the stimulus of light, though they contract well during convergence and accommodation (reflex iridoplegia, or Argyll-Robertson's sign).

Stages.—Such is the condition presented by a somewhat advanced stage of the disease. Patients often come to us at earlier stages, either when the gait is merely unsteady, or even when there is no apparent difficulty of walking. This latter has been called the "præataxic stage." The symptoms which then attract the patient's attention are, first, the pains; secondly, some of the accessory symptoms, of which we shall presently speak. The commonest of these are—transitory paralysis of the ocular muscles, giving rise to squint or ptosis, or difficulties of micturition, usually consisting of a lack of complete control over the sphincter of the bladder. For diagnosis we must look for three main symptoms: (1) the characteristic pains, (2) the absence of patellar tendon reflex, (3) the "Argyll-Robertson pupils." Where all three are present, the diagnosis of tabes may be made; when one or other fails (it is usually the pupil-sign), we can only surmise, and confirm our surmise by the presence of accessory symptoms.

The disease is progressive as a rule; from the præ-

ataxic stage it passes to that of complete ataxia, and then to the paralytic stage. In this last stage the patient becomes unable to stand or walk, and the advent of cystitis or of bedsores may end his days. Nevertheless, the progress of the disease is in most cases very slow, being measured by years, and it may stop short at any stage. Even when it does not so stop, some early symptoms, such as the pains, often pass off as the ataxia develops.

Accessory and anomalous symptoms.—We must now turn to the accessory symptoms. These are very various, and often so obtrusive as to mask the real character of the disease. As to the organs of special sense, we have already mentioned the condition of the pupil and ocular paralysis. Sometimes there is complete ophthalmoplegia. Optic atrophy is a tolerably common event, and may be one of the first symptoms. It produces limitations of the field of vision, colour-blindness, amaurosis, and finally complete blindness. Many patients with tabes will be found, on examination, to hear imperfectly. The reason may be doubtful, for they often have an opaque and thickened membrana tympani, indicating a chronic dry catarrh of the tympanum; but in some cases, at any rate, the perosseous hearing is imperfect, and the mischief presumably nervous. Vertigo, another symptom of this disease, may be due either to the aural condition or to ocular paralysis, or to the loss of muscular sense.

The pains, as we have said, may occur in other places than the legs. With the pains, and resembling them in their paroxysmal character, are associated disturbances of various internal organs known as "crises." A gastric crisis may begin with lightning pains between the scapulæ, which work their way round to the epigastrium; then vomiting sets in, and for several days it may be the patient vomits after taking food or drink, or even without this. A "rectal crisis" consists of tenesmus and pain at the lower bowel, followed, it may be, by diarrhœa, or even bloody

stools. Similarly urethral, bronchial, and pharyngeal crises have been described. As to the vascular system, an undue frequency of pulse has been noted; further, there is often valvular (usually aortic) disease. Ecchymoses, sometimes large, sometimes in the form of purpuric spots, have been seen, particularly after a bout of pains. Difficulties of micturition we have noticed as a common early symptom; in the genital system there has been observed, in the early stages, increase of sexual desire with a capacity for frequent satisfaction not evinced in healthy men; later, sexual impotence.

The paralytic symptoms constitute an interesting group. Many of these occur early and are transitory; ptosis and ocular paralysis are the commonest; hemiplegia and paraplegia are also seen; their anatomical basis we do not know. An important form of paralysis is bilateral paralysis of the abductors of the larynx (*vide* p. 157); this also may occur quite early in the disease. Paralysis with muscular wasting is seen sometimes; as, for instance, in one-half of the tongue, or in the limbs. This may either be due to a neuritis, or to spread of the central degeneration into the motor cells of the cord or medulla. Under trophic lesions we may reckon:

(1) Perforating ulcer of the foot; this is generally associated with anæsthesia, or at least some sensory disturbance of the part; it may recover, sometimes quite rapidly.

(2) Disease of bones and joints; the bones become fragile and liable to fracture from very slight causes, or a joint becomes suddenly swollen, the swelling extending, it may be, into the adjacent tissues of the limb. The swelling may subside without much further damage, or the disease may go on to complete destruction of the joint, leaving the limb "like a flail."

(3) The nails and teeth may come out.

Lastly, we must add that though mental symptoms

form ordinarily no part of tabes, sometimes a state of mental exaltation arises, and the patient may pass into a condition resembling general paralysis of the insane. This is one way in which tabes may terminate.

As to the causation of tabes, syphilis is so frequent an antecedent, that it is commonly thought to be at least a predisposing cause, although we do not understand its mode of action. Exposure to cold, and injury to the back or to other parts, have also been alleged as causes. The disease is much commoner in men than in women.

Treatment is unsatisfactory, as is the case with most degenerative nervous diseases. As to drugs, mercury and iodide of potassium have been used, but without the good results that would be wrought on a syphilitic inflammation or a gumma. Nevertheless, iodide of potassium is certainly useful. Nitrate of silver is recommended, and arsenic. For relief of the lightning pains antifebrin and antipyrin are very prompt and efficient remedies. A strong constant current applied from the spine to the feet appears to do good to some cases. Faradisation of the spine with the wire-brush has been employed abroad. Suspension treatment appears to have had its day. In spite of apparent inefficacy of treatment, the fact that pauses and even arrests of the disease may occur naturally should lead us to persevere, in the hope that we may find a means of producing arrest artificially.

Lateral sclerosis, or spastic paraplegia.—Just as ataxia, with abolition of tendon reflexes, points to disease of the posterior columns of the cord (or more strictly to disease of the external part of these columns), so there are symptoms which point to disease of the lateral columns, or, strictly speaking, to that part of them known as the pyramidal tracts. These symptoms, as we have already mentioned, are loss of power in the limbs, with increase of tendon reflexes, and eventually rigidity. This condition is known, with reference to the lower limbs, as "spastic paraplegia."

There is increasing weakness of the legs; in walking they are lifted insufficiently, so that the toes do not clear the ground, and the toes of the boots wear out; as he gets worse, the patient has to walk very slowly, standing on the one leg with his body leaning forward, and dragging the other laboriously forward; finally, the increasing weakness and rigidity prevent his walking at all, and his lower limbs become fixed, probably in the position of extension, with internal rotation of the thigh, though they may assume the (still more inconvenient) position of flexion at the hip and knee. Increase of tendon-reaction and ankle-clonus are marked, till the access of extreme rigidity prevents the production of them. The gradual onset of spastic paraplegia followed by the development of similar phenomena in the arms, if unaccompanied by other spinal symptoms, is said (by eminent authorities) to be characteristic of a primary degeneration of the lateral columns (pyramidal tracts), just as locomotor ataxy is characteristic of primary degeneration of the posterior columns. And to such disease have been given the various names, "idiopathic lateral sclerosis," "spasmodic tabes," or simply "spastic paraplegia." There are reasons, however, why we should be cautious in diagnosing pure "lateral sclerosis" during life. First, because the mere symptom of spastic paraplegia may occur in the course of, or even as the main manifestation of, several other diseases, such as transverse myelitis, or compression myelitis, of the dorsal cord; disseminated sclerosis; amyotrophic lateral sclerosis (*vide infra*); hysteria. We must therefore exclude all symptoms referable to other parts than the pyramidal tracts, mainly, that is, sensory symptoms, affections of the sphincters, trophic symptoms (viz., bedsores, muscular atrophy with reaction of degeneration), tremors, cerebral symptoms; and must be certain (which is no easy matter) that we have to do with an unalloyed, slowly progressive, spastic paralysis of the limbs. Secondly, we must remember that although spastic paralysis of

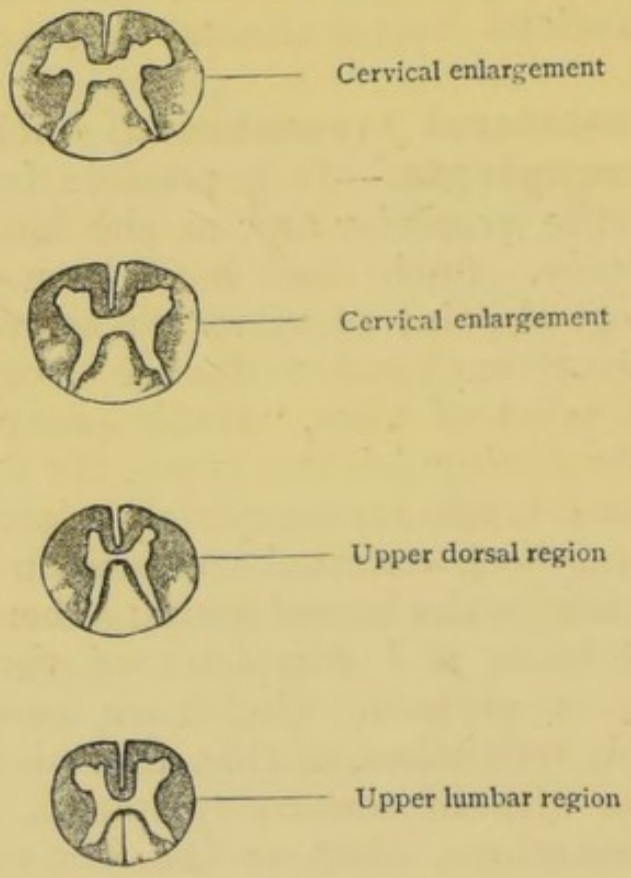
one kind or another is extremely common during life, an idiopathic lateral sclerosis is extremely rare post-mortem, so much so that its existence (except as an accessory to a more general nervous degeneration, such as general paralysis, or in combined degenerations of various spinal tracts) has been actually denied.

Postero-lateral (combined) sclerosis, or ataxic paraplegia.—It is possible for disease to develop in the posterior and in the lateral columns simultaneously. Such cases have been called (from the pathological point of view) "postero-lateral sclerosis," or "combined system disease"; or again, from the clinical point of view, "ataxic paraplegia." We may roughly distinguish two types, the first in which the posterior columns are completely sclerosed, the disease involving both external and median parts of the column; while in the lateral columns there is sclerosis (but less intense and complete) of the pyramidal tracts. It is probable that such cases clinically will resemble true tabes, in that there is inco-ordination, with or without sensory symptoms, and absence of tendon-reactions. But to the inco-ordination is added loss of power, and the legs are dragged in walking, not tossed about. In the second type the lateral sclerosis (degeneration of pyramidal tracts) preponderates, while the posterior sclerosis is incomplete. Then the symptoms are mainly those of spastic paraplegia with exaggeration of tendon-reactions, while inco-ordination, sensory symptoms, bladder troubles, &c., may be present in just such degree as to indicate the mixed nature of the affection.

The clinical characters of a postero-lateral sclerosis consist therefore in a combination of the symptoms producible by disease of the parts of the cord affected; excepting perhaps in the behaviour of the tendon-reactions. This (to speak only of the knee-jerk) is seemingly regulated as follows. If, in the lumbar and lower dorsal region, the zone of entry of the pos-

terior nerve-roots (*vide* p. 16, footnote) is sclerosed, then the knee-jerk is lost, however great the lateral sclerosis; if, however, this part is healthy, the knee-

FIG. 59.



Postero-lateral sclerosis.

The *posterior* columns are completely degenerated, excepting in (1) a narrow zone abutting on the grey matter; (2) a small triangular area close to the apex of the posterior cornu, in the upper lumbar region. (This latter corresponds with the zone of entry of the posterior nerve-roots, and since it was healthy the knee-jerks were in this case retained.)

The *lateral* columns show degeneration affecting in an irregular fashion (1) the cerebellar tracts chiefly in the cervical region. (2) The crossed pyramidal tract chiefly in the dorsal region.

The *direct* pyramidal tract of the right side is also affected in the three upper sections.

jerk remains, and is even exaggerated owing to the lateral sclerosis.

The commonest form of postero-lateral sclerosis appears to be that in which the gait is partly un-

steady, partly dragging, and the tendon-reactions are excessive; sensory symptoms, and particularly lightning pains, are slight or absent; the pupils act normally, or are occasionally of the Argyll-Robertson type; a history of syphilis is less frequent (according to Dr. Gowers) than in uncomplicated tabes.

In addition to the degeneration of the posterior columns and of the pyramidal tracts, the cerebellar and antero-lateral tracts (one or both) may be diseased; so that there is a combined sclerosis of several systems of fibres. The degeneration of these tracts makes no difference to the symptoms so far as we know. Or it may be that the lines of the diseases do not completely identify themselves with the systems of fibres, as known to us.

A combined sclerosis is not unfrequent in the spinal cords of general paralytics.

Hereditary ataxia, or Friedreich's disease.—There is a form of ataxia which differs in several ways from ordinary tabes. It generally occurs (unlike tabes) in several members of a family; mostly in collateral members—brothers and sisters, or cousins—sometimes among the ancestry too. It attacks the patients during youth, or even during childhood. The earliest symptoms are not, as in tabes, pains or anomalous paralyses, but a simple progressive inco-ordination of the lower limbs, trunk, arms. There are unsteady, irregular movements and swayings, which may look like mild chorea. The gait is unsteady and lurching. The tendon-reactions, as in tabes, are almost always abolished. The pupils, on the other hand, are quite normal.

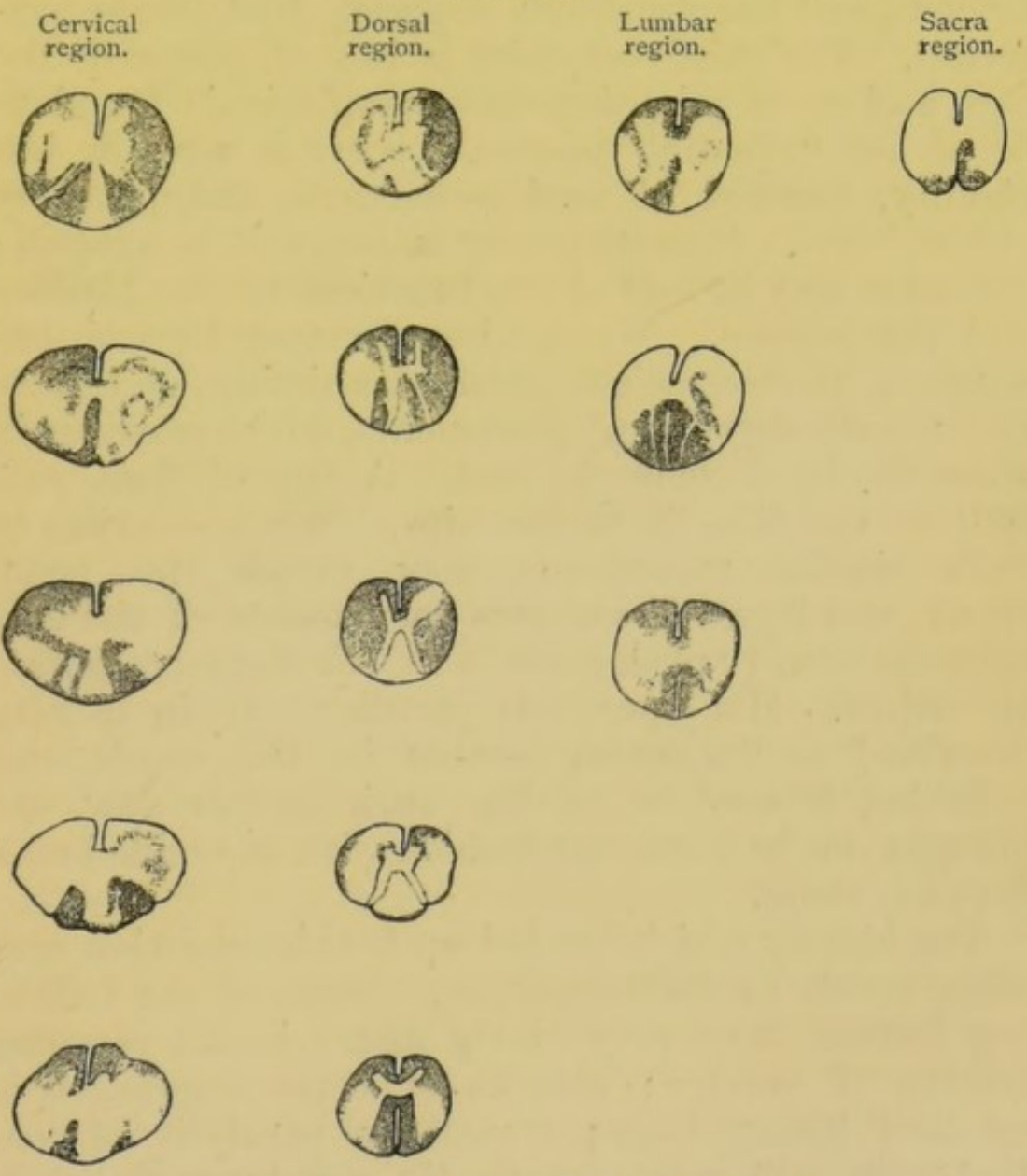
As the disease progresses other phenomena develop. The speech becomes peculiar, blurred and drawling, with elision of syllables. Nystagmus appears, though often not till late. The spinal column becomes curved, the foot becomes humpy-looking, and the toes turn up. The lower limbs, from being simply ataxic, become actually weak, and a certain amount of rigidity may

develop. The face becomes expressionless and feeble-looking, even though intelligence is unimpaired. The disease is slowly progressive; and probably most cases are terminated by intercurrent disorders. Acute specific diseases appear to have a particularly deleterious effect on such patients. Not many cases have been investigated post-mortem, but it would appear from them that the disease consists in a "combined system degeneration," in which the posterior sclerosis forms the earliest and most completely developed factor. Hence the rule that the tendon-reactions are abolished, but in some very rare cases they may be retained or even exaggerated.

Disseminated or insular sclerosis, "sclerose en plaques." — Disseminated sclerosis is a disease that affects the brain, and sometimes the nerve-trunks, as well as the cord. But since spinal symptoms are almost always present, and since they contrast in many ways with those of tabes, we shall consider it here. Anatomically it is a "diffuse" lesion, characterised by the presence of islets or patches of disease irregularly scattered through the nervous system, and involving indiscriminately the various tracts and systems of grey or white matter. These patches are dull pink, translucent, and raised, or grey and contracted, according as they are recent or old. Within these patches there is an overgrowth of neuroglia and degeneration of nerve-tissue. As the neuroglia becomes thickened, and gradually converted into a fibrillar mesh-work, so does the myeline of the white fibres perish and become absorbed. The axis cylinders, according to most authorities, become at first enlarged; later on they may perish, but, as a rule, some of them are said to persist even in patches of advanced sclerosis. This persistence of the axis cylinders has been used to explain another fact—viz., that secondary degeneration does not take place with the same regularity as in other forms of myelitis. (Thus, when a patch of disseminated sclerosis involves

the whole area, say of the cervical region, we may still find the pyramidal tracts healthy at a lower level of the cord.) When the disease affects the grey matter there may be degeneration of the ganglion-cells, but these, too, appear able to hold out in the presence of considerable disease in their neighbourhood.

FIG. 60.



Disseminated sclerosis of the spinal cord.

Little is known as to its causation. Unlike tabes, it affects women oftener than men, and commences comparatively early in life, usually before thirty. Exposure to cold, injury, acute infectious diseases, &c., have been observed to precede its attack; and

still more frequently mental factors, such as fright, anxiety and worry, chagrin and disappointments. Syphilis has no relation to this disease.

Typical case.—A classical case of disseminated sclerosis presents a sufficiently characteristic picture. The patient is most frequently a woman of middle age, or younger. Her aspect may be vacant and fatuous, and her behaviour suggests that she is hysterical. The chief complaint is loss of power in the legs, and on examination this paraplegia is found to be of the "spastic" type—*i.e.*, there is more or less rigidity, increase of tendon-reactions, and probably ankle-clonus. Sensation may or may not be affected; the same may be said of the functions of the bladder and the rectum. When she attempts to use her hands a tremor seizes them, consisting of coarse, imperfectly rhythmical movements, which cease again when the hand comes to rest. A cup of fluid gets spilt as she lifts it to her lips. When she rises to walk similar movements may attack the head, trunk, and legs. Tremulous movements of the eyeballs are seen ("nystagmus") as she fixes or follows an object. Her speech is peculiar. It is usually described as "scanning"—that is, the words and syllables, instead of having their proper flow, are brought out in a staccato fashion with an undue break between them.

The history which has led up to this condition may differ much in different cases. Some of the following factors may very likely figure in it: repeated attacks of vertigo; attacks of coma suggestive of cerebral hæmorrhage; transitory paralysis of one limb; or, still more frequently, numbness or loss of sensation in various parts of the body and limbs, which has also passed away; ocular paralysis, and temporary blindness. After these premonitory symptoms, or along with them, the paraplegia has gradually and permanently developed.

The prognosis, when the disease is well established,

is not good. Probably the paraplegia will become absolute, and the contractures of the legs extreme, and the patient, thoroughly bedridden, will die of cystitis, bedsores, or intercurrent disease. Or bulbar symptoms may supervene, and dyspnœa or difficulty of deglutition may prove fatal. Or she may die in one of the apoplectic attacks, to which we have alluded. But it is important to know, on the other hand, that the course of the disease may be interrupted by remissions of the symptoms so complete as to suggest a cure. Such remissions have even been observed to last two or three years. Their occurrence must make us feel uncertain of prognosis and diagnosis too.

We know of no remedy as yet that can be relied on to cure this disease.

Anomalous cases.—The diagnosis of disseminated sclerosis where it does not conform to, or has not yet developed into, the type we have sketched above, is a matter of great difficulty. The islets of disease may be placed anywhere, and hence the symptoms (even such as are directly dependent upon the organic lesions) may be various and variously grouped. The tremor, the nystagmus, and the affection of speech are perhaps the most characteristic symptoms, and it is for these especially that we should look out in doubtful cases. But they may appear only at a late period, or not at all. The condition of the eyes deserves study. Nystagmus, as we said, is common here. If we except Friedreich's ataxia, certain cases of peripheral neuritis, and growths in the upper part of the pons which gradually affect the ocular nuclei and nerve-roots, it is an uncommon symptom in other nervous diseases. Paralysis of ocular movements occur both in this disease and in tabes; Charcot affirms that in tabes such paralyses generally have the character of nerve-trunk paralysis, in disseminated sclerosis that of nuclear paralysis; in other words, they are paralyses of conjugate movements (*vide* p. 119). As to the pupils, they act normally, as a rule, to light

and accommodation, unlike the pupils of tabes. Affections of vision occur which are rather puzzling. The sight of one eye may be impaired and perhaps regained again, without any definite ophthalmoscopic change being seen; or there may be optic atrophy of various degrees. Such atrophy, however, is not necessarily progressive, as in tabes; in fact, there are very few cases recorded where total blindness has resulted from this cause. In some rare instances optic neuritis has been observed.

The symptoms may be so scanty, while the disease is yet developing, as to make a definite diagnosis almost impossible. There may be little more than a paraplegia, indistinguishable from that of chronic myelitis, or from hysterical paralysis. Hemiplegia may be the leading symptom; this may be of several kinds; either a hemiplegic tremor, or a transient hemiplegia following a quasi-apoplectic seizure, or a permanent hemiplegia which is chiefly remarkable by its gradual onset. Or the disease may begin with inco-ordination of gait and lightning pains, or gastric crises, like those of tabes. Ataxy with paresis of the lower limbs, affection of speech, and nystagmus, are symptoms which may be found both in this disease and in Friedreich's ataxy; but in the latter disease the tendon-reactions are usually abolished, and the symptoms are developed in a more definite course. Nystagmus, with paralysis of ocular movements, tremors, and weakness of the limbs, coupled with exaggeration of tendon-reactions, and possibly some difficulty of speech, may be seen in tumours high up in the pons as well as in disseminated sclerosis. But the commonest difficulty that arises is the distinction between hysterical paralysis and disseminated sclerosis, by reason of the age, sex, and general character of the patients, and the anomalous grouping and course of the symptoms, which may be much the same in both affections. Two additional statements may be made here, which will set forth this difficulty still more strikingly: first, that,

according to Charcot, sclerosis may become engrafted upon long-continued hysterical disease; secondly, that, according to Westphal, cases occur indistinguishable during life from disseminated sclerosis, and actually proving fatal, wherein after death no trace of organic lesion can be found.

General paralysis of the insane, or paralytic dementia.—General paralysis is a disease which affects both the cortex cerebri and the spinal cord. We shall notice it here, partly for the sake of comparison with the other degenerative diseases of the spinal cord, partly because the general physician has chiefly to do with it in the form of a spinal disease, or at least with that variety of it in which mental symptoms are inconspicuous. Like tabes dorsalis, it is much more common in men than in women. Overwork, the anxieties of business, over-indulgence in alcohol or in sexual intercourse, injuries to the head, are alleged causes. Syphilis, even if it do not produce the true disease, may be followed by a condition closely resembling general paralysis.

The condition of the brain in an advanced case of general paralysis has been thus described. The dura mater (and perhaps even the bones of the skull) is thickened. Beneath the dura are sometimes found exudations which have the appearance of a blood-clot. The arachnoid is milky and opaque; there is much sub-arachnoid fluid. The convolutions themselves are shrunken; the pia is tough, and when peeled off takes with it part of the surface of the convolutions, so that they have an eroded appearance. The lining membrane of the ventricles is rough and thickened. The grey matter of the convolutions is thinned, and shows an opaque outer layer with a line of congestion beneath. The minute changes appear to consist in an overgrowth of the neuroglia, and atrophy of the cells and nerve-fibres of the grey matter. Probably general paralysis should be regarded as a primary disease of the brain substance; but good authorities maintain that the disease

originates in the vessels, and that the nervous changes are secondary to repeated congestion. In the spinal cord there may be degenerations, like those we have been considering, either of the pyramidal tracts or of the posterior columns.

Mental symptoms.—The most typical cases of the disease exhibit a combination of mental and bodily symptoms, from which has originated the name "general paralysis of the insane." The insanity will most likely manifest itself in some such ways as these. The patient's habits and character undergo a change; he becomes careless and incapable in business matters, and at home egotistical, conceited, extravagant, perhaps immoral. He may embark in all sorts of foolish schemes and expenditures before it is recognised that he is mad. Then actual delusions appear, of the "expansive" or grandiose character. He has millions of money, hundreds of wives, and has the strength of many men, &c. He will ramble on in his talk from one scheme to another, or will hurry hither and thither in pursuit of imaginary business. He is not violent, as a rule, unless thwarted or contradicted. After some time this state of exaltation begins to give way to a state of stupid contentment, though the patient will still be found to have delusions when questioned. The paralytic symptoms have also in the meantime been making way. Finally, he will lapse into complete dementia, and mind and body alike failing he will die, probably in some three or four years from the commencement of his illness.

Physical symptoms.—Bodily symptoms accompany these mental changes, and they are important to recognise; for there is a large class of cases in which the delusions and mania do not develop. Such patients are not lunatics, but, equally with those who are, they undergo a process of mental enfeeblement, and end in the same state of "paralytic dementia."

One of the most characteristic and earliest physical symptoms relates to the speech. As the patient

begins a sentence he hesitates slightly, and there are fine tremulous movements of the lips. Towards the end of a long and difficult sentence or word he slurs the syllables and perhaps drops some out. "National Hospital for the Paralysed and Epileptic" becomes "N-national Hospital for the Parlysed and Etlettic." The speech differs therefore from the "scanning" of disseminated sclerosis, and from the drawl of patients with right hemiplegia; in later stages, however, the general paralytic lapses into a mere drawl. Usually when he shows his teeth or puts his tongue out, his lips and tongue will shake, perhaps very much. His hands shake as he uses them; but it is a fine tremor, not the coarse vibration of disseminated sclerosis. There is a feebleness of the limbs, and it is generally for this that the patient (when not mentally affected) seeks advice. It will be found on examination either that there is mere motor weakness with exaggeration of tendon-reactions, or that there is an ataxic condition with absence of tendon-reactions, or that these two states are more or less combined. These facts stand in relation to the presence of lateral or posterior sclerosis, or combined postero-lateral sclerosis—all of which have been found in the spinal cord post-mortem. As to the eyes, there is no nystagmus, and the ocular movements are normal; but the pupils may be unequal, and they often exhibit reflex irido-plegia, contracting under light but not to accommodation. Optic atrophy may occur, but it is not very common. It is obvious that an ataxic gait, with absence of tendon-reactions, and pupils of the type just mentioned, will suggest the diagnosis of tabes dorsalis, but general paralysis should be suspected where there is mental enfeeblement, where the speech is affected, where the ataxia or feebleness has developed rapidly, and where there has been an absence of lightning pains. Nevertheless, though the two diseases must be distinguished, they have evidently a close relation to each other. For, as the general

paralytic may have symptoms of tabes, and actual posterior sclerosis, so may the patient, who for many years has had tabes, develop the mental and bodily symptoms of general paralysis (*vide* p. 213). Another set of symptoms remains to be noticed; they often occur early in general paralysis and are always of importance. These are the so-called "congestive" attacks. The patient may become suddenly comatose, as if from cerebral hæmorrhage. Or he may be attacked with convulsions, either general convulsions rapidly repeated, constituting a "status epilepticus" (*vide* p. 289), or convulsions limited to one arm, leg, or one side of the body. Or he may become for a time hemiplegic, with or without convulsions or coma. Such symptoms may occur indeed in any degenerative nervous disease (we have already noticed their possible occurrence in disseminated sclerosis), but they are particularly frequent in general paralysis. Such attacks usually leave the patient worse in every way; or he may die in them. Indeed, it is said that a fatal attack of this nature may be the very first symptom of the disease.

General paralysis is a very fatal disease, usually terminating within three or four years from its commencement. The course of those cases where the delusions are predominant can evidently be watched only in asylums, and for a full description of them we must refer to text-books on lunacy. It is important to be aware of the existence of the other class of cases, which show themselves simply by progressive paralysis and mental enfeeblement, partly because delusions may at any time appear, partly because the fatal termination is the same. Nevertheless, there may be remissions in some cases, and others may run an abnormally slow course. We know of no drugs that will cure the disease; though, where it can be connected with syphilis, iodide of potassium in large doses has appeared to do good.

Progressive muscular atrophies.—Muscular

atrophy is a symptom that occurs in many nervous diseases. In its most marked form, it indicates a lesion of the lowest parts of the motor tract—that is to say, either the large cells of the anterior cornua, or the nerves that connect these cells with their muscles, or the muscles themselves. Disease of the motor apparatus above the level of these cells—viz., of the cerebrum and pyramidal tracts—causes as a broad rule not muscular wasting, but the opposite phenomenon, muscular rigidity. (With the anterior cornual cells of the cord we include their homologues, the motor nuclei of the medulla.)

Under the heads of infantile spinal paralysis (acute anterior cornual myelitis), diffuse myelitis, and neuritis, we have described the diseases which commonly are the cause of acute muscular atrophy; we have now to describe some disease in which the chief symptom is a chronic and progressive atrophy. Some of these undoubtedly have their primary seat in the spinal cord, others in the muscular tissue itself, some perhaps in the nerves.

Amyo-trophic lateral sclerosis (Charcot's type).—Amyo-trophic lateral sclerosis is the name given by Charcot to a disease characterised anatomically by two main factors—degeneration of the white fibres forming the pyramidal tracts, and degeneration of the anterior cornual cells. The clinical correlatives of these anatomical factors are spastic paralysis, and paralysis with atrophy of muscle. Let us consider a typical case. The age of the patient, who may be man or woman, ranges usually from twenty-five to fifty years. The first symptom is weakness of the hands. This may be accompanied by numbness, or other disagreeable sensations, or even by distinct pains, but such sensory phenomena are not marked nor permanent. Soon the intrinsic muscles of the hands are found to be wasting; then the forearms become small, flabby, and waste also. The weakness and wasting thus spread through the whole upper limbs.

If we examine the patient at a comparatively early stage, we shall probably find the following condition: marked wasting of hand-muscles, forearms small and flabby if not wasted, indications of weakness and atony in upper arm and scapular muscles. The muscles which are beginning to waste show well-marked intermittent twitchings of their muscular bundles (fascicular tremors, *vide* p. 92).

Electrical examination generally shows, in muscles where the wasting is declared but not extreme, an incomplete reaction of degeneration—*i.e.*, farado-contractility retained, but galvanic formula, and perhaps character of the muscle twitch, altered. But in some muscles the reactions may be normal, and in extremely wasted muscles the electro-contractility may have disappeared. Lastly, and this is most important, this atrophic paralysis is accompanied by increase in the tendon-reactions. This exaggeration may be observed in the arms, provided the wasting has not advanced too far; and still better in the legs, even before the patient complains of weakness here. It is in fact the herald of the second stage of the disease.

This second stage is marked by the access of paraplegia. The paraplegia is at first due to the lateral sclerosis, and is therefore of the spastic type; the feet are dragged in walking, there is increased knee-jerk, and probably ankle-clonus. There is no sensory paralysis, and no paralysis of the sphincters; no bedsores. But there will not be an extreme degree of rigidity, for the reason that before the lateral sclerosis has had time to become extreme, degeneration of the motor-cells will begin in the lumbar enlargement, introducing the opposed condition of flaccidity. Thus there is found in the lower limbs first rigidity more or less, then a kind of compromise which has been called "flexibilitas cerea"; lastly, flaccidity and atrophy, as in the arms.

In the third stage, the disease attacks the bulbar nuclei. Nasal voice, progressive difficulty of articula-

tion and of swallowing are the usual symptoms. Wasting and fibrillary twitchings are seen in the tongue, the palate moves badly, the lower part of the face becomes expressionless. Extension of the disease to the respiratory and cardiac centres may obviously induce fatal attacks of dyspnœa and of syncope, if the patient have already survived his state of paralysis and mal-nutrition, with its risks from intercurrent disorders.

The causes of the disease are practically unknown. The prognosis is bad, for the progress is more rapid than that of most spinal degenerations, and death results within three or four years, although exceptional cases may run a longer course.

A typical case is easy to recognise, but there are varieties. Thus some cases begin with bulbar symptoms, such as paralysis and wasting of the tongue; sometimes the weakness is for a time predominant in the arm and leg of one side, so as to bear a rough resemblance to cerebral hemiplegia; sometimes the paralysis of the legs may be atrophic from the first. It would appear indeed that a certain class of cases, in which post-mortem degeneration of the anterior cornual cells is found coupled with lateral sclerosis, and which therefore correspond anatomically to amyotrophic lateral sclerosis, do not exhibit spastic phenomena during life. The explanation presumably is that the cells are attacked before the pyramidal fibres. This occurs particularly when the disease begins in the bulbar nuclei. The case then exhibits paralysis with wasting of the tongue, paralysis of the lips and lower face, of the palate, and it may be of the larynx. As the disease advances, atrophy of the hand-muscles, shoulder-muscles, and other parts begins. This has been called "progressive bulbar paralysis."

The morbid anatomy consists in a degeneration of the anterior cornual cells and of the pyramidal tracts. There is disease also of the motor-nerves and of the muscles, partly secondary to that of the motor-

cells, partly it may be primary. The disease in the pyramidal tracts has been traced up into the crura cerebri, and in a few instances beyond this—viz., through the internal capsule and corona radiata even into the motor convolutions.

Chronic degeneration of anterior cornual cells (type Aran-Duchenne).—In the second form of progressive muscular atrophy, which is dependent upon spinal disease, sometimes known as the "type Aran-Duchenne," the degeneration is limited to the anterior cornual cells, the lateral columns not being affected.* Probably this form is much less common than was formerly thought. It is distinguished from amyo-trophic lateral sclerosis by the following features:

There are no spastic phenomena, so that the legs are not stiff nor the tendon-reactions exaggerated.

The wasting begins in the hands, but its method of extension is different. Instead of involving several adjacent muscles rapidly and *en masse* (as does amyo-trophic lateral sclerosis), it picks out muscles, and even parts of a muscle, one by one, and that slowly, so that it may remain for a long time localised in a few muscles.

There are involuntary muscular twitchings, but of quite small tracts of fibres (fibrillary tremors).

The paralysis does not precede or outrun the muscular atrophy as in amyo-trophic lateral sclerosis, but is proportionate to it. (Similarly it is said that, electrically, the muscular reaction becomes simply deficient in proportion as the muscle wastes, but it is probable that qualitative changes will be found with the galvanic current if carefully looked for.)

The disease proceeds very slowly and may last for

* Some authors maintain that the lateral columns become affected sooner or later in all forms of spinal amyo-trophy. In that case the anatomical distinction between this disease and amyo-trophic lateral sclerosis breaks down. But the clinical types are certainly distinct, though there are (as we have indicated in the text) sundry intermediary forms.

years. The patient may be reduced to a mere skeleton ere death ensues, from respiratory complications, or from spread of the disease to the medulla.

Peroneal type.—A third form has recently been isolated from the group of muscular atrophies, which has the following characteristics. It commences in the muscles of the lower leg, most commonly the peroneal group. From this circumstance it has been called the peroneal type of muscular atrophy (Howard Tooth). Thus, a club-foot is often the first symptom. The two legs are affected either simultaneously, or one shortly after the other. As time goes on, all the muscles below the knees are attacked, so that the leg looks shapeless and thinned. The skin may become cold and blue. As the patient is frequently a child, this condition is readily mistaken for a late stage of acute anterior cornual myelitis, unless attention be paid to the history, which is that of gradual onset. The thigh-muscles appear able to hold out against the disease for a long time, and while they do so the patellar tendon-reaction is retained. Later, it may be some two or three years later than the legs, the intrinsic muscles of the hands begin to waste, and after them the muscles of the forearms become thinned like those of the legs. From the most deeply affected muscles electro-contractility may have disappeared, in others reaction of degeneration, in various stages of completeness, has been discovered. A remarkable fact concerning this type of atrophy is that it often affects brothers and sisters of one family, and occasionally (it would seem) members of succeeding generations; in this it resembles the diseases we shall next describe. Concerning its course and ultimate conclusion, we have yet much to learn; we only know that it spreads very slowly. The morbid anatomy is as yet uncertain; most authorities believe it to be a form of neuritis. If this be so, it explains the facts noted by Charcot and Marie, that in some cases there are pains and even anæsthesia of the legs and soles.

Myopathic types (Erb).—The remaining forms of progressive atrophy are considered to be due neither to disease of the cord nor of the nerves, but of the muscle substance itself; hence they are sometimes called "myopathic" atrophies, or "muscular dystrophies." There is a form which begins in early adult life (usually before twenty), or less commonly in childhood, and which has therefore been called "juvenile muscular atrophy." It begins, not like the spinal atrophies, in the small muscles of the hands, but in the larger muscles which move the scapula and shoulder-joint, trapezius, pectoralis serratus, &c., or those of the upper arm. The deltoid is often intact (or even enlarged).* The erectores spinæ are often weak, and the muscles which pass from the pelvis to the thigh suffer. In some cases the calf-muscles may be enlarged and hard. Even the atrophied muscles have not the flaccid feel of true spinal amyo-trophy; and electrical testing shows that, though their contractility may be diminished to both currents, there is no reaction of degeneration.† Neither do the muscles show fibrillary or fascicular twitchings.† There are no spastic symptoms; the knee-jerks are never exaggerated; indeed, they gradually diminish and disappear. Such disappearance is due, not to lesion of the nervous arc, but to invasion of the quadriceps extensor cruris by the disease. Bulbar symptoms do not develop. As in the peroneal type, there is often a family proclivity to the disease. The course of the disease is slow, and may be interrupted by long stationary periods.

There is a variety of this form, distinguished as follows: it begins in childhood (hence sometimes

* If, as may be the case, the deltoid remains intact while the spinati and flexor muscles of the elbow are atrophied, this in itself suggests that the lesion is not spinal, since the nuclei for these muscles lie near together.

† These statements hold good for most myopathic cases, but are perhaps not of universal application.

called "infantile"), and the muscles of the face are affected from the first. At first sight the facial palsy is not very obvious. The lips are thick, somewhat everted and protruding; both lower face and forehead are expressionless, and move little, if at all, on laughing, &c.; the eyes cannot be completely closed. The large muscles of the shoulder girdle and limbs atrophy later.

It would appear, from such examinations as have been made of these forms of muscular atrophy, that the spinal cord and nerve are free from disease, but that the muscular tissue shows some such changes as follows: simple atrophy of some fibres, perhaps hypertrophy of others, longitudinal splitting of fibres; multiplication of the muscular nuclei, sometimes to such an extent that they invade and disorganise the muscle substance; sometimes vacuolisation of muscle-fibres; overgrowth of the interstitial tissue with multiplication of its nuclei; infiltration of the interstitial tissue with fat in some extreme cases.

Pseudo-hypertrophic paralysis.—In pseudo-hypertrophic muscular paralysis the muscles (or at least some of them) appear enlarged and feel unduly firm, but nevertheless are weakened. The anatomical changes in them consist, according to Dr. Gowers, of an overgrowth of fatty tissue, or it may be merely of connective tissue in the interstices between the muscular fibres, with simple atrophy or perhaps degeneration from pressure of these fibres. There may be no disease in the spinal cord. It is a disease that begins in childhood, as a rule, about five or six years of age; though even before this, lateness in learning to walk, or unsteadiness upon the feet, may have been observed. It is apt to run in families, and males are more often affected than females, though the disease is transmitted through the female. The calf-muscles are those in which enlargement shows itself most strikingly and constantly; the muscles of the thigh, the glutæi, of the upper arm, and (very frequently

indeed) the infra-spinatus, may also be enlarged. Other muscles may be small or congenitally absent, particularly the pectoralis major and latissimus dorsi; and in course of time the enlarged muscles may waste. The muscular weakness produces certain peculiar symptoms. In walking, the patient keeps his trunk thrown backwards, because his erectores spinæ are weak; and his trunk oscillates from side to side with each step, because his glutæi medii are weak and balance it ineffectively. He may be unable to walk upstairs. If put on the floor in the prone position and told to rise, he first pushes himself off from the floor by his hands, and, after he has thus half raised himself, completes the action by placing his hands on his thighs, and so gradually pushing himself into the erect position. Lateral curvature of the spine often ensues from weakness of the muscles. Club-foot may be produced from gradual contraction of the paralysed muscles. The patellar tendon-reaction disappears as the extensors of the knee lose power. Electrically there is gradual diminution of contractility, without reaction of degeneration.

As the disease progresses, the patient becomes quite unable to walk, and finally even to sit up. Death usually comes from intercurrent disease, especially upon the side of the lungs, usually between twelve and twenty years of age, according to Dr. Gowers, except in cases where the symptoms have shown themselves at a later age than usual, when the progress is often much slower. The same author insists on the importance of keeping the patient as long as possible from becoming bedridden, partly because muscular exercise appears to retard the disease, partly because deformities from contracture make more rapid progress when he is once laid up. Courses of gymnastics, massage, and judicious tenotomies he recommends with the view of retarding the disease.

The close relation between this and the myopathic form of muscular atrophy is evident from the following

considerations. In this disease there is apparent hypertrophy of some muscles, with deficiency of others; in that, atrophy of some muscles with (sometimes) hypertrophy of others; in both, the cord is unaffected; in both, the electrical conditions of the muscles and the condition of the tendon-reactions are the same; in both there is often found a family proclivity to the disease. Lastly, intermediate cases are known of which progressive weakness of muscle (the common feature of the two diseases), without either atrophy or hypertrophy, has been the only characteristic. Indeed, by some authors, pseudo-hypertrophic paralysis and myopathic atrophy are considered to be essentially the same disease.

Thomsen's disease, "myotonia congenita." —Thomsen's disease is a rare affection of the muscular system which, like those we have just been considering, runs in families, and develops early in life, sometimes quite in infancy. Its principal feature is this, that when the patient begins a voluntary movement, the muscles are thrown into a condition of spasm, which interferes with the movement, or may even temporarily fix him to one position. This phenomenon takes place only at the commencement of movement, and generally passes off in a few seconds. Naturally it is best shown in the muscles of the limbs, but those of the face, tongue, and eyes may be affected also. Curious effects may be produced where the spasm is extreme; thus one patient in mounting a horse had to stand with one foot in the stirrup before he could get the other foot off the ground; a second patient had attempted to strike another man, but was held fast by the spasm till his adversary knocked him down. Usually, however, a mere stiffness and awkwardness of movement is caused. The muscles are often enlarged, and firm to touch, but they are not strong in proportion to their enlargement. The effect of a blow on the muscle is to produce, not a transient contraction as in health, but a tonic contraction, lasting

several seconds, of the fibres which have been struck. Similarly, electrical stimulation of the muscle produces a tonic contraction, instead of a single spasm, in response to each shock. This is best seen with the constant current. Further, according to Erb, the application of a strong constant current without make or break produces certain rhythmical wave-like muscular contractions which proceed from the area of the kathode to that of the anode. The tendon-reactions are normal, or in some cases absent. The peculiar muscular spasm is aggravated by cold, by anxiety to hurry, and by the knowledge that other persons are observing it. The disease does not appear to endanger life or health, but only to cause inconvenience. No post-mortems have yet been made; but in excised pieces of muscle Erb has found hypertrophy of muscle-fibres with increase in the number of nuclei.

Some general remarks on the group of muscular atrophies.—Before returning to diseases of the cord and its membranes, from which we have unavoidably digressed, some general remarks may be made on the forms of muscular atrophy which we have described. Their classification into clinical types is based, it will have been noticed, on observation of the following points :—

1. Where does the atrophy begin, in the small muscles of the hand, large muscles of shoulders or pelvic girdle, facial muscles, or leg muscles ?

2. How does it subsequently spread, piecemeal or wholesale ? with a distribution that suggests a spinal origin, or that is irrespective of this ?

3. What is the physical condition of the muscles—*i.e.*, do they show fibrillary twitching or not ? are they flabby or firm ? is there electrical reaction of degeneration or not ? is the paralysis proportionate to the atrophy or in excess of it ?

4. Are other nervous phenomena present besides muscular atrophy of the limbs, such as sensory affec-

tions, rigidity, and excess of tendon-reactions, bulbar symptoms?

5. What is the age of the patient at the onset? is there a family proclivity to the disease? what is the rate at which the disease progresses?

The clinical types correspond fairly well with distinctions in morbid anatomy; thus in the type of Aran-Duchenne there is disease of the spinal motor-cells, in amyo-trophic lateral sclerosis disease of the spinal motor-fibres as well, in other forms disease of the muscles, in the peroneal type probably disease of the nerves.

Nevertheless they all have this in common, that they are degenerations of the motor system of nerve and muscle, though they may differ in the point which they attack and the course which they take. It is not surprising if such differences are not always uniform. And thus we find that in cases which correspond mainly to the "myopathic" type—*i.e.*, to primary disease of the *muscle*, features are sometimes found which are ordinarily thought to be distinctive of *spinal* disease—viz., fibrillary twitching and reaction of degeneration. And if we turn to morbid anatomy, we see that amyo-trophic lateral sclerosis (the form of muscular atrophy whereof the morbid anatomy is best known) may begin sometimes in the pyramidal tracts, sometimes in the spinal motor-cells, sometimes in those of the medulla, and that eventually it may involve the whole motor tract, from cortex cerebri to the muscles inclusive. Here therefore we have a degeneration of the motor system which may vary in its point of attack, or may perhaps attack more than one point, and which becomes rapidly generalised; in the other types we have less intense and more local degenerations, which exhibit, each of them, features that differentiate them broadly, but that do not absolutely and widely separate them.

Treatment may do good, but cannot be relied upon to produce an absolute arrest or cure. It is

doubtful whether anything can be done to stop the march of amyo-trophic lateral sclerosis. The other forms of muscular atrophy are usually treated by massage and galvanism : as drugs, strychnia, arsenic, and iodide of potassium are given. Strychnia may also be injected into the wasting muscles.

Arthritic muscular atrophy. — Atrophy of muscle may occur in connection with disease of joints. This is not simply the atrophy of disuse, for the joint disease may be slight (not incapacitating it for movement), and yet the atrophy be well marked. It is held to be due to a reflex irritation, started by the joint disease in the trophic spinal cells. Certain muscles are more readily affected than others ; thus, in disease of the shoulder-joint, the deltoid supra- and infra-spinatus waste, in disease of the hip the glutæi, in disease of the knee the quadriceps, and generally the extensors (it is said) rather than the flexors. The tendon-reactions are often increased. There is no reaction of degeneration. The joint mischief must be rectified wherever this can be done; the muscles rubbed, and strongish galvanism applied both to them and to the joint. As this form of atrophy is recoverable, or at least non-progressive, it is important to distinguish it from the more serious diseases that we have just been describing.

We must now return to disease of the cord and its membranes, to notice those which depend on other causes than simple inflammation, softening, or degeneration.

Hæmorrhage into the cord, unless as the result of inflammation, new growth, or injury, is so extremely rare that it need not detain us. Syphilis, tubercle, and new growths may affect the cord or its membranes. Of these tubercle usually occurs in the form of a meningitis, and tubercular spinal meningitis independently of cerebral meningitis, is not common.

Syphilitic affections of the cord. — Syphilis affects the cord frequently and in several ways.

(1) As a meningitis: this may be subacute and diffuse, as we have already said; there will be probably less pain than in an acute general meningitis; the periphery of the cord itself is likely to suffer permanently. Or else the meningitis may be less acute and more localised.

(2) Syphilitic arterial disease may lead to local softening; should this occur (as it most often does) in the dorsal region, there will be simply a paraplegia of sudden onset, concerning the cause of which we can only conjecture.

(3) Gummatous nodules may affect the nerve-roots, the membranes, or the cord itself; the nervous symptoms will point to gradually developing disease of these parts: the syphilitic nature of them is inferred from general considerations, to which we shall recur when we discuss cerebral syphilis.

A hemiparaplegia (*vide* p. 82) is very likely to be syphilitic, since it requires a small lesion limited to one side of the cord, and a gumma springing from the pia mater perhaps fulfils these conditions best. But even an ordinary paraplegia, where other causes can be fairly excluded, is not unlikely to be syphilitic, if we look merely to the comparative frequency of syphilitic nervous lesions. On the other hand, hereditary syphilis is said to affect the cord very rarely indeed.

Tumours of the cord and membranes.—A new growth upon the spinal membranes produces at first symptoms referable to the nerve-roots which it irritates or involves. These consist of sensory and motor irritation, followed by sensory and motor paralysis in the districts supplied by these roots. Thus, when the dorsal region of the cord is affected, the chief symptom will be pain around the trunk, easily mistaken for pleurisy, rheumatism, intercostal neuralgia, &c. If the disease be opposite the cervical or lumbar enlargement there are pains in the limbs, and perhaps muscular spasms, followed by paralysis and wasting. In addition to such "excentric" pains, there may very

likely be pain and tenderness at the seat of the disease. Afterwards, as the cord itself becomes damaged by the invasion or pressure of the growth, paraplegia with the other symptoms of transverse myelitis follows. We surmise that the disease is a new growth when such a train of symptoms has arisen without obvious cause, and advances steadily uninfluenced by treatment (especially by antisyphilitic treatment, which should always be fully tried), and when caries of the vertebræ, syphilis, and other such sources of disease can be fairly excluded.

New growths may also originate within the substance of the cord. These will give rise to the symptoms of a myelitis (paralysis with wasting of the muscles supplied from the affected part, followed by paralysis with rigidity of those supplied from the parts below); but these symptoms will have originated without apparent cause, and will have developed (in most cases) gradually, and will show signs of constant extension along the length of the cord. Moreover, the disease is characterised by pains much more severe than in simple myelitis, and these not merely at points corresponding to the upper level of the disease (root-pains), but in all the paralysed limbs. Probably these pains are caused both by irritation of the intra-spinal nerve-roots, and by gradual spread of the growth to the membranes.

It is needless to add that the diagnosis of intra-spinal tumour will be greatly aided by the presence of a new growth elsewhere, or by the history that such an one has been removed ; but more often than not the internal tumour stands alone. Nothing can be done medically for such a case, except to treat symptoms ; but a growth, capable of being accurately localised, and limited to the membranes, has been successfully removed by operation (Gowers and Horsley).

Cavities in the cord—hydro-myelia, syringo-myelia.—Dilatations of the central canal of the cord, or canals which have closed imperfectly, are occasion-

ally found in the bodies of young subjects, without having given rise to any definite symptoms during life. To simple dilatations of the canal with fluid, the name hydro-myelia has been applied; indicating the analogy of this spinal condition to the condition of the cerebral ventricles known as hydrocephalus.

In adults, the spinal cord sometimes exhibits a longitudinal cavity or cavities. This condition has been called "syringo-myelia" (*syrinx*, a pipe). There are two factors in syringo-myelia: (1) the longitudinal excavation of the cord; (2) the tissue which surrounds the excavation. The cavity, which is usually most marked in the cervical region, may either occupy the position of the normal central canal, or may be evidently distinct from it. In the latter case, it is usually placed in the posterior part of the cord, and therefore we are left in doubt whether it may not indicate an imperfection in the original formation of the central canal. (The normal canal is formed during development by the folding over, from before backwards, of the tissues which are to constitute the cord; and subsequent closing in of the gap left where these folds join.) These abnormal cavities may or may not have an epithelial lining. Outside this lining (if it exist) may come a firm but thin fibroid layer. Outside this again comes a formation of more importance. It appears to consist of an overgrowth of the neuroglia layer which normally surrounds the central canal; this overgrowth may be so abundant as to cause considerable enlargement of the cord, and to constitute, in the opinion of some authors, a veritable new growth. It may be highly vascular, and exhibit hæmorrhages into its substance. Opinions seem to differ as to whether an abnormal and dilated canal is the primary factor, and the growth of neuroglia around it secondary, or whether a new growth first forms and then breaks down in its centre, forming a longitudinal cavity.

These excavations in the cord have been considered, till recently, as mere pathological curiosities; but it

would appear that symptoms may be present which are more or less distinctive of them. The train of symptoms depends not so much on the presence of a cavity, as on the fact that there is a progressive disease in the region of the grey matter (particularly its central or posterior parts) which reaches its maximum in the cervical region, and which may give rise to secondary degenerations of the white matter. Gradual loss of power in the upper limbs may attract the patient's attention ; in course of time muscular atrophy develops, doubtless because the anterior grey cornua are affected. But from the kinds of muscular atrophy we have hitherto considered, this disease is distinguished by its sensory abnormalities. The sense of touch and the muscular sense are preserved ; but the appreciation of temperature and the sensation of pain (one or both) are lost in many parts of the body. There are also nutritional disturbances (other than muscular atrophy) in the skin, in the nails, in the joints, in the secretion of sweat. Spontaneous (and painless) ulcers and whitlows may form. Lateral curvature of the spine may develop : the reason for which is not obvious. In the lower limbs there is (in the later stages) paralysis with rigidity, due probably to descending degeneration in the lateral columns. Sometimes it is said the lower limbs are ataxic, and their tendon-reactions disappear. The course of the disease is slow, and there may be long periods of remission of the symptoms. On the other hand, sudden exacerbations may take place, which may possibly be ascribed to the occurrence of hæmorrhages into the newly formed gliomatous tissue.

CHAPTER VII.

ORGANIC CEREBRAL DISEASE.

Symptoms.—We will now briefly enumerate the symptoms which point to intra-cranial disease: (1) those which are general; (2) those which are referable to disease of particular parts of the brain, or, as they are sometimes called, "localising symptoms."

(1) Of general symptoms, those which point most definitely to organic cerebral disease are:

Paralysis, usually of the hemiplegic type, in contradistinction to the paraplegia of spinal disease. The paralysed limbs do not (as a rule) waste, but in time they become rigid.

Paralysis of cranial nerves.

Paralysis of sensation, mostly either hemianæsthesia or affections of special senses.

Convulsions, particularly if they be limited to a particular part of the body.

Optic neuritis.

Others, less definite in their meaning, but nevertheless sufficient to direct our attention to the state of the brain, are:

Headache.

Local pain, or tenderness to pressure or percussion.

Vomiting.

Coma in its various degrees.

Delirium.

Mental affection.

(2) Symptoms indicative of disease of particular parts of the brain:—

A. Of the cortex cerebri, and of the subjacent white matter. There are two main classes of symptoms—those which indicate destruction of nerve-tissue (paralysis), and those which indicate its irritation (convulsive movements, sensory auræ, &c.). The irritative phenomena are generally better marked in cortical than in sub-cortical disease; the paralytic are much the same in both.*

a. Frontal lobes. No very definite symptoms can be connected with disease of these parts. We except for the present Broca's convolution.

β. Motor area (*vide* pp. 49–51).

Paralysis of the face, arm, leg, or even of such small parts of them as the thumb, hallux, toes, fingers, &c. The paralysis is limited—*i.e.*, is a strict "monoplegia"—when the lesion is limited and non-progressive. In a progressive lesion the paralysis spreads from the face to the arm and leg, or *vice versâ*, till the patient becomes hemiplegic.

Convulsions.—These also have a local character. They begin in a definite part, not unfrequently the thumb or great toe, and spread in a definite manner (*e.g.*, from the thumb, up the forearm, then down the lower limb, and to the face; or up the lower limb, down the upper, and to the face). Consciousness may either not be lost at all, or not till the convulsions have spread over a considerable area. Afterwards, the parts which have been specially convulsed often exhibit a temporary paralysis. The focus of disease is to be sought in the cortical centre for the part wherein the convulsions first begin: the spread of irritation to adjacent centres is shown by the definite

* That is to say, destruction of a cortical centre produces the same effect as destruction of the fibres which connect it with the parts below (peduncular fibres). Destruction of fibres connecting centre with centre (commissural and associative fibres, *vide* p. 53) may perhaps have different effects.

way of spreading or "march" of the convulsions. This type of fit, called sometimes "Jacksonian epilepsy," sometimes "epileptiform convulsion," is of particular importance, because it indicates local, and probably organic, disease. The probability of organic disease is rendered still greater if double optic neuritis be present; we should always carefully look for it.

γ. Occipital lobes.

Hemianopia indicates disease of this part, or of the fibres which connect it with the lower visual centres (optic radiations), or of the optic tract. The half field on the opposite side to the lesion is lost. Disease of the angular gyrus perhaps causes impairment of vision in the opposite eye. Visual spectra may occur in connection with irritation of these parts.

δ. Temporo-sphenoidal lobe.

At the anterior extremity of this (region of the uncus or subiculum cornu Ammonis) is the centre for taste and smell. Cases of tumour here are on record wherein fits were present beginning with auræ of taste and smell. Quite at the other end of the lobe (posterior part of the first convolution) is the centre for hearing. Enlargement of the temporo-sphenoidal lobe (as in abscess or tumour) may by pressure cause symptoms referable to adjacent parts —*e.g.*, hemianopia from pressure on the optic tracts, hemiplegia or hemiplegic convulsions in face, arm, leg of the opposite side from pressure on the internal capsule or on the motor convolutions.

If the temporo-sphenoidal lobe, or the island of Reil, of the *left* side be diseased, it is probable that there will be aphasia of the sensory type (word-deafness or word-blindness). Aphasia of the motor type (inability to express thoughts in words with retention of the capacity for understanding speech and writing) is mostly indicative of disease of the posterior part of the left third frontal convolution (Broca's convolution).

B. Region of the basic ganglia.

The capital symptom is hemiplegia of the opposite side from interference with the posterior limb of the internal capsule, or hemianæsthesia when the posterior extremity of its posterior limb is affected. Disease in the neighbourhood of the posterior part of the capsule may also cause choreoid movements of the opposite side (Charcot).

C. Of the cerebellum.

Disease of the cerebellum (or at least of its middle lobe) is indicated by a disturbance of equilibrium leading to an unsteady reeling gait, and finally inability to stand. There may be rigidity of the trunk muscles causing retraction of the head and opisthotonos. The pressure of a cerebellar tumour may cause: (1) an accumulation of fluid in the cerebral ventricles, which in young children shows itself by enlargement of the head; (2) paralysis of the limbs from compression of the pyramidal tracts; (3) nystagmus or even oculo-motor palsy from compression of the nucleus and root-fibres of the third nerve; (4) facial and auditory paralysis from a similar cause. Absence of tendon-reactions, an occasional effect of intra-cranial disease, is perhaps more frequent in disease of the cerebellum than in that of other parts. (The combination of absent tendon-reactions with unsteadiness of gait must not be mistaken for tabes dorsalis.)

D. Of the corpora quadrigemina.

The symptoms (as enumerated by Nothnagel) are not unlike those of cerebellar disease—viz., unsteady gait, with which is associated nystagmus and ocular paralysis. This latter is usually incomplete, the up and down movements of the eyeballs being affected more than the other movements.

E. Of the pons and medulla.

A lesion sufficiently extensive to affect both lateral halves causes paralysis of all four limbs (cervical paraplegia). Such a lesion is often a hæmorrhage, contraction of the pupils is then apt to occur, pre-

sumably from irritation of the adjacent oculo-motor nuclei or fibres.

There may be vaso-motor disturbances and hyperpyrexia.

A lesion of one-half the pons causes cross-paralysis —*i.e.*, paralysis of one third, sixth, or facial nerve (according to the level of the lesion), combined with hemiplegia or, it may be, hemianæsthesia of the other half of the body.

Tumours of the pons are characterised by the progressive paralysis of cranial nerves and of the limbs.

An acute lesion of the medulla of any magnitude is incompatible with life; the commonest chronic affection is progressive bulbar paralysis, described above (p. 229).

Glycosuria has been observed in tumours of the fourth ventricle (in correspondence with the facts ascertained about glycosuria produced experimentally).

Diseases of cerebral dura mater.—Chronic inflammation of the dura mater may take the form of thickenings of the membrane with adhesions to the brain beneath, or that of a sub-dural hæmatoma, which we have already described (*vide* p. 75). In either case the symptoms are so indefinite that we shall not describe them here. Acute inflammation of the dura occurs chiefly in diseases or injuries of the cranial bones (of which indeed it forms the internal periosteum). The membrane may simply look dark and discoloured, or pus may collect between it and the bone. In ear-disease, with caries of the petrous bone, the condition under which it commonly occurs, such an inflammation may be marked by no further symptom than exacerbation of the pain in the neighbourhood of the ear. It would seem, however, that such severe symptoms as drowsiness, twitchings, and even optic neuritis may develop, which suggest general meningitis or cerebral abscess, but that nevertheless recovery may take place.

Tumours may originate in connection with the

dura mater; they give rise to symptoms in so far as they affect the cerebral surface beneath or the outgoing nerve-trunks.

Thrombosis of sinuses.—Along with affections of the dura mater we may consider thrombosis of the cerebral sinuses. This arises either (1) together with inflammation of the dura or under the influences of causes that affect the dura (ear-disease, bone-disease, injury, &c.); or (2) from such causes as may produce thrombosis of veins elsewhere—viz., the puerperal state, pyæmia, marasmus, sluggishness of circulation from heart-disease, gout (?). The symptoms of intracranial thrombosis are—pain, œdema of the parts drained by the sinus, where these can be observed (thus, in thrombosis of the lateral sinus there may be a puffy swelling behind the ear, in the district of the emissary vein; in thrombosis of the cavernous sinus œdema of the eyelids), disorders of cerebral functions. It is true these characters may be insufficient to establish a diagnosis. Thus, in ear-disease, where there is already pain, and where the mastoid region may be already swollen from disease of the cells beneath, and where brain symptoms may be present from other causes, it may be impossible to tell when the lateral sinus becomes thrombosed. A symptom that may help us to diagnose this is the presence of tenderness and induration along the course of the internal jugular vein (Bowlby), but this is not necessarily present.

Thrombosis of the longitudinal sinus and of the veins emptying into it has been observed to give rise to (1) pain in the head; (2) convulsive twitchings, first in one upper limb, then in the other, evidently due to irritation of the cortex cerebri:* thrombosis chiefly of the straight sinus and velum interpositum to give rise to extreme congestion of both optic thalami, and hæmorrhagic softening of the right thalamus with hæmorrhage into the right lateral ventricle; the

* Sir Dyce Duckworth, "Clinical Society's Transactions," vol. xxiii; and Mr. Arbuthnot Lane in same volume.

symptoms in this particular case being mainly—headache, vomiting, stupor, twitching of fingers, especially the left hand, with some paralysis of the left hand.

Thrombosis of the cavernous sinus* was indicated by proptosis of the eyes, pain in the forehead (from irritation of the frontal division of the fifth nerve), ophthalmoplegia from involvement of the motor nerves of the eye in their passage through the walls of the sinus; there was in this case no œdema of the lids.

Acute general meningitis of cerebral pia mater.—In a well-marked case of acute general meningitis, puriform lymph is found covering the surface of the brain, on the convexity as much as the base, held in the meshes of the sub-arachnoid space. It may form an almost uniform layer, or may be principally distributed along the course of the vessels. The brain itself may be injected and softened. The choroid plexus may share in the inflammation of the meninges, and then there is turbid fluid in the ventricles, and the ganglia may be softened. We have already enumerated the causes of such a disease (*vide* p. 76).

Fever and pain in the head are early and constant symptoms. The height of the temperature may vary much in different cases. The headache is very severe, and by no means proportionate to the fever. There will probably be signs of so-called cerebral irritation—*e.g.*, injection of the conjunctivæ, photophobia, sensitiveness to noise, dislike of being touched or disturbed. Vomiting may be expected, and that apart from food. Examination of the fundus oculi may show the presence of optic neuritis; if so, we may conclude that the foregoing symptoms are due to actual intra-cranial disease; but if there be no neuritis, we cannot draw the opposite conclusion. But sometimes convulsions, or partial paralysis, especially paralysis of the ocular muscles, may occur to stamp the

* Dr. Sidney Coupland, "Ophthalmological Society's Transactions," vol. vii. p. 228.

disease as cerebral. The head is often drawn back, the muscles at the back of the neck being rigid; this is thought to indicate meningitis at the base of the brain, or of the upper cervical cord. Retraction of the abdominal wall may sometimes be seen. On the side of the vascular system there may occur (1) the tâche cerebrale—that is to say, the ready appearance of a white streak, bordered by red flush, when the finger-nail has been drawn across the skin; (2) abnormalities in the pulse-rate, which, instead of mounting *pari passu* with the temperature, may remain infrequent, while the rhythm of the pulse is somewhat irregular.

As the disease advances, the pain and symptoms of irritation give place to an apathetic condition, which deepens into coma. This may or may not be accompanied by actual paralysis. The pulse-rate quickens, the respiration becomes embarrassed, and the temperature may rise very high. Sometimes meningitis (especially in those instances where it complicates grave pre-existing disease) manifests itself only by the sudden access of fatal coma.

Undoubtedly, some patients recover from even grave symptoms of meningitis, but the severe attacks, as a rule, prove fatal. Patients with chronic ear-disease are sometimes seized with severe pain in the head, fever, and perhaps even optic neuritis, which all prove transient. It is conceivable in such a case that the inflammation has not advanced beyond the dura (p. 247). The remedies usually applied are application of cold to the shaven head, leeches to the mastoid region (or even general blood-letting), a smart mercurial purge, saline draughts or iodide of potassium; and for the headache, bromide or morphia, if necessary.

Tubercular meningitis.—By far the commonest form of meningitis is that due to tubercle. Its morbid anatomy we have already described. Its symptoms are essentially the same as those of a simple meningitis, but may manifest themselves in a

somewhat different fashion. They develop more gradually, and are on the whole less severe, though the event appears to be uniformly fatal. The patient is usually* a child from three to twelve years of age. The health may have been failing for some time previous to the outbreak of the disease; there has been change of disposition, sleeplessness, cough, and wasting and the like. Then fever develops, usually not of high degree, headache, vomiting, perhaps convulsions. The diagnosis will have to be made from a febricula in a nervously disposed subject, or from typhoid fever. The abdomen is retracted, not distended nor tender as in typhoid; there is no rash, the temperature does not mount regularly, the pulse is apt to be infrequent and slightly irregular. The tendon-reactions are said to be normal, diminished, or absent,† not increased, as in typhoid. Sometimes optic neuritis is to be found. Sometimes also tubercles may be seen upon the choroid. While asleep the child often utters a short sharp cry, known as the "hydrocephalic cry." The head is retracted. Paralysis of the cranial nerves, when it occurs, is important as indicating probable mischief at the base of the brain, the part upon which tubercular meningitis specially concentrates itself. But hemiplegia may appear, or unilateral convulsions, or convulsions of one limb, and that without a correspondingly localised lesion to be found post-mortem, for the functional disturbance extends far beyond the area of the brain actually affected with tubercle. Before the second and fatal stage of coma sets in, there may be a temporary improvement in all the symptoms, likely to inspire misleading hopes of recovery. We have stated that tubercular meningitis is almost always

* *Vide* table of eleven cases in adults, in Dr. Norman Moore's "Medical Pathology," p. 161.

† This may be due in some cases to the presence of spinal meningitis, in others absence of knee-jerk is seen where the lesion is purely cerebral.

associated with tubercle elsewhere, is often indeed part of a general tuberculosis. On this account, in a doubtful case of cerebral disease, the presence of strumous glands, phthisis in the lungs, or even a strong family history of phthisis, makes it all the more probable that the disease is tubercular meningitis. The fact may also help to explain the great fatality of the disease.

Basal meningitis or cervical opisthotonos of infants.—Under the head of "cervical opisthotonos of infants," Drs. Gee and Barlow* have described a form of meningitis affecting the base of the brain, particularly the neighbourhood of the occipital foramen, and glueing together the cerebellum and medulla. The causation of it is not fully determined. It affects children under two years of age, and therefore occurs earlier in life than tubercular meningitis is wont to do. The essential symptom of it is holding back of the head (which in other forms of meningitis is common but not essential). The onset may be sudden or gradual; some children recover soon, some die, in others the disease persists for many months. In addition to the retraction of the head, there may be fever and vomiting, rigidity of limbs, convulsions, and hydrocephalus. This last symptom is presumably due to closure of the communication between the spinal canal and cerebral ventricles.

Epidemic cerebro-spinal meningitis.—Epidemic cerebro-spinal meningitis is an acute specific disease, wherein the effects of the poison manifest themselves chiefly by inflammation of the meninges, just as diphtheria localises itself upon the fauces, and typhoid upon Peyer's patches. It is uncommon in this country. Its origin can generally be traced to defective sanitation. In addition to fever, pain in the back, and other symptoms of acute meningitis, cerebral and spinal (which we need not repeat again),

* "St. Bartholomew's Hospital Reports," vol. xiv. p. 23.

there is often severe abdominal pain, and a petechial eruption occurs (from the second to the fourth day). Death may occur from collapse, even before the meningitis has manifested itself. Sporadic cases of this disease are sometimes seen, and this possibility should be borne in mind when a case of acute meningitis presents itself, for which no cause can be found.

Syphilitic meningitis is less acute, and more localised than the forms we have described. We shall reserve the consideration of it for the present.

Cerebral abscess.—Inflammation of the brain substance takes place in cases of meningitis, and also in the neighbourhood of tumours, hæmorrhages, and the like. It is likely that encephalitis may also arise as a primary disease, but as yet we know little of it, save of that localised form of it which terminates in abscess. The diagnosis of cerebral abscess is often a matter of much difficulty, it may indeed be impossible, and that for the following reasons. (1) A chronic abscess may exist for long without giving rise to any symptoms. (2) Symptoms when they do arise are not necessarily distinctive of abscess, as contrasted with other diseases of brain, or of its membranes. This is true both of the general symptoms, and of the localising symptoms. The former may indicate in a vague way grave cerebral disease, the latter disease of a particular part of the brain, without giving us a clue as to what the nature of that disease is. (3) The conditions which lead up to the development of cerebral abscess, notably ear-disease with caries of the petrous bone, may themselves give rise to cerebral symptoms—*e.g.*, headache, vomiting, vertigo, fever—without the actual formation of abscess.

The possibility of an abscess must not be forgotten in any case of obscure cerebral disease; but that possibility presents itself most forcibly when one of the recognised causes of cerebral abscess actually exist. These causes (to recapitulate them from p. 65), are chronic disease of the middle ear, or of the nasal

cavities, disease of the cranial bones, injury to the head, pyæmia, empyæma, bronchiectasis. By far the most frequent cause is chronic ear-disease, and we shall therefore address ourselves to this condition. Where grave cerebral symptoms arise in the course of a chronic otitis, which treatment of the ear-disease fails to relieve, an abscess may be suspected, particularly if the localising symptoms point to the sites which such abscesses specially choose—viz., the temporo-sphenoidal lobe or the cerebellum. And if we can exclude such other sequelæ of chronic otitis as meningitis, disease of the lateral sinus, pyæmia*, the diagnosis of abscess may be considered probable.

We will consider briefly, (1) the otitis itself; (2) the general cerebral symptoms indicative of abscess; (3) the symptoms (if any) indicative of its locality.

(1) The otitis is usually chronic, and accompanied by disease of the mastoid cells, or caries of other parts of the petrous bone. (Facial paralysis of the peripheral type, and on the side of the affected ear may be an indication of such caries.) The cerebral symptoms may follow an exacerbation of ear trouble such as acute pain in the ear, suppression or great increase of discharge, &c.; but such a sequence is by no means necessary. For an abscess, formed long ago, may have remained "latent" till some cause quite independent of the original ear-disease lights up mischief around it and so gives rise to symptoms. The otitis itself, or some complication short of abscess (possibly inflammation of the dura mater?), may give rise to serious cerebral symptoms, as we have already said; it would appear that even optic neuritis may arise in this way. This fact complicates the diagnosis; but the practical lesson is that the ear-disease, mastoid

* It must be remembered, however, that any one of these three may co-exist with abscess; and further, that to exclude them definitely may be almost impossible; we can only proceed on probabilities.

disease, &c., should be promptly treated and the effect of such treatment closely watched.

(2) The general symptoms. These, as we have said, are not necessarily distinctive. The commonest are—pain in the head, possibly at the seat of the abscess; perhaps local tenderness also; drowsiness, stupor, coma, sometimes delirium; optic neuritis, paralysis. The temperature often remains low. Convulsions, violent delirium, and acute symptoms generally point rather in the direction of meningitis than of abscess, though no absolute rule can be laid down.

(3) The localising symptoms. These may be very imperfect: there may indeed be none. An abscess of the temporo-sphenoidal lobe might be expected to induce some abnormality of taste, smell, or hearing; but the patient is generally deaf from other causes, and may be in too dull a state for testing of the senses, The same reason may obscure such symptoms as hemianopia and aphasia, though these have been made out. Hemiplegia of the opposite side, involving successively face, arm and leg, may occur; this being the order of proximity of the motor centres and their peduncular fibres to the temporo-sphenoidal lobe. Ptosis and inequality of pupils may be seen. But the site of the abscess is more often guessed from our knowledge that this lobe is the commonest site, than deduced from the actual symptoms.

The only means of cure for an abscess is to open and drain it. This has been done successfully in several instances; and since the disease, when once serious symptoms have arisen, is certain to end fatally, it seems reasonable to trephine and open the abscess, when once we are convinced of its presence and locality. But if, as is more often the case, we only strongly suspect it, the only course is first to treat the ear-disease thoroughly, and then, if the patient still remain in a critical condition, trephine and puncture the temporo-sphenoidal lobe. A successful issue, even if pus be evacuated, must not be too confidently ex-

pected; for it is always possible that the abscess may have ruptured into the ventricles or into the meninges before the treatment has been undertaken.

Abscess of the cerebellum may be suspected in a case of otitis, where there is occipital headache, vertigo, and staggering gait. But it may give rise to no localising symptoms.

Abscess in other parts can only be diagnosed, when there are localising symptoms pointing to disease at a definite part of the brain, and when there is present some one of the known causes of intra-cranial abscess. It is evident that an abscess near the cortex of the motor area may be dealt with surgically, provided we can localise it accurately, and provided that it has not extended too deeply.

Cerebral hæmorrhage.—Cerebral hæmorrhage occurs most commonly in connection with atheroma of the cerebral arteries, and more particularly when such arteries are subjected to a high arterial pressure, as is the case in chronic Bright's disease. In a large proportion of the patients brought in dead into hospital, we find the following associated facts: cerebral hæmorrhage, atheroma of cerebral vessels, hypertrophied left ventricle, granular kidneys, gout. According to some authorities hæmorrhage does not take place until small aneurysms have formed, the rupture of one of these determines the hæmorrhage. These are called miliary aneurysms, they are numerous and very small, and are to be seen, after macerating away the brain substance and clot, studded along the vessels. Or the blood may come from numerous capillary vessels. Cerebral hæmorrhage most frequently originates, as we have already said, from a branch of the middle cerebral artery which skirts the outer side of the lenticular nucleus. Aneurysms of a larger kind, generally about the size of a pea, may rupture and give rise to hæmorrhage; these may be seated on the larger trunks (meningeal or circle of Willis); their origin may be sometimes traced to

previous embolism. Fatty degeneration of arteries, vascular tumours, venous nævi, the congestion consequent on venous thrombosis, &c., are other, though rare, causes of cerebral hæmorrhage. We omit here the intra-cranial hæmorrhages caused by fracture of the skull and laceration of the brain from injury.

The usual sites for hæmorrhage may perhaps be given in this order: neighbourhood of the basic ganglia (as above described), pons, meninges, centrum ovale and convolutions, cerebellum, these last-mentioned sites being uncommon.

Let us consider the commonest forms of cerebral hæmorrhage. The patient is usually of middle age or past it, since the liability to arterial disease and granular kidney increases with advancing years. He may have been in good health till the actual attack, or at most may have had such slight "warnings" as headache, giddiness, or even transient attacks of loss of power, numbness, and paræsthesiæ. The leading features of the attack are coma and hemiplegia, but their mode of onset may vary. In a small hæmorrhage there may be no real coma; the patient may be merely faint, giddy or sick, or even lose the use of his limbs without any such accompanying affection. Or he may wake from his night's sleep and find himself paralysed; or he may experience some strange sensation of numbness, tingling, &c., in the limbs about to suffer, and perhaps lose power in them, and then will come the loss of consciousness. Cases where the symptoms of paralysis and coma come on by degrees, but advance steadily hour by hour, have been called "ingravescent apoplexy," and are said to be always fraught with danger. Lastly, the patient may be stricken down suddenly, unconscious and paralysed, an apoplexy in the original and etymological sense of the word. However it has arisen, in a cerebral hæmorrhage of any severity we usually find the patient comatose. The depth of this coma, as

indicated by the impossibility of rousing him, the stertorous breathing, labouring pulse, relaxation of the limbs, difficulty of swallowing, blunting of reflex action, and the persistence of the coma, are a measure of the gravity of the case. He may die in this condition, or he may not recover consciousness for several days. The hemiplegia may be somewhat masked while the patient is deeply unconscious. We have already mentioned the points to look to (*vide* p. 86)—viz., whether the limbs of one side fall, when raised, in a more flaccid and inert way than those of the other, whether the face is drawn (we need hardly add the distortion is towards the non-paralysed side), whether one cheek is more puffed out than the other. Further, we should note whether the eyes and head turn to one side, remembering (1) that the presence of such a "conjugate deviation" is reckoned a symptom of some gravity. (2) That the deviation of the head and eyes, in a paralysing lesion of one hemisphere, is *towards the side of the lesion.* The state of the tendon-reactions at this stage will probably be of little help towards diagnosing the hemiplegia. As regards the skin-reflexes, it has been asserted that in the early stages of a hemiplegia they are absent on the paralysed side, though retained on the other side.

In some cases there are convulsions. Their presence leads us to suspect that the hæmorrhage involves the meninges, or has ruptured into the ventricles; should either event have happened the prognosis is bad. It is quite possible, however, that convulsions may occur merely as the effect of irritation, and it should be remembered that in some severe epileptic attacks there is transitory hemiplegia —a combination which may simulate hæmorrhage with convulsions. Sometimes the paralysed limbs are rigid, even at this early stage; this probably is also caused by the irritation of the hæmorrhage. The bladder is likely to become distended, and there-

fore the hypogastrium should be always examined, and the urine drawn off if necessary.

The first danger, immediately after the attack, is collapse from the shock of the hæmorrhage. If the skin be cold, the body temperature low, and the pulse feeble, small doses of brandy or diffusible stimulants must be given, and warmth supplied to the epigastrium and the limbs. The second is probably from gradual increase of the hæmorrhage, indicated by increasing coma, and paralysis spreading to the organic reflexes of swallowing, respiration, &c. A large or advancing hæmorrhage will probably rupture into the cerebral ventricles; this is a fatal complication. Whether any medical means can arrest hæmorrhage is somewhat questionable; it is at any rate advisable to keep the patient at absolute rest, with the head slightly raised, and cool; usually a purge, calomel or croton oil, is given, and mustard plasters applied to the calves. A third danger appears later, during what we may consider the period of reaction, from a day or two to a week after the attack. After some initial improvement, perhaps after consciousness has begun to return, the temperature rises and continues to mount steadily, then the respiration becomes embarrassed, the face becomes blue, and the skin clammy; finally, the patient dies, apparently from the side of the lungs. After death in this way there is found extreme engorgement of these organs; and since the lung on the paralysed side suffers most, it has been urged that such "congestive pneumonia" is really a trophic lesion depending directly on the paralysis. The same remark applies to another phenomenon sometimes seen at this stage—viz., the appearance of an acute bedsore on the buttock of the paralysed side.

In more favourable cases consciousness returns: the amount of the hemiplegia will now be more easy to estimate. We next begin to look for improvement of the hemiplegia; and however complete the para-

lysis be at first, it does not follow that it may not improve—perhaps in a few days, perhaps in a few weeks.

The earlier such improvement begins, the quicker it goes on. Sensory paralysis, if there be such, usually mends sooner than the motor: the movements of the leg return before those of the upper limb; and those of the arm and forearm, before those of the thumb and fingers. But there may be little or imperfect amendment, and the patient then remains a chronic hemiplegic.

Varieties according to site of lesion.—Hæmorrhage into the pons is necessarily graver than hæmorrhage into one hemisphere: it is distinguished by the contracted state of the pupils, and by the fact (if the distribution of the paralysis can be made out) that either all four limbs are paralysed, or that there is cross-paralysis. Vaso-motor disturbances and high rises of temperature are apt to take place.

Meningeal hæmorrhage is almost always fatal, convulsions or twitchings of the limbs, and delirium, are common accompaniments. Thus it is liable (as Dr. Wilks remarks) to be taken for uræmia.

We need hardly add, that according to the amount of the hæmorrhage so the symptoms may vary in degree, from those of a fatal disease to such as scarcely interrupt the patient's avocations; and according to the position of the hæmorrhage, so may the paralytic symptoms vary in kind. The ordinary form of paralysis, hemiplegia, is caused by pressure on or rupture of the internal capsule. Where there has been mere pressure the paralysis may recover; where the capsule has been destroyed, it remains lastingly.

Cerebral softening from arterial obstruction.—Softening of the brain substance, apart from that produced by venous congestion (which we have alluded to under the head of thrombosis of the sinuses), and apart from that produced by inflammation in the neighbourhood of a tumour or a meningitis, is pro-

duced by obstruction of the cerebral arteries. The central cerebral arteries are terminal, and those of the meninges have not free anastomoses; hence the block of any vessel beyond the circle of Willis produces immediate loss of blood-supply, and therewith loss of function, and next a necrosis of the brain tissue, which manifests itself post-mortem as softening, in the district of the blocked artery.

Embolism and thrombosis. — The arterial block may be due either to embolism or thrombosis. Emboli generally consist of fragments, swept by the circulation from a diseased cardiac valve; or it may be of portions of clot from the interior of an aneurysm; their favourite place of lodgment is the middle cerebral artery at one or other of its bifurcations, and more often (it is said) the left middle cerebral than the right. Thrombosis may take place in any artery; the two main causes thereof are syphilitic arterial disease (which we shall discuss later on) and atheroma.

Between the symptoms of cerebral softening and those of cerebral hæmorrhage no distinction can be drawn that is absolutely diagnostic. Coma it is said more frequently accompanies the latter; but as in a slight hæmorrhage coma may be absent, so it may be present in an extensive and sudden softening. Perhaps in a hæmorrhage of any degree the phenomena of shock are likely to be more severe than in softening. Softening from embolism would seem to entail a very sudden onset of paralysis, whereas in hæmorrhage there may be prodromata; but in softening from thrombosis the paralysis may develop (though this is not invariable) by degrees. In some few instances embolism of the arteria centralis retinæ may be seen, and may be taken as the index of an analogous process within the brain. Sounder distinctions are perhaps to be sought in the antecedents and constitution of the patient. When he is of middle age or past, and has a hard pulse with tortuous temporal arteries,

a heaving cardiac impulse, and watery urine containing a trace of albumen, with maybe a gouty history, we suspect a hæmorrhage. In old people with calcareous arteries and feeble circulation, we think of thrombosis from atheroma. Thrombosis in younger people is mainly the result of syphilitic arteritis. In a young patient with signs of valvular disease, hemiplegia is most reasonably explained by embolism. Yet we have twice seen recently the following order of events: a patient, who is in hospital for severe heart disease, becomes suddenly unconscious and hemiplegic, embolism is naturally diagnosed; but post-mortem hæmorrhage is found from an aneurysm, the aneurysm having been probably caused by some previous embolism. Though speedy death may unquestionably result from extensive softening, hæmorrhage is the most dangerous process to life; on the other hand, the prognosis, *quoad* recovery, is worst in softening; for here the paralysis depends mainly on destruction of tissue, whereas in hæmorrhage it may be largely due to the effects of pressure.

The characters of confirmed hemiplegia.—Permanent hemiplegia is produced by destruction of the motor tract in the internal capsule, or indeed above that level, provided the lesion be sufficiently extensive; and the hemiplegia has eventually the same characters, whatever was originally the nature of the lesion. It is worth while considering this condition of "late hemiplegia." The distribution of motor paralysis, the presence or absence of aphasia, and of sensory abnormalities, is mainly dependent on the seat of the original lesion. Commonly, however, sensory paralysis, and to a certain extent speech difficulties,* tend to disappear, and the paralysis of the leg

* This probably does not hold good for aphasia dependent on cortical or sub-cortical disease; but the minor forms of speech difficulty, mostly difficulties of articulation, which accompany right hemiplegia, generally improve before the hemiplegia itself.

and face mends sooner than that of the upper limb, and the coarse movements at the shoulder and elbow return sooner than the fine movements of the fingers and head. A striking feature of a permanent hemiplegia is rigidity. Such rigidity, when developed slowly at some distance of time from the original attack, is due to a descending degeneration of the pyramidal tract. Its advent is heralded, as a rule, by the appearance of exaggerated tendon-reactions and ankle-clonus in the paralysed limbs. We have already described the position which the rigid limbs assume—the stiff, extended leg, the clenched fingers, the semi-flexed and semi-pronated forearm (*vide* p. 102). Occasionally the tendon-reactions become exaggerated on the sound side as well, and in rare cases there may be double ankle-clonus, with even rigidity and paralysis of both legs. This seems to indicate some spread of the degeneration to the pyramidal tract of the sound side, we do not precisely know the mechanism of it. Another rare complication is the appearance of muscular wasting with electrical changes due to the advance of degeneration from the pyramidal tracts to the anterior cornual cells. Involuntary movements form another class of complications. Most commonly such movements are of the kind known as athetosis (*vide* p. 91) —viz., slow continuous movements, principally of the fingers, hand, and forearm ; these are seen mostly when the hemiplegia is incomplete, and when it has originated in early life. Less often there is ataxia of the paralysed limbs, or tremors, suggestive of disseminated sclerosis, or even of paralysis agitans. Pain in the paralysed limbs is not unfrequent ; it appears to arise spontaneously, may be very severe, and is refractory to treatment. Convulsions are frequently associated with hemiplegia. The association may be of various kinds: thus, in children, hemiplegia may follow immediately upon an attack of convulsions, the hemiplegia persists and the convulsions are repeated at intervals ; probably both symptoms are due to some

one common cause. Similarly, in adults, an arrested cortical lesion (generally a gumma or a local meningitis) may give rise both to hemiplegia and to convulsions. But there is a variety of convulsion which may be more truly called "post-hemiplegic." Here the hemiplegia originates in some ordinary vascular lesion, hæmorrhage, embolism, or the like; after a time, and without apparent spread of the disease, a fit of convulsions takes place, and subsequently others. Sometimes their connection with the hemiplegia is shown by the fact that they are limited to, or commence in, the paralysed limbs; sometimes they are general convulsions with loss of consciousness, and recurring with tolerable regularity, like true epileptic fits, and remediable, like them, by treatment with bromide. In most cases of hemiplegia the mental functions are not affected; but occasionally the patient becomes gradually irritable, forgetful, or foolish. Where the hemiplegia is dependent on such arterial disease as atheroma or syphilis, such symptoms may be reasonably ascribed to general cerebral mal-nutrition.

Double hemiplegia, or "Di-plegia."—It may happen that a patient has had two attacks of hemiplegia, one on each side, and that the effects of both persist. This is called double hemiplegia, or di-plegia. In this case functions may suffer which escape in ordinary hemiplegia, as, for instance, that of swallowing, and perhaps that of micturition. In some rare cases the lower face and tongue have been paralysed from a double lesion, one in each cerebral hemisphere. Such a condition closely resembles paralysis of the tongue and face from disease of their bulbar nuclei (*vide* p. 229), and has hence been called "pseudo-bulbar paralysis." But there is no wasting of the tongue, nor change in the electrical reactions.

Hemiplegia in children.—Hemiplegia of cerebral origin occurs in young children with much greater frequency than might have been anticipated. It is distinguished from the effects of anterior cornual myelitis (infantile *spinal* paralysis) as follows: (*a*) by

the distribution of the paralysis. In the spinal disease a hemiplegic distribution is unusual, and the face is never affected. (β) By the tendon-reactions; these are exaggerated in the cerebral disease, normal or absent in the spinal.* (γ) By the nutrition of the limbs. Although in the cerebral paralysis there may be a general shrinking, and even shortening from arrested development of the bones, yet the wasting does not, as in the spinal paralysis, pick out groups of muscles, and there is no electrical reaction of degeneration. The causes of hemiplegia in children are somewhat obscure. Doubtless it is sometimes due to embolism or arterial thrombosis (for such thrombosis hereditary syphilis may be responsible, which is capable, like the acquired disease, of producing cerebral arteritis). Other cases may be due to mal-development of the brain, or to injury during birth. Infantile hemiplegia often sets in with convulsions, as if the seat of the disease were in the cortex. To account for this, two theories have been proposed: (1) that the original disease is a thrombosis of the meningeal veins which drain the cortex (Gowers); (2) that, as in infantile spinal paralysis there is an inflammation limited to the anterior cornua of the cord, so in the cerebral disease there is inflammation of the large cells of the motor area of the cortex, acute polio-encephalitis (Strümpell).

Treatment.—When hemiplegia has persisted for any length of time (say for a year), and when, in addition, contractures of the paralysed limbs have slowly and steadily supervened, we may assume that there is tolerably complete descending sclerosis of the pyramidal tract of the paralysed side. Such a condition must be regarded as incurable. It may be advisable, with a view of preventing the extreme flexion of the fingers, to apply faradism to the extensor muscles from time to time, and to practise passive manipulations of the joints,

* Absent, where the muscle which normally enters into the reaction is paralysed: thus, in atrophy of the quadriceps cruris the knee-jerk is abolished.

in order to obviate secondary ankyloses and deformities. In milder cases, where there is less contracture, and some retention of power, faradism may be useful as a sort of gymnastic to the muscles. But it must be used carefully, as strong currents are likely to increase contractures. Massage is also useful in such cases. The rigidity of lateral sclerosis, just like the pains of posterior sclerosis, are aggravated by cold, hence the limbs should be kept warm, and hence an equable climate, free from damp, cold, and from biting east winds, is the best for such patients. Hydropathic treatment may also be of use. But it is evident that we can neither repair the lost nerve-tissue, nor obviate the sclerosis which follows its loss.

Cerebral tumours.—In a case of intra-cranial tumour it is obvious that the nature of the tumour will cause differences in the symptoms, according to the rate of its growth, and the degree in which it actually destroys or merely displaces the cerebral tissue. Thus, a fibroma which springs from the dura, and simply displaces the adjacent brain-substance, may cause next to no symptoms. Malignant tumours, whose advance is rapid and unaffected by treatment, cause marked symptoms both of nervous irritation and paralysis. In a third class of tumours, those due to tubercle or syphilis, we may witness sometimes the development of grave symptoms followed by their subsequent arrest, it may be as a consequence of treatment, or it may be spontaneously. The nature of an intra-cranial tumour, hidden as it is from sight and touch, must be a matter of conjecture only, a conjecture based upon the intensity and rapidity with which the symptoms develop, upon the physical condition and history of the patient, and possibly upon the occurrence of tumours in other parts of the body.

General symptoms.—Of the general symptoms of such a disease—*i.e.*, those which indicate its presence without indicating its locality—the most important are:

(1) Pain in the head. In any rapidly advancing growth this is likely to be very severe; more so than in any other form of cerebral disease, excepting perhaps meningitis. It is less intermittent than headache from migraine. In any case of severe and persistent headache we should examine for the next symptom—viz.:

(2) Optic neuritis. This, as we have seen, is a sign that very frequently accompanies gross organic disease within the cranium, and of such organic diseases intra-cranial tumour is the one that most frequently gives rise to it. It is present in most cases of tumour at some stage or other of their development. In the course of some tumours optic neuritis may be observed to appear and to pass on into optic atrophy, the tumour all the time continuing its development. Or such a "post-neuritic" atrophy may be seen when the patient first comes under observation, indicating that a tumour has formerly existed, or exists in a quiescent state, or has been developing during a considerable time. The exact relation of optic neuritis to tumour is not definitely known: we cannot infer from the neuritis the position, nor the stage, nor the character of the tumour; we only know that the two things frequently co-exist.

(3) Vomiting may accompany cerebral tumour. It has been ingeniously suggested by Dr. Buzzard that such vomiting may be due to a neuritis of the pneumogastric, analogous to optic neuritis. We cannot distinguish it from vomiting due to cerebral disease of other kinds. From the vomiting of gastric disease it is distinguished by the absence of other signs of gastric disease, and by the fact that it has no constant relation to the ingestion of food; from the vomiting of migraine, by the fact that the headache and other symptoms are not terminated by it.

(4) Coma occurs in the later stages of many cerebral tumours. It is doubtless often an index of increased intra-cranial pressure, either from mere size of the

growth, or from the accumulation of intra-ventricular fluid which its pressure has occasioned.

All the symptoms we have enumerated, and in addition convulsions, perhaps even localised convulsions, may occur in uræmia. Probably they will then appear somewhat brusquely; whereas in organic cerebral disease they have a steady development; but in any case the urine should be examined, to eliminate, if possible, the presence of Bright's disease.

Localising symptoms.—Localising symptoms, those which point to the seat of a tumour, may be absent, vague, or precise, according to its position. They attain their greatest precision when it is seated in the motor area of the cortex. The comparatively slow evolution of a tumour, and the fact that it infiltrates the nerve-elements before destroying them, usually allows a period of irritation. This is evidenced (in the motor cortex) by attacks of convulsions, of the type known as epileptiform fits, or "Jacksonian epilepsy." We have already described these * as being particularly characteristic of organic disease of the motor cortex (more often than not a tumour of this part); let us emphasise again the importance of observing in what member the convulsion begins, because the focus of disease probably corresponds with the motor centre for this member; and the manner in which the convulsion spreads ("march of the spasm"), because the limbs are often involved in an order which corresponds to the order of proximity of other centres to the focus. The "discharge," as it is called, begins at the focus of disease, and the neighbouring centres are "let off" in turn. The temporary paralysis which often follows in the limbs that have been convulsed is reasonably attributed to the exhaustion of the centres. But as the disease proceeds this temporary paralysis becomes permanent, and independent of the convulsions; this may be taken to indicate a second stage, in which actual

* *Vide* p. 244.

destruction of nerve-elements is taking place. This stage is important, because the probabilities of complete repair are now small, and because it sufficiently distinguishes the case from certain puzzling instances in which Jacksonian epilepsy occurs without organic disease. If the tumour still progresses, it is probable that more general cerebral symptoms will arise, such as coma, paralysis of all the limbs, rise of temperature with pulmonary congestion; these are usually the beginning of the end. If the tumour be arrested, as may be seen in cases of gummata principally, the patient's life may be saved, but he may remain either non-paralysed but liable to convulsions, or subject to convulsions and withal permanently paralysed. This will depend on the amount of damage done to the cortex.

Convulsions which have commenced in the manner just described may become, as a case proceeds, so far general that it is impossible to be sure of their local origin; and sometimes convulsions, which from the outset have been general, in association with double optic neuritis, may be the only indication of a cerebral tumour.

Similar symptoms of irritation and paralysis of the sensory organs may arise when a growth is situated in the sensory sphere of the brain. The difficulty of investigating them is much greater than in the case of the motor phenomena, neither can we at present refer them with equal accuracy to definite parts of the brain. We may notice here that even when the tumour is in the motor area, a certain numbness or paræsthesia may be present in the limbs that are affected by convulsions.

We shall not attempt to describe the symptoms produced by growths in each and every part of the brain. We have given above (*vide* p. 244 *et seq.*) a short sketch of such localising symptoms, which will hold good for tumours as well as for other diseases. Given a certain set of symptoms, we have to think whether

they can be explained on the supposition that some definite spot in the brain is diseased. Only let us remember also the following facts. First, that the effects of a tumour may extend far beyond its actual site; obviously this holds for effects of irritation, but it holds also for paralysing effects. For softening of brain-tissue may take place around the growth, or its pressure may annul the functions of nerve-elements that are structually healthy. Secondly, that tumours may be, and often are, not single, but multiple. Thirdly, that when a growth slowly infiltrates the tissues (this is seen particularly well in gliomata of the pons) the functions of the part affected may persist much longer than we should expect.

Sites and varieties of cerebral tumours.—There are hardly any parts of the brain in which malignant growths may not occur. These are mostly sarcomata of a mixed type, or the small celled variety, peculiar to the nervous system, known as gliomata; they are to be found most commonly in the cortex, cerebellum and pons, also in the centrum ovale, basal ganglia, upon the cranial nerve-roots, in the pineal and pituitary bodies, or growing from the dura mater. Infiltrating glioma of the pons occurs in children; the organ becomes enlarged and nodular, yet may still preserve its general shape. Sarcomatous growths of the brain are generally primary, and they do not (perhaps because they have not time) give rise to secondary growths elsewhere. Carcinomata of the brain are less common, and almost always secondary to growth in other parts of the body.

Tubercular tumours occur as caseous masses, sometimes large and solitary, sometimes smaller and multiple; they are commonest in children, though not unknown in adults. The cerebellum and pons are favourite seats for them. They may give rise to no symptoms, while thoroughly quiescent; or symptoms may arise and quiet down again. They may, on the other hand, slowly progress to a fatal issue:

or again a tubercular meningitis or a general tuberculosis may arise in connection with them and cause death.

Gummata affect the surface of the brain rather than the deeper parts.

Diagnosis.—The gradual development of symptoms, and the presence of optic neuritis, distinguishes a tumour from cerebral hæmorrhage and from most cases of softening. But it may happen in a very vascular tumour that a vessel suddenly gives way; and the symptoms due to this occurrence may be the first really to attract attention; a careful inquiry into the patient's antecedent condition must be made ere a diagnosis can be formed.

An abscess may be difficult to distinguish from a tumour. If the predisposing causes of abscess (ear-disease, bone-disease, injury to the head, &c.) do not exist, and have not been in operation, this militates against that diagnosis. In cerebral sarcomata, it must be remembered, there is often a history of a blow on the head. An abscess situated in the temporo-sphenoidal lobe may remain latent a long time, and then give rise to severe symptoms such as coma, &c.; whereas in most tumours the initial symptoms are better marked, and the progress of them more steady. Perhaps, too, optic neuritis is commoner in the early stages of tumour than of abscess.

Treatment.—It is evident that in cases of malignant tumour, little can be expected from purely medical treatment. It will be right, however, to give iodide of potassium at once, and increase the dose to 20 or 30 grains three times a day or more. For as a rule we cannot be certain that the disease is not syphilitic; if it is, prompt treatment may arrest or cure it; if not, no harm will have been done by such treatment. It is just possible, indeed, that growths other than syphilitic may thus be influenced for good. Where we suspect tubercular disease, cod-liver oil, iodide of iron, or other tonics, with a generous diet, must be

given ; tubercular disease is not necessarily aggressive, and an improvement in the patient's nutrition may perchance arrest its growth.

Lately, some cerebral tumours have been successfully dealt with by trephining the skull and removing the affected part of the brain. Some requisite conditions for such operations are the following : that the tumour should be single, situated superficially, in a part of the brain which can be reached by trephining, and which can be operated upon without danger to life ; and, further, that the symptoms should be definite enough to fix the exact site of the disease, and the requisite position of the trephine-hole. The motor area of the cortex is perhaps the only position for a tumour which satisfies these conditions. The feasibility of the operation proposed, the amount of risk which in the opinion of the surgeon it will involve, the prospect of complete or partial ablation of the tumour, and of cure or relief for the patient's symptoms, are further questions which must be well weighed for each particular case. The possibility of a multiple tumour must also be remembered. If medical and surgical treatment alike fail us, attention must still be given to the nutrition and nursing of the patient. He may be fed with the nasal tube, when unable to take food in the ordinary way ; and this, with attention to the state of the bladder, and the prevention of bedsores, may keep him alive long after consciousness has failed.

Chronic hydrocephalus.—By chronic hydrocephalus is meant a gradual accumulation of fluid within the cranium, and in most cases within the ventricles of the brain. The causation of this is not always clear. In some cases it seems evidently due to pressure on the venous channels which take the blood from the brain, as when a cerebellar tumour presses upon the veins of Galen, or to closure of the apertures whereby the cerebro-spinal fluid normally escapes from the ventricles into the sub-arachnoid

space, as when a meningitis at the base of the brain has sealed the foramen of Magendie. But sometimes no such mechanical cause can be found, and we have to suppose that there has been excess of secretion, probably inflammatory, from the lining membrane of the ventricles. This membrane indeed may be thickened, sometimes even blocking up the Sylvian aqueduct (so that the mechanical factor is re-introduced); and, moreover, the character of the fluid, which in certain cases contains more albumen than does ordinary cerebro-spinal fluid, seems to indicate that some further process had been at work than the mere penning up of the normal secretion.

The effects of such an accumulation of fluid, if large, is to arch the corpus callosum upwards, rendering it tense and thin, to flatten out the floor of the third ventricle, pressing the large ganglia downwards and outwards, and to convert the lateral ventricles into large arched chambers over which the cortex and white matter is stretched in the form of a thin layer, from the outer aspect of which the natural sulci have been long since obliterated. The pressure affects the skull too, and mostly in a manner that is visible during life, for the disease, more often than not, begins before thorough ossification has taken place.

Indeed, it may begin before birth; hydrocephalus in the fœtus forms one of the recognised causes of difficult labour. The fontanelle is rendered gaping and tense; the bones on each side of the sutures begin to open out "like the petals of a flower," and the head enlarges, laterally and antero-posteriorly rather than upwards, so that it appears somewhat flattened in relation to its size. The orbital plates are driven downwards by the pressure, and the eyeballs forced forwards, so that the upper lid no longer partially covers the cornea, but leaves a ring of sclerotic exposed above it. This gives a staring look, which, with the bulging forehead above and the

relatively small face below, is very characteristic of a hydrocephalic child. The skin over the enlarging cranial vault may become tense and thin, and the superficial veins enlarged, the hair scanty. Sometimes the stretching of the brain-tissue and of the cranium becomes so great, that the head becomes translucent like a hydrocele. The enlargement is not always symmetrical on the two sides, and sometimes one part of the head may enlarge more than others. Irregular ossification may go on, so that Wormian bones are found in abnormal places, perhaps in the centre of an arrested suture.

The mere enlargement of the head may sometimes cause inability to sit or stand upright, or even to hold it up without support. But there are other symptoms dependent on disordered function. Fits are one of the most common; among others are inability to walk, and to use the hands properly, or actual paralysis with increased tendon-reactions, or even rigidity of the limbs; blindness, with optic atrophy, squint, nystagmus: deafness sometimes, irritability, the impairment of intellect, or complete idiocy. A rapid increase of fluid may lead to fatal coma.

The prognosis is not good; yet the disease may come to a standstill, and there are well-known instances in which the patient has lived to adult life (or even old age), and that with his full share of intellectual power. As to medical treatment, mercury has been recommended, especially pills of mercury with squill (Watson), iodides, cod-liver oil and tonics generally; and in the way of surgical relief, cautious tapping through the distended suture with a fine trocar and cannula; or mechanical support and pressure by means of bandages or strapping to the skull.

Hydrocephalus may come on in adult life, either as a consequence of some other grave cerebral disease (meningitis, tumour, or the like), in which case the symptoms of the primary disease preponderate; or in rare cases it may occur primarily. In two such

cases which the author recently examined in the post-mortem room of St. Bartholomew's, the symptoms during life were simply such as would suggest a cerebral tumour—viz., pain in the head, vomiting, and optic neuritis, but no such cause for the effusion could be found at the autopsy.

SYPHILIS OF THE NERVOUS SYSTEM.

We have had to make several allusions to syphilis of the nervous system: we must now consider this object more particularly.

Morbid anatomy — Gummata. — There are several ways in which syphilis may affect the nervous system. Definite tumours (gummata, syphilomata) may form in it; they are described as consisting of three layers, the periphery is a zone of small lymphoid cells, within this is a ring of fibroid tissue, and in the centre a structureless or caseating mass. Gummata may be extremely difficult to distinguish from tubercles: their interior is more vascular, and (hence) caseation is not so marked a feature; giant cells, though sometimes present, are not so common as in tubercles. The discovery of tubercle bacilli will of course settle the pathological diagnosis. Gummata can be absorbed and leave only an inert scar, this is much less commonly the case with tubercle.

Syphilitic inflammation.—Apart from definite tumours, we may find a syphilitic inflammation; in this the tissues undergo an infiltration with the small round lymphoid cells, and though these have not the definite massing or structure of a gumma, doubtless the process is essentially the same. Such syphilitic inflammation is common in the cerebral meninges and in those of the cord: the process is generally subacute and more or less local, herein differing from an acute general meningitis; it does not concentrate itself upon the base of the brain and Sylvian fissures with such uniformity as does tubercular meningitis, but may affect either the base or the convexity. The pia is infiltrated with a somewhat thick, dirty-looking

lymph, and the peripheral parts of the underlying nerve-tissues are apt to be affected as well. Syphilitic infiltration also may occur in the nerve-trunks and nerve-roots, particularly the cranial nerves. The nerves are thickened or show fusiform swellings, and appear grey or reddened according to their state of vascularity: microscopically they show accumulations of the small round cells in their interior, probably with abundant blood-vessels, while the nerve-tubes are in various stages of disintegration.

Nodes.—In these two processes, syphilomata and syphilitic infiltration, we have examples of syphilis attacking the nervous system directly. The attack may be rather more indirect. Thus, patches of subacute inflammation may form in the dura mater, analogous to the nodes that are seen outside the skull; if these occur at the foramina where nerve-trunks leave the skull, the nerves are likely to be compressed and paralysed. Perhaps too such nodes, if large enough, may irritate the subjacent cortex cerebri and so cause convulsions, &c.

Arteritis.—But of far more importance than such nodes in its influence on the nervous system is syphilitic disease of the arteries. This is particularly frequent in the arteries of the brain—the basilar artery, circle of Willis, carotids, and sometimes their meningeal branches. The main characteristic of the disease is an overgrowth of the tunica intima, or more strictly of the deeper layer of it which lies between the epithelial lining of the interior surface and the fenestrated coat of Henle. This part of the coat is thickened with a material which appears to be partly fibroid, partly cellular. The thickening is sometimes in nodules, sometimes runs all round the vessel, and takes place at the expense of the lumen, so that it projects inwardly and narrows the calibre of the vessel. The narrowing may be so great as practically to occlude the vessel, or to cause occluding thrombi to form. In addition to this affection of the intima (end-arteritis)

there is generally an infiltration of the external coat (peri-arteritis) with the vascular growth of small round cells which we have already noticed in connection with syphilis. Syphilitic arteritis appears to the naked eye as nodular thickenings of the vessels; these are isolated, and do not involve the arteries so generally as does atheroma, neither do they become calcareous as does atheroma. The effect of such disease may be varied and widespread. The loss of elasticity and partial occlusion of the arteries may cause a semi-starvation of the brain, producing variable phenomena of irritation and paralysis, complete occlusion may produce necrotic softening with permanent paralysis as its result. Again, as the distribution of the arteritis varies so will that of the nervous lesions resulting from it; and the particular symptoms will be as variable as the seat of the lesions.

Degenerations and neuroses.—There is yet another way in which syphilis acts as a cause of nervous disease; that is by producing, or perhaps we should say predisposing to, chronic degenerations and functional nervous affections. Statistics make it almost certain that syphilis is in some way concerned in the production of tabes dorsalis, and in that of general paralysis to some extent; again, it may be a factor in some cases of peripheral neuritis: but we do not know how. For in the morbid anatomy of such diseases we find nothing characteristic of syphilis, neither do the symptoms respond quickly and certainly to antisyphilitic treatment. We must content ourselves therefore with the vague supposition that they are the outcome of a vicious state of system which syphilis has contributed to produce. The same must be said of the syphilitic neuroses, epilepsy and neuralgia in particular, which have been described (doubtless with reason) by some authors.

Diagnosis of nervous syphilis.—But, omitting this last and very difficult question, how can we tell that a patient's nervous symptoms are due to

syphilis? Mainly by consideration (1) of the evidence of past or present syphilis; (2) of the symptoms themselves—their character, development, and grouping; (3) of the effect produced on them by antisyphilitic treatment.

(1) The presence or history of syphilis does not of course prove the nervous disease to be syphilitic, but it is a step in that direction. We must either prove or assume the existence of syphilis. The primary infection has often been unnoticed or forgotten by the patient, for it may have been long ago; and from many women it may be (as has well been said) "cruel and useless" to ask for such a history; we have then to rely upon later evidences. For information upon this head we must refer the reader to treatises on constitutional syphilis, only reminding him of the importance of looking for syphilides, scars on the legs, nodes on the skull and tibiæ, affections of the tongue and throat, perforating ulcers of the palate, and choroiditis. The history, in married women, of a series of miscarriages without obvious cause has some diagnostic value.

The nervous symptoms have no special connection with the severity of the original syphilitic symptoms. They may follow a severe or a mild attack of syphilis. Indeed it often happens that the only history obtainable is that of a chancre, with none of secondary symptoms. It may be that in these mild cases there has been absence or insufficience of treatment, and hence the nervous sequelæ. As to time, it is most common for nervous phenomena to appear at a late period of constitutional syphilis, it may be some years after the infection, so that they are commonly ranked as "tertiary" symptoms. But this is not invariable, the nervous system may be attacked within a few months, and while so-called "secondary" syphilides are in bloom. Syphilitic meningitis and diffuse inflammations of the nerve-roots, &c., are more likely to appear early than are gummata and arterial disease.

(2) The nervous symptoms themselves.

Their outbreak may be attended with some such conditions as these: a cachectic state, described by Buzzard, in which the patient appears anæmic, sallow, and earthy-looking, as he would after long-repeated ague; headaches, continuous and severe; pains in the bones of the legs and skull, severe and aggravated at night; giddiness or paralysis, which may be incomplete and transient, in the district of the cranial nerves; particularly ptosis and ocular palsies. Such preliminaries, however, are by no means necessary; grave nervous affections may appear in a patient who has made no previous complaint.

We cannot single out any symptoms as absolutely characteristic of nervous syphilis, for the disease acts by producing local lesions, whose clinical expression is the same, whatever their cause. Yet they may be suggestive of some of the syphilitic processes we have described. If the symptoms of cerebral tumour develop somewhat rapidly in a syphilitic subject, we naturally think of a gumma. Epileptiform fits with double optic neuritis are common under such circumstances, for gummata specially affect the surface of the brain. Again, they are frequently small, and do not create much inflammation in their neighbourhood, hence their effects may be localised within narrower limits than those of other tumours. Thus a hemiparaplegia may indicate a gumma involving one-half the cord, or a paralysis of one external rectus oculi coupled with paralysis of the limb on the other side, may indicate a gumma at the egress of one sixth nerve. Cerebral symptoms of a more diffuse character, such as convulsions, which are unilateral, but have no definite place of commencement nor definite march, may very likely be due to a meningitis. The symptoms of a syphilitic meningitis are less acute than those of an ordinary purulent meningitis, neither have they the regularly fatal progress of the tubercular disease. Paralysis of nerve-trunks is a

frequent occurrence in syphilis of the nervous system. It may be due to a syphilitic infiltration of the nerve, and is most frequent in the cranial nerves. It may be bilateral, thus both third nerves, or both facial trunks, may be paralysed. Such symmetry indicates a constitutional cause. Sometimes the grouping of nerve paralysis suggests disease of the orifice through which several trunks pass; thus we may have paralysis of the facial and auditory of one side from periosteal swelling around the meatus auditorius internus, or paralysis of the oculo-motor nerves and the fifth from disease near the cavernous sinus or sphenoidal fissure, or unilateral paralysis of the tongue, palate, and vocal cord, when the hypoglossal and spinal accessory are implicated in syphilitic disease at the base of the brain. It is not very common to find syphilitic disease of the nerve-trunks of the limbs. Arterial syphilis may exhibit both general and local symptoms. There is generally headache; faints, giddiness, and transient convulsions may occur. These are thought to be caused by interruptions in the equability of the blood-supply, from damage to the elasticity of the vessels or from partial occlusion of them. Transient paralyses may in like wise happen, either of a limited area, such as the eye-muscles or face, or else a hemiplegia. A peculiar state of hebetude has been described. The patient is not actually comatose, but somnolent. He can be roused perhaps when pressed with questions, or sufficiently to take his food, but he soon relapses into stupor. This condition may last for days or longer, sometimes it clears off for a short time and then comes on again. Even these symptoms, vague as they are, may not be present, and the first fact indicative of syphilis of a cerebral artery may be an attack of permanent hemiplegia caused by thrombosis and consequent cerebral softening. The syphilitic origin of such a hemiplegia can only be arrived at by excluding other causes. Embolism may be excluded when the heart and vessels appear to be healthy; thrombosis from atheroma, and

cerebral hæmorrhage, when the patient is under middle age and has no renal disease.

More characteristic than the individual symptoms is the grouping of them. There are not many conditions other than syphilis in which several organic lesions arise at divers parts of the nervous system. Hence, when the symptoms point to multiple lesions, it is probable that these may be syphilitic. Say that a patient who is paraplegic develops epileptoid fits with optic neuritis, or paralysis of some cranial nerves, or that a hemiplegia is complicated by symptoms distinctly spinal, we cannot explain his condition except by supposing more than one focus of disease, and for these syphilis is a likely basis. We must remember, however, that malignant new growths are sometimes multiple in the brain, though not usually in both brain and cord, and that a very wide-reaching influence is exercised by such diseases as tabes dorsalis, general paralysis, multiple neuritis, occasionally tubercle, still more by disseminated sclerosis and by hysteria. Care and experience are therefore necessary to form a judgment from such data.

(3) The effect of treatment and the evidence of past organic lesions which have been arrested, may confirm the diagnosis. Upon active syphilitic processes iodide of potassium given in sufficient doses has a more prompt effect than on any other form of disease; so that when the progress of nervous disease is checked or reversed by the action of this remedy, the fact may be reckoned as confirmatory evidence of syphilis. The same may be said of mercury. We must of course assure ourselves that the disease is progressive and its subsidence spontaneous. For instance, a hemiplegic attack may be treated with iodides from the commencement, and progressively improves; but this proves nothing, since the natural tendency here is towards improvement.

We cannot maintain the converse proposition, that where antisyphilitics do not cure, the disease is not syphilitic. They will not cure when there has been

destruction of nerve-tissue, as when a gumma has destroyed the cerebral cortex, or softening has taken place in consequence of arterial thrombosis.

Treatment.—As to the treatment of cerebral syphilis and nervous syphilis in general, where symptoms arise which we conclude, or even reasonably suspect, to be due to syphilis, the patient should be immediately and energetically treated with iodides or mercury, or both. Iodide of potassium in large doses is said to act more quickly than mercury. Ten grains three times daily should be given at once, and the dose rapidly raised to twenty or thirty grains; or in urgent cases we may begin with the larger doses. It is important often to gain time and catch up the disease (so to speak) before tissue has been destroyed which cannot be replaced. Even larger doses have been given. The drug may be given by enema if the patient cannot take it by the mouth. Treatment by iodide should be supplemented by treatment with mercury. This may be given in any of the usual ways: inunction is often a convenient plan. It is advisable to give a prolonged course of mercury, even in cases where cure is apparently rapid; for lesions may exist (such as arterial disease) without obvious manifestations, and relapses have to be obviated as far as possible.

It is no exaggeration to say that by prompt and sufficient antisyphilitic treatment, in an early stage of the disease, life and eyesight, and use of limbs, have been saved. In cases of longer standing such good results cannot be expected; for probably the disease will have advanced beyond the stage of deposit to actual destruction of nerve-tissue. This we cannot repair, nor stop the secondary degenerations that may follow it; but we may still perhaps, by antisyphilitics, remove deposits, relieve pressure on cells and fibres, or at least prevent the occurrence of further mischief.

Mercury is said to be efficient in some cases upon

which the iodides produce no effect. In arterial syphilis it is said to be decidedly the better drug. It is also said that after prolonged treatment with iodine patients become so saturated with it that it loses all curative or preventive powers. The general conclusion would seem to be that we should use iodides to make a first and quick impression on the disease, and a mercurial course to ensure permanent benefit.

For other methods of treatment, such as Zittman's method, hydropathics, the Aix treatment, &c., we must refer our readers to larger works.

Nervous disease from hereditary syphilis.—In hereditary syphilis nervous affections occur which anatomically, at any rate, correspond to those of the acquired disease. For instance, there may be gummata of the brain, infiltrations of the nerve-roots or of the meninges, or even arterial disease. Sometimes we find in children, apparently as a result of hereditary syphilis, a diffuse induration of the cortex cerebri, and occasionally of the deeper parts of the brain and the cord also. This is caused by the growth of a fibroid tissue; atrophy of the cortical cells and other nerve elements takes place. In hereditary syphilis gummata of the cord and local softenings of the cord are said to be rare, common as they are in acquired syphilis. The commonest clinical manifestations are these: convulsion-fits, either unilateral and presumably due to a meningeal lesion, or general, like those of true epilepsy; hemiplegia, or sometimes a "cerebral di-plegia;" paralysis of cranial nerves. In a rare case witnessed by the author, a young woman with hereditary syphilis exhibited a fusiform swelling upon one median nerve with paralysis in the district supplied by it. She afterwards had some epileptiform convulsions. Syphilitic children may become the subjects of a progressive dementia, the development of which is not unfrequently attended by fits or by hemiplegic attacks. In cases of this kind has been found

post-mortem the induration of the nerve-centres, which we have just noticed. It may be combined with meningitis or arterial disease, or may stand alone. Lastly, it is not impossible that syphilis in the parents may contribute to the production of neuroses and nervous degenerations in their offspring ; but this is a subject on which we know but little.

CHAPTER VIII.

DISEASES OF WHICH THE ORGANIC BASIS IS NOT KNOWN.

We must now pass to a large and important class of diseases, which differ from those we have hitherto considered, in the fact that their morbid anatomy is unknown to us. Some of them, indeed, have such definite symptoms and so definite a course as to indicate an organic basis not yet discovered; of others (and notably those which are commonly called "hysterical"), the manifestations are so varied and capable of such rapid change, that we can hardly hope to trace back the perverted nervous activity to any structural change.

EPILEPSY.

The epileptic fit.—In epilepsy the most striking feature is the "fit." Let us describe briefly an epileptic fit. The patient, who may appear to be in good health, suddenly, or after some slight warning, loses consciousness, and falls, uttering it may be a peculiar cry as he falls. His face is pale, his eyes open and staring, perhaps rolled upwards or sidewards; his limbs and face are contorted and for the moment fixed, and his breath held; then the face becomes blue, and gradually the fixed spasm gives place to a series of convulsive movements. These again pass off, and in some two or three minutes from the commencement he is lying deeply asleep and exhausted. The tongue is very frequently bitten; the

excreta, especially the urine, may be passed under him. There are two other kinds of "fit" with which we may contrast this.

Other types of fit.—(*a*) The epileptiform convulsion, or Jacksonian epilepsy. In this, as we have already pointed out, the convulsions begin in a definite member and spread in a definite way; consciousness is not lost (if at all) till they have spread over a considerable area, so that the patient is a witness of his own fit; a localised paralysis often succeeds the fit. Whereas in typical epilepsy consciousness is lost suddenly, quickly, and early, the muscular spasm is general from the outset, or at least follows no definite order; and afterwards, instead of a localised paralysis, there is a general paralysis in the shape of coma. (*β*) The hysteroid fit. In this there may be a sudden and no doubt real loss of consciousness; but it would appear to be less deep; thus, in falling, the patient rarely injures herself, neither (as a rule) is the tongue caught by the teeth so as to be severely bitten. She can, moreover, be roused by the application of such stimuli as cold water and faradisation. The convulsions are not mere muscular spasms, but movements which seem to be guided by some idea—scratching, biting, kicking, and the like. They may continue for much longer than true epileptic convulsions: an hour or more. The patient may regain consciousness suddenly and completely, without the post-epileptic sleep. The urine is not passed involuntarily, as a rule. The eyes are usually closed, and the lids resist when we try to open them.

It is important to bear in mind these different kinds of "fits." The usual import of Jacksonian epilepsy we have already pointed out; the prognosis and treatment of hysteroid fits may be quite different from that of true epilepsy. But, though such types exist, many cases will be found in practice of a mixed character, which it is impossible to put into one or the other category.

Preliminary phenomena—the "aura."—We must consider further the epileptic fit and its accessories. The approach of a fit may be heralded for a day or two beforehand by changes in the patient's mental state, such as the access of forgetfulness, irritability, despondent feelings, or by a feeling of exhilaration and *bien-être;* or perhaps by startings or twitchings of the limbs. Or it may take him while in his usual health. In any case, the immediate warning, or "aura," is of more importance. For not every epileptic fit begins with absolute suddenness; there is often some short preliminary to the loss of consciousness. The "aura" is tolerably constant for each particular case, but in different cases is of very various kinds. It may consist of strange sensations, tingling, numbness, pain, &c., in a limb or limbs: twitching of a limb, or movements beginning in a definite part (like those of Jacksonian epilepsy);* phenomena referable to the special senses, either so-called "crude" sensations of lights, colours, balls of fire, ringing and whistling noises, disagreeable tastes and smells, or more elaborate sense-phenomena, such as visions, spoken words; visceral phenomena, such as palpitation, disagreeable feelings in the epigastrium, &c.; mental phenomena, such as dreamy states, misty memories, feelings of having been in the same place or heard the same words before. The patient often states that the warning is "giddiness." On inquiry, we sometimes find that this means a feeling of faintness; but sometimes he means by it a true feeling of vertigo—that is, of movement. A feeling of turning round is the commonest; and this corresponds to the fact that at the commencement of the fit the head and eyes often turn to one side, or the whole body may rotate round and round.

Post-epileptic states—automatism.—In the

* When the aura gradually ascends up a limb, the fit can sometimes be averted by a tight ligature round the limb, or by the previous application of a circular blister.

period that immediately succeeds the convulsions, it has been noticed that for a brief time the tendon-reactions are absent, they then quickly return and become exaggerated, there may even be ankle-clonus. Deep sleep most commonly succeeds the epileptic attack; but sometimes this is replaced by a state in which the patient goes through certain actions automatically, or again he may appear to be actuated by delusions, or may become delirious and violent. The automatic actions are often those which the patient is accustomed to in his ordinary life, such as washing, undressing, &c., but they may be far more complicated. It is in this post-epileptic state that patients have committed crimes, made false accusations against others, and done various acts of which they are entirely unconscious when they have completely recovered themselves. Such automatism is more common after the minor epileptic attacks called "petit-mal" than after downright convulsions; indeed, many authors think that an epileptic may pass into a state of automatism without even an attack of "petit-mal"; it is then called the equivalent of the fit. After a severe epileptic attack, the patient has generally a headache and experiences a certain loss of memory and mental confusion.

"**Petit mal**" is an important factor in epilepsy. It is an epileptic fit shorn of its convulsions, and in which the loss of consciousness is reduced to an extremely short duration. The patient may simply turn pale, stare vacantly, twitch a little, drop what he is holding, and then recover himself. Or the attack may be so transitory as scarcely to interrupt him even in such elaborate actions as reading, playing an instrument, &c. There are many degrees of petit mal, so that patients themselves will classify their "sensations" and "turns" into slight and severe. We may reckon also as petit-mal those abortive attacks in which the aura, usually well known to the patient, makes its appearance, but the convulsion-fit does not follow.

According to Trousseau, attacks of petit mal have a more pernicious effect on the nervous system generally than have the major attacks; this may be because they are more frequently repeated. As we have stated, they are frequently followed by curious post-epileptic conditions. Petit-mal may precede, even by several years, the development of the typical convulsions; this we should remember when called to treat obscure symptoms of giddiness, faintness, and anomalous sensations in young people. We should remember, too, in fixing the duration of an epilepsy, to ask whether the patient suffered from such symptoms before he actually had fits.

Some epileptics have fits only at night, or indeed in sleep; and they may have no knowledge of these, except they find their tongue bitten, their bed wetted, or their limbs sore and wearied. Others have fits only after waking from sleep, and therefore chiefly in the morning: others in the day only. Or they may come according to no rule of time.

Time and frequency of fits.—The frequency with which fits are repeated varies much, not only in different cases, but in the same patient. Perhaps it is most common to have long intervals, months or longer, at the commencement of a case, and as the fits are repeated they tend to become more frequent. In some cases a certain periodicity can be traced. Some patients will have none for many months, and then have them in batches. The "status epilepticus" is a condition in which the patient has a series of fits, entering upon a second fit before he is well recovered from the preceding. This is an alarming state of affairs, and death (which is not common as a direct result of epileptic fits) may occur in it. A want of regularity in the recurrence of the fits will not surprise us if we reflect—first, that the laws according to which the disease itself develops are practically unknown to us; secondly, that many circumstances may act either as the determining causes of fits or as agents in warding them off.

Causes which favour or prevent the occurrence of fits.—We will mention some of these circumstances, though we cannot speak with much generality, for the influence of his surroundings is a question that should be studied specially for each epileptic. All sources of irritation, mental and bodily, may favour the production of a fit. Examples are: dentition, gastro-intestinal irritation from indigestible food or from worms (in children particularly); genital irritation due to the onset of puberty, to menstruation, to coitus (sometimes), to masturbation; injuries, particularly blows on the head, the irritation of old wounds and scars; ear-disease, changes of season and climate; divers states of nutrition; excess in alcohol; mental states, such as worry and anxiety, fright, passion, excitement. The badly constituted nerve-centres of the epileptic respond to the ordinary stimuli of life by a manifestation of his disease. Some conditions appear to ward off fits. During acute diseases they often cease. They may disappear while a suppurating wound or sore is open, to reappear when it closes. Practical use has been made of this fact by the application of setons for the relief of epilepsy. Many epileptic women are free from their fits during pregnancy. This may be due to the disappearance of menstruation, for fits are usually worse about the time of the menses. Abstinence from flesh food has been said to control epilepsy.*

Predisposing causes.—The deeper-lying causes of epilepsy—those, namely, which produce the vicious state of nervous system which lies at the bottom of its manifestations—are difficult to discuss. It is probably a matter of inheritance in most cases. It

* A hospital patient volunteered the following statement: When he was in work and could get food, he became subject to fits; these caused him to lose his situations, he then became destitute and semi-starved, and the fits would disappear. According to Dr. Haig's views, the production and excretion of uric acid (largely determined by the amount of meat eaten) are factors in the production of fits, as of other nervous phenomena.

is common to find in the ancestry or collateral branches of the epileptic's family a history of such neuroses as insanity, intemperance, hysteria, or epilepsy itself. The disease generally begins during the first half of life (say under thirty). Rickets in children favours the development of infantile convulsions; such convulsions may be repeated from time to time, thus passing over into ordinary epilepsy; or a child may get rid of its convulsions, but at some later period become epileptic. Acute diseases such as scarlet fever may favour the development of epilepsy. Puberty is a not uncommon time for the outbreak of epilepsy, or a later time, say between seventeen and twenty-five. The disease then may commence during all the time that the nervous system is undergoing development, adaptation, and consolidation. It may commence too when the nervous system is breaking down through senility. When it commences during adult life, we should look for such extraneous causes as alcoholism, syphilis, or Bright's disease.

Course of epilepsy.—The course of the disease is not very regular. It is generally held that the longer the fits go unchecked, the more certain and frequent does their repetition become. Their type may change from nocturnal to diurnal, from severe convulsions to petit mal, or reversely. Usually the patient's health is good in the intervals of the fits, while the disease is in an early stage. As the disease proceeds, the headaches and the loss of memory, which we have mentioned to be a temporary effect of the fits, become a source of permanent trouble. A deterioration of intelligence and character sets in; he is apt to become more and more forgetful, and stupid; despondent, irritable, suspicious, and impulsive. A chronic epileptic often has coarse features, with a downcast, dazed, or stolid expression. Some patients become such dangerous lunatics at the time of their attacks that confinement is imperative; others become permanently insane. As to termination—death in a fit is not common, but it may result from injuries

sustained in a fit; many patients drift into asylums; others continue chronic epileptics, till intercurrent disease terminates their lives. When once the disease is established, a complete and permanent recovery is not probable. A patient may under favourable circumstances have such long intervals between his fits as to be practically well; but it would be unsafe to say that he will be liable to them no more.

Treatment.—In the treatment of epilepsy drugs play a considerable part. No one can doubt the favourable influence of bromides. Bromide of potassium is the salt generally given; bromide of sodium or of ammonium, or a mixture of the three salts, may occasionally have advantages. We should find, if possible, the minimum dose that will control the fits (pot. bromid. gr. xv–xx ter die, may be called an average dose), and administer this for a lengthened period. Say that at the end of a year there have been no fits, then the dose may be lessened, or the medicine given less often, and thus cautiously dropped. This of course presumes that the patient bears the medicine well; luckily epileptics are tolerant of it, and can often take large doses for long periods. Its ill effects may be as follows: (*a*) The bromide rash, a kind of acne, often severe and pustular. The addition of arsenic may relieve this. (*β*) The state called "bromism." The patient loses appetite and flesh, and becomes sleepy, listless, and lethargic. This may pass off when the bromide is stopped, though his fits may return. We must notice, however, that the occurrence of fits, quite independently of medicine, sometimes seems to clear the atmosphere of an epileptic's mind.

No drug can, upon the whole, compare with the bromides, but others may do good: belladonna (tinct. bellad. ℥v–℥xxx ter die); zinc salts (zinci valerian. gr. iij–gr. v); borax, gr. x–gr. xxx. Borax is said to influence some cases not amenable to bromides;* it

* In some chronic cases, where bromide has been administered for long periods, I have found a change to borax do great good.

may produce nausea and vomiting, or an eczematous rash. Nitro-glycerin has been recommended.

As valuable adjuncts to bromide we may mention digitalis, belladonna, iodide of potassium, arsenic. Salicylate of soda has been recommended on the ground that it favours the excretion of uric acid.* No doubt some of these drugs enable us to minimise the dose of bromide.

Petit mal is often very intractable; and bromide certainly has far less control over it than over the severer fits.

The general treatment of epilepsy is an important but wide question. Did we know, and could we control, all the exciting causes of our patient's fits, we might do much to cure him. A few general remarks may be made. Children's education should not be neglected, but they must not be pressed with examinations and the like; a large and rough school is evidently an unsuitable place for them. Girls and boys must be carefully watched through those critical times, the onset of menstruation and puberty. Epileptics should cultivate regular hours; should have a plain, wholesome diet, without too much meat and with little, if any, alcohol; should avoid both constipation and violent purges; should aim at securing a quiet, uneventful life, free from excitement, yet regularly occupied, in some trade or profession if they can manage it; outdoor work is the best for them, but the risk of sudden falls must be remembered. Marriage is no cure for epileptics, and as a rule they should be dissuaded from it, both on account of the increased anxieties and responsibilities it entails, and of the risk that they will propagate a neurotic offspring.

Little can be done for the patient during the fit itself, save such obvious precautions as preventing injury during the fall, preventing tongue-biting by

* *Vide* footnote to p. 290.

slipping something between the teeth, such as a spoon, handle of a brush, &c., loosening tight clothing from about his neck, &c. The fit must run its course, and it is best not to disturb the after-sleep. For the status epilepticus it is right to try such remedies as these: inhalation of chloroform, chloral, bromide of potassium in large doses (by enema if necessary); an ice-cap to the head. If there seems danger of exhaustion, the patient must be fed and brandy given, by the mouth or rectum.

Chorea.—In St. Vitus' dance, chorea Sancti Viti, or briefly "chorea," the patient is subject to involuntary movements of the face, tongue, limbs, and trunk, sudden, abrupt, and generally variable; not spasms of isolated muscles, but co-ordinate movements, as if from a reflex start, or a quick voluntary impulse on the part of the patient. The face grimaces; the limbs, as has been aptly said, appear to be tossed about by some unseen agency. The movements may go on while the patient is lying still, or may interrupt him in the performance of voluntary actions; they are worse while he is being watched or examined, and cease while he is asleep. In a mild case there may be little more than occasional twitchings of the face or limbs, difficult to distinguish from mere nervousness. The patient drops things involuntarily, and gets into trouble for awkwardness or carelessness. After protrusion of the tongue, he draws it back with a snap. There is a peculiar abruptness of voluntary movement and gesture. Cases often begin in such a way. From such mild manifestations there is every gradation up to wild, constant jactitation of the limbs and body, preventing sleep and feeding, and leading to serious injury or exhaustion. The movements may be limited to the face or one limb (which is important as suggesting the cerebral origin of the disease), to the face and limbs of one side, or they may be limited in area at first, and then become general. Where the movements are specially marked

on the right side of the body, there are often difficulties of speech.

The movements are the most striking and characteristic mark of the disease; but some other points must be noted: (1) Some few cases appear to be characterised by paralysis, rather than movement. This "choreic paralysis" generally affects one arm; the limb is limp, not rigid; there is neither exaggeration of tendon-reaction, wasting of muscle, nor change in electrical reaction. The onset is not sudden, but subacute, coming on in the course of a few days, or a week or two. These characters are almost enough to differentiate it from other forms of paralysis in young people, and on inquiry or close observation some evidence of twitching movements will probably be found. (2) Mental affection. In the presence of the facial spasms and the abruptness of manner which form part of the disease, it may be difficult to judge of minor modifications of disposition and intelligence. Probably there is some slight mental disturbance in many cases: in a few the patient becomes absolutely, though temporarily, insane. The disease has then been called "chorea insaniens." (3) In a large proportion of instances there is evidence of valvular disease of the heart. The mitral valve is much more frequently affected than the aortic. The murmur may be found on first examination, or may develop during the course of the disease. Permanent valvular disease, with secondary hypertrophy or dilatation, may remain. Post-mortem, in the (unfrequent) event of death during chorea, a ring of tiny nodules (granulations) may be seen near the edge of the mitral valve.

Chorea is a disease of children, before puberty of boys and girls alike, and of young women. Its antecedents and affinities are very interesting. On the one hand, it appears due to agencies that directly affect the nervous system. Fright is the commonest and best established of these causes; mental strains of other kinds might be added. Thus, children pre-

paring for School Board examinations, when ill-equipped in brain and body for such a task, break down in this manner; and the chorea of pregnancy is apt to be most severe, when the pregnancy has followed a seduction. Again, in the patient's family, or sometimes in the patient's self, we may expect a history of other neuroses.* On the other hand, it is closely connected with rheumatism. Chorea may develop during acute rheumatism, or rheumatism during chorea; the same person may have at separate times rheumatism or chorea; and the affection of the cardiac valves is a common feature in both diseases. Again, if in the patient's own person chorea appears to stand alone, we yet may frequently find on inquiry into the family history that other members have suffered from rheumatism. It is not unreasonable to suppose that in many instances at least chorea and rheumatism are but different manifestations of some one diathesis. More precise explanations of the disease have, it is true, been attempted. One of these is the "embolic" theory. Starting from the fact of valvular disease, it is maintained that the disease is due to minute fragments of fibrin swept from the valves into the arteries of the cortex cerebri, and there forming emboli, the effect of which is to irritate the cortex. For this theory there is no anatomical evidence, and it will hardly explain the course of the disease. We must admit that as yet we do not know the exact causation of chorea.

The movements of chorea may develop from the small beginnings which we have described, or they may arise somewhat suddenly. Some cases recover within six weeks or so; others recover imperfectly

* Some years ago, the author had under treatment a young man who suffered from epilepsy; the convulsions were largely unilateral. His fits passed off, and he was attacked by movements differing in no respect from those of ordinary chorea on that side. When this attack subsided his fits began again.

and continue for a long time subject to a mild form of the disease. Relapses are frequent. It is rare for death to occur, but in the severe cases this sometimes happens.

Treatment.—Obviously it is advisable to put the patient in quiet and comfortable surroundings; the mere removal from a poor home to hospital may do much good. A case of any severity must be put to bed; and when the movements are very violent and uncontrollable, the bed must be so arranged that the limbs cannot be injured against its sides, nor the body be thrown out of it. Plenty of nourishing food must be given, finely cut up, or in a fluid form where mastication and swallowing are impeded, or by the nasal tube if necessary. Bad cases will need alcohol also. For such bad cases chloral administered in full doses appears to be the best drug; in less acute and more persistent cases arsenic should be given, and that freely. Where arsenic fails, sulphate of zinc often does good. In anæmic, poorly nourished children, iron and cod-liver oil may be administered at the same time as the arsenic.

"Hereditary" chorea. — We may mention here a rare form of disease, characterised by movements like those of chorea, but which differs in the following respects. It attacks older people, males or females past middle age. Its development is more gradual, its course chronic, and it is generally thought to be incurable. It is often accompanied by a progressive dementia. Lastly, it runs in families. It has been called "chorea of the adult," or "hereditary chorea."*

Tetany.—Tetany (which has nothing to do with the graver disease, tetanus) is characterised by a peculiar tonic spasm of the hands, and sometimes of the feet as well. The fingers take a position mainly

* Sometimes also "Huntingdon's chorea"; just as St. Vitus's dance is called "chorea of Sydenham."

due to spasm of the interossei; the two most distal joints are in extension, and the metacarpo-phalangeal joint flexed, while the points of the fingers and thumb are approximated so that they form a sort of cone;* there may be also some flexion of the wrist. The spasms generally come on during sleep, they may pass off during the day to recur the next night, or they may be continuous. A painful cramp accompanies them. Trousseau says that they can be brought on by pressure on the nerve-trunks or arteries of the limb, until the disease is extinct. Swelling on the back of the wrists is sometimes complained of.

In addition to the actual spasm, we must notice:

(1) The phenomenon of "facial irritability." If the finger-nail be drawn somewhat roughly across the facial nerve, where it crosses from the ear towards the eye, the eyelid blinks from a twitch of the orbicularis muscle.

(2) There is, according to Erb, increased electro-irritability of the nerve-trunks generally, but without qualitative changes. This may not be easy to demonstrate.

Tetany is most common in young children. In them it is accompanied by, and probably due to, rickets. In conjunction with it, laryngismus stridulus often occurs; sometimes also the child has convulsions. It is also seen in women, either in young women of hysterical temperament without other apparent cause, or in women who are debilitated by lactation.† Epidemics of tetany, probably of hysterical type, have been reported from girls' schools. Bromide of potassium, with cod-liver oil, and suitable dieting in the case of rickety children, forms an appropriate remedy.

* The posture is usually compared to that which the obstetrician gives his hand in order to insert it into the uterus.

† Tetany has also been ascribed to gastric disturbance (the disease has followed the operation of washing out the stomach), and to the removal of the thyroid gland.

Paralysis agitans.—A typical case of paralysis agitans will probably present the following features: The patient is usually in advancing years, of either sex, perhaps a man most commonly, and complains of weakness and tremor. The tremors, as we have already said, are rhythmical, rapid, and commonly small in amplitude; they affect particularly the muscles of the thumb and fingers. The movement has been compared to that of making pills or rolling a cigarette. They may involve the wrist-joint and larger segments of the limb, and then produce a coarser flapping movement. They have this characteristic, that they are present while the patient rests the limb, and often (at least in the early stage) stop when he begins to use it. The legs may shake also, and the tongue. The head, according to some authorities, does not shake. Along with the tremor, and sometimes independently of it, there exists an embarrassment of movement producing the effects of paralysis, though there may not be actual loss of muscular power. The patient's aspect is peculiar. His face and eyes have an immobile look. His head is bent a little forward and held stiffly; his trunk bent forward at the hips. His speech is deliberate, his voice is sometimes piping, like an old man upon the stage (as Dr. Buzzard puts it), sometimes whispering. Sometimes he cannot walk without hurrying, as if he were trying to prevent himself falling forward. Sometimes a peculiar phenomenon is seen: when he has once begun to walk he cannot stop himself; or again, if given a push backward, he has to go backwards until some obstacle stops him. He is often very sensitive to heat, and complains of spontaneous "flushes of heat." He may be tormented by a sense of restlessness both by night and by day, and is apt to be sleepless at night. He will sometimes say that he feels the tremor "internally."

There is no affection of sensibility. The tendon-

reactions are generally normal, sometimes exaggerated. The mind is usually unaffected, although the patient is (not unnaturally) disposed to be gloomy about himself; but in some instances insanity has developed.

Among the antecedents of paralysis agitans we often find such influences as these: exposure to damp and cold, rheumatism, mental shocks, particularly fright, or prolonged mental strains as from failure in business, family troubles, and disappointments. Occasionally, though rarely, two or more members of a family suffer. As a rule, the disease begins gradually: tremor is noticed in one hand, whence it may spread either to the leg, so as to assume an hemiplegic distribution, or to the other hand, and then to the legs. The tremor is sometimes preceded by pains in the limbs. In some instances the tremor gets less, but the weakness increases. Indeed, there are cases which exhibit no obvious tremor, and here the diagnosis may be difficult, the peculiarities of physiognomy and of gait being the chief guide. Some cases develop in the form of a paraplegia, with a very gradual onset, and without obvious causation. It is less common for the disease to begin abruptly, but this has been observed, especially after frights. The disease progresses slowly, but very surely; the tremor becomes general, the weakness increases, till at last the patient cannot walk, cannot stand, and may lose the use of his hands. When once bedridden he is liable to the complications of bedsores, cystitis, &c., unless he dies from some intercurrent malady.

Treatment.—Treatment, it is to be feared, will do little to stop the disease; but nevertheless some relief may be obtained. Electricity, either in the form of the constant current or of static electricity, has done some good. Of drugs, iron in large doses, or nux vomica, or strychnia, undoubtedly benefit

some patients; iodide of potassium or arsenic may also be given.

Diagnosis.—A well-marked case is easy to recognise; but in others there may be difficulty of diagnosis. The disease has to be distinguished from disseminated sclerosis; the characters of the movements are different, there is no nystagmus, and as a rule no excess of tendon-reactions, and the age of the patient is more advanced; also from general paralysis, from hysterical tremor, from toxic tremors as of mercury, lead, alcohol. What line can be drawn between the tremor of paralysis agitans and the tremor of senility is difficult to say.

Writer's cramp.—There is a class of nervous affections which have been called "neuroses of occupation," or professional "cramps," and which originate as follows:—In the course of a man's avocation he may be called upon to perform repeatedly, and for a length of time, some movement or movements of a highly specialised nature, such as writing, playing a musical instrument, working a telegraph, and so on. As a result the motor apparatus concerned in the movement breaks down, so that on attempting to perform it he is seized either by motor spasms or, it may be, by pains in the hand or arm, which prevent him from going on. There may be no loss of gross muscular power, no wasting or other sign of organic disease; and for all other purposes except his particular work his hand may be as useful as ever. The commonest and most typical affection of this kind is that known as writer's cramp. This is apt to attack clerks whose business necessitates their writing many hours a day. Often the patients are poorly nourished, anæmic people of a neurotic type, or with neurotic family history. In such people writer's cramp may appear even with a limited amount of writing, say three or four hours daily. Some kinds of writing seem to favour its development more than others, those, for instance, on which the attention must be kept

closely fixed—those which necessitate an uncomfortable position of the hand,* steel pens rather than quills, &c. An extra long bout of writing often determines the appearance of the disease. Then whenever the patient sets about writing and has got through a few lines, he is either seized with a pain in the hand or arm which causes him to desist, or more commonly muscular spasm arises, his fingers assume a cramped position which renders further writing impossible, or spasmodic movements set in which may even fling the pen from his fingers. If the patient learns to write in such a way as to avoid the use of the small hand-muscles, say with movement of the wrist or elbow, or even with the left hand, it may happen that after a time the affection begins in the newly employed parts. This would seem to show that the disease is not merely a peripheral affection, but that its cause is in the nerve-centres themselves. The best plan for the patient is to give up the occupation of writing. If this cannot be done, a temporary holiday should be secured, and the writing minimised subsequently. Attention should be given to the general health. Local treatment of the following kind is advantageous: either massage (which is said to have effected cures), galvanisation,† or moderate faradisation of the hand and forearm, repeated regularly and frequently for a considerable time.

Migraine.—Hemicrania, which name has been abbreviated into migraine or megrim, consists essen-

* Thus a post-office official with this affection informed me that a certain kind of writing in connection with the telegraph in which the hand is kept in one place, and not drawn along from left to right as in ordinary writing, is particularly apt to set up writer's cramp. His statement was drawn from observation of his own case, and that of several fellow-clerks.

† This may be given after the plan recommended by Dr. Poore. A constant current is passed from shoulder to hand, and the patient is instructed to perform regular rhythmical movements of the hand and fingers during its passage.

tially of a paroxysmal headache, often limited (as the name implies) to one side of the head, with which headache other phenomena may be associated. The paroxysms may appear at regular intervals, such as a week or a month, or they may be quite irregular in their appearance. They often are determined by some such circumstances as these: overwork, irregularity in meals, indigestible food (from which cause and from the presence of vomiting migraine is often called "bilious headache"), strain upon the eyes, exposure to bright lights. These last causes harmonise with the fact that visual symptoms are often present. Picture galleries and theatres are thus potent provocatives. But co-operating with these external causes there must be a predisposition on the part of the patient. The exact nature of this predisposition we do not know; it may be that nervous instability, often inherited, which manifests itself in other patients as epilepsy* and other neuroses; it may be (as strongly urged by Dr. Haig) the gouty diathesis. Neither do we know the exact pathology of the paroxysm; it has been ascribed to spasm and to dilatation of the cerebral vessels, and that grand *deus ex machinâ*, the sympathetic nerve, has been invoked for the explanation of this spasm; again, it has been likened to the explosions of epilepsy taking place in a sensory sphere of the cortex. An attack may be heralded by drowsiness and depression, or occasionally with extreme irritability; the pain in the head then begins, often in one definite spot, over one eye or in one temple, or at least upon one side of the head; thence it radiates and may become general. Usually the migraine terminates in the course of a

* It is stated by good authorities that epilepsy and migraine do not occur in the same subject. A patient attending at Queen's Square Hospital for typical migraine has described to me attacks which pass over into convulsions with unconsciousness. Dr. Liveing and others recognise such an interchange between epilepsy and migraine.

day ; sometimes it lasts two or three days. This may constitute the whole of the attack, but often there are additional symptoms. The commonest is vomiting, which occurs usually towards the end of the attack, and therefore appears to relieve the headache. Visual symptoms are common also; the patient may see flashes of light, colours, &c., or sometimes an elaborate zigzag of light forming a kind of fortification or battlement pattern ; dark spots may appear in his field of vision, or he may become temporarily hemianopic. Less commonly the limbs are affected by tingling and numbness, anæsthesia, or motor paralysis, in the arm of one side, or having a hemiplegic distribution. There may be temporary aphasia. These forms of paralysis end with the attack. Another kind, which although transitory lasts longer than these, is paralysis of the third or of the facial nerve of one side; recurrent paralysis in these districts seems often to originate in attacks of migraine. Sometimes in a severe attack of migraine the drowsiness and lethargy become so great as almost to amount to coma, so that were it not for the history of previous attacks we might be led to suspect a cerebral tumour.

Treatment.—In a bad attack of migraine the patient should remain recumbent, in a darkened room protected from noise, and take only fluid food in small quantities at a time. Antipyrin should be given, say in 10-grain doses every hour, up to three or four doses; its effect is usually rapid and satisfactory ; and the more so, the earlier it is given in the attack. Other medicines are guarana, citrate of caffein, strong coffee or tea, alcohol. Our treatment of the case during the intervals must be guided by our surmises as to its causation. Country air, outdoor life, iron and other tonics may be best for anæmic, ill-developed patients; those with nervous heredity may have a course of bromides in small doses; alkaline bicarbonates with gentian or calumba, and attention to diet, benefit cases where digestion is at fault.

The "uric acid headache."—According to Dr. Haig, many cases of migraine are to be reckoned as a "uric acid" headache. The radical fault is the presence in the system of an excess of uric acid (we may say, the gouty diathesis). This may be either locked up in the tissues or pass into the blood, whence it is excreted by the urine. Such paroxysmal headaches are marked by the fact that during the paroxysm there is an excess of uric acid in the urine. This makes it probable that they are caused by its presence in the circulating blood. They may be relieved by the administration of acids (say a drachm of dilute nitro-hydrochloric acid in divided doses); which, rendering the blood more acid, drives the uric acid back into the tissue. But during the interval we should aim at (1) diminishing the production of uric acid, which is best effected by cutting off from the diet all forms of butcher's-meat, malt liquors, and strong wines; (2) aiding its excretion as fast as formed, which can be done by prolonged administration of salicylates, or alkalies.

Neuralgia.—We have already alluded to the subject of neuralgia and the nerves (trigeminal, sciatic, cervico-occipital, intercostal) which it principally affects. The pain of neuralgia is often of two kinds: a dull continuous ache, principally in the site of the nerve-trunk; or sharp darting twinges, principally in the peripheral distribution of the nerve. Both these kinds of pains, but particularly the latter, are apt to occur in paroxysms, or at least to have paroxysmal exacerbations. The pain of a neuralgia, in the strict sense of the term, is limited to the area of a particular nerve, but as it gets worse its original distribution may be concealed by irradiation to other parts. (Painful affections of many organs, for which no cause can be found, are often loosely spoken of as "neuralgia" of the uterus, kidney, &c.) Lastly, we speak of a simple neuralgia only where we have reason to believe that there is neither organic disease

of the nerve itself, nor disease of the parts around directly invading or pressing upon it. It is most important both for prognosis and treatment to ascertain that the neuralgia is not thus symptomatic of organic disease. Bilateral neuralgia suggests disease of the nerve-centres, or of the bones or membranes around the spinal nerve-roots. The causes of neuralgia, like those of many other neuroses, are partly reflex, partly constitutional. A source of peripheral irritation, involving the termination of a branch, may produce neuralgia either of that branch or of others, or sometimes even of more distant nerves. Thus, a face-ache may be dependent on bad teeth, while the patient has no pain in the teeth, and denies that there is anything the matter with them. Such reflex causes of neuralgia must be always looked for, since in their removal lies the best chance of curing the disease. We may enumerate among them carious teeth (by far the commonest cause of facial neuralgia); anomalies of refraction and overstrain of the eyes as a cause of frontal neuralgia; inflammation of the nasal cavities, frontal sinuses, and of the ear in various neuralgiæ of the face and head; fæcal accumulations, ulceration of the cervix uteri, &c., in neuralgia of the lower extremities. Exposure to cold may be the starting-point of a neuralgia. Among constitutional causes, the commonest is general anæmia and mal-nutrition, which doubtless acts by setting up mal-nutrition of the nerve; a neurotic predisposition (both causes are seen at work in delicate young women of an hysterical temperament); fatigue and sleepless nights, the gouty diathesis, an inveterate taint from malaria, or from syphilis. Constipation is a condition which should be inquired after and treated. The general remedies—the reader will apportion them to their appropriate classes—are proper feeding (with a moderate allowance of good wine), rest from work, change of air and scene, quinine (the drug of most general application in

neuralgia), iron, bromides, alkalies with vegetable tonics, arsenic, iodides. In addition to this general treatment, and the removal of such sources of irritation as we may have been able to find, remedies for the neuralgia itself will always be in demand. There is a long list of such remedies—quinine, gelsemin, croton-chloral, antipyrin, citrate of caffein, alcohol, internally; morphia by the mouth or subcutaneously; local applications of great variety—fomentations, simple or with poppy-heads, menthol, chloral-camphor pigmentation, belladonna, aconite, cocain, morphia; counter-irritants—the galvanic current applied without make or break, and with the anode over the painful nerve. Lastly, in severe and obstinate cases, and particularly in the excruciating paroxysmal neuralgia known as "epileptiform neuralgia," or "tic doloureux," surgical aid may be invoked with the view either of stretching the nerve, of excising a portion, or even removing some of its deeply seated parts, such as Meckel's ganglion.

Additional symptoms.—In addition to the pain of neuralgia, there may be sometimes tenderness. This may be diffuse, spread over the whole area of the neuralgic pain; but more commonly it is localised at certain points in the course of the nerve, chiefly those where it emerges from beneath bones and fasciæ. These "tender points" we have already noticed. Sometimes an attack of neuralgia leaves a temporary anæsthesia. Trophic changes are sometimes witnessed; thus, the hair may turn grey in the district of a nerve affected with neuralgia; or an herpetic eruption may occur. The common affection, herpes zoster, or shingles, is generally preceded by intercostal neuralgia. Trophic changes in the muscles, such as wasting with reaction of degeneration, point to a neuritis rather than a neuralgia.

There are some important diseases affecting the nervous system, concerning which we believe it best

to refer our readers to text-books on general medicine and surgery; these are delirium tremens, tetanus, hydrophobia. Neither shall we discuss here the subjects of myxœdema, Graves' disease, Raynaud's disease, angina pectoris.

Hysteria.—The name hysteria, though doubtless old-fashioned and faulty, is still generally employed to denote a certain type of disease, whereof we will try to enumerate a few characteristics, though we shall not attempt a definition of it.

General characters.—The disease occurs chiefly in women, most frequently of all in young women; in girls and boys also; sometimes in men. It is not therefore necessarily connected with the uterus (as the name would imply); nor with the ovaries; nor indeed with the sexual system at all. It is essentially an affection of the central nervous system; but sources of peripheral irritation, particularly when seated in the sexual organs, may have a powerful influence in determining its outbreak. The symptoms of the disease are very variable; and they vary, not only in different patients, but in one and the same patient: so that mutability of symptoms, sudden appearance and sudden disappearance of them, often suggests to us that they have an hysterical basis. It is, on the other hand, possible for hysterical symptoms to persist unchanged for years. The disease may present a remarkable counterfeit of other nervous diseases; and this not merely of such general affections as epilepsy, &c., but of local nervous disease such as hemiplegia, paraplegia, or paralysis of still more local origin. On this account the name "neuro-mimesis," or, *anglice*, "nervous mimicry," is sometimes applied to it. Yet it depends, so far as we can at present determine, on no actual organic lesion. Hence death from hysteria is almost unknown, and the presence of certain symptoms (which point to organic disease) may be taken as negativing the diagnosis of hysteria. Such symp-

toms are—optic neuritis, optic atrophy, reflex iridoplegia, bedsores and cystitis, electrical reaction of degeneration in muscles, complete and permanent absence of knee-jerk. We may go further, and say that the more closely the grouping and sequence of symptoms resembles that which would follow any known local lesion, the less likely is the disease to be hysterical. Again, an emotional or volitional element is often distinctly present. We do not mean that mere psychological states can produce bodily disease, but that the circumstances under which strong emotions develop may produce, or perhaps remove, hysterical manifestations; and that circumstances under which the will becomes enfeebled or strengthened, favour or oppose respectively the development of hysteria. Lastly, hysteria is a genuine morbid state; it is not synonymous with humbug. Hysterical patients, like others, and perhaps more often than others, may practise deception; but this is no necessary part of the complaint; it is often improbable, and sometimes impossible, that they could manufacture the symptoms they exhibit.

Causation.—As in epilepsy, so in hysteria, the radical fault is nervous instability, whose exact nature is unknown to us. Such fault is in the graver cases often inherited: a family history of various neuroses may be elicited. It may be produced also by a life of luxury and idleness, leading to ennui and concentration of the thoughts upon self; and hence it is commonly thought that young women of the better classes are the most frequent victims of the disease. But it may be seen under quite opposite conditions in poorly fed, anæmic, overworked subjects. Sudden shocks of all kinds—frights, disappointments, griefs—prolonged strains of anxiety and overwork, are potent factors in its production. It may follow or accompany other diseases, either as part of the mental and bodily debility which results from any severe and long illness; or, it would seem, as a reflex result from disease

and irritation of the pelvic organs; or as an accompaniment of organic nervous diseases, such as hemiplegia, disseminated sclerosis, &c. Toxic influences produce it, alcoholism often, plumbism, and other such causes sometimes. Injuries may determine curious attacks of local hysterical paralysis, or a general and chronic form of neurosis which we shall describe later. According to Grasset, phthisis and hysteria have a close relationship; the two diseases frequently appearing in members of the same family.

Symptoms.—There is no definite course to a case of hysteria; sometimes one set of symptoms may be present, sometimes another. Even this feature of change is not invariable; some symptoms (notably that of paraplegia) may remain unchanged and unaccompanied by others for a long time. Hence we can only make an enumeration of such phenomena as may in various instances be found; they may be pieced together differently in different cases.

Fits.—These vary much in severity. In a slight hysterical attack there is no loss of consciousness, only a burst of tears or laughter, or both alternately, with some jactitation of the limbs perhaps, and a choking feeling at the throat. Or, again, the patient may lose consciousness and fall, remaining for a short time limp and motionless, or perhaps rigid. Or there may be convulsions, so that the attack bears a general resemblance to epilepsy. We have already contrasted these "hysteroid" attacks with those of epilepsy: we will again enumerate a few points of difference. In hysteria the face does not turn pale or blue, and is not drawn as a rule; the eyes are closed, not staring, the lids tremulous and resist opening; the tongue is not bitten, there is less foaming at the mouth; instead of uttering one solitary initial cry, the patient may scream, moan, or even talk throughout the attack; the movements are apt to be violent, so that several people are required to hold the patient; and purposive, not mere tonic and clonic spasms; and the face may

at the same time assume expressions of fear, rage, &c. The movements may go on for a very considerable time; the excreta are not passed involuntarily; rapid recovery may take place, and though the patient may be exhausted afterwards, the fit is not followed by the coma or the dazed mental condition so often seen in epilepsy. In spite of the fall and the violence, the patient usually sustains little real injury. The attack can often be cut short by appropriate remedies. In a long-continued hysterical attack the temperature does not rise to the height which it may reach in the "status epilepticus."

Hysterical fits, like epileptic fits, may be preceded by an aura; the most frequent warnings are sensations in the lower abdomen or epigastrium, palpitation, choking at the throat ("globus"), vertigo, noises in the ears.

Under the title of hystero-epilepsy, Charcot describes a still graver type of hysterical fit. This begins with an attack simulating true epilepsy—cry, loss of consciousness, tonic and clonic spasms, coma, perhaps even tongue-biting; next follows a period of wild and extravagant movements and postures, sometimes called "clownism." Opisthotonos is frequent, or an attitude like that of crucifixion. This passes into a stage wherein the patient expresses by action and gesture emotions such as fear, hatred, love, sometimes even acting out by voice as well as gesture some drama connected with her life-history. In the final stage she has hallucinations, seeing spectres, rats, and other objects which terrify her; and the attack may leave her hemiplegic, hemianæsthetic, with contractures of a limb or limbs, &c. Fully developed hystero-epilepsy appears to be unfrequent in this country. Lastly, it would seem that in some few cases, apparently purely hysterical, attacks may occur consisting of convulsions limited to one limb or part of it, without loss of consciousness, so as to simulate "Jacksonian epilepsy." Probably such con-

vulsions will have a less definite order of spread; and if there be optic neuritis, mere hysteria will of course be negatived.

A fit which can be provoked by touching or pressing some definite part of the body is more often than not hysterical.

Motor paralyses.—Hysterical hemiplegia may come on suddenly after a fit, or gradually. It usually affects the limbs only, not the face or tongue, neither (when right-sided) is there true aphasia. The paralysed limbs may be limp, or there may be contracture; if contracture be present it probably develops *pari passu* with the paralysis, and is not (as usually in organic disease) a late phenomenon. Hysterical paraplegia, like hemiplegia, may be sudden or gradual in development, flaccid or rigid in character. If both lower limbs be paralysed, without trace of rigidity, the knee-jerks normal or somewhat exaggerated, the muscles well nourished and normal in their electrical reactions, we have a condition (as pointed out by Dr. Buzzard) which tallies with no known organic disease, and is therefore probably hysterical. Unfortunately the legs in hysterical paralysis are very often rigid, and then it may be quite impossible to diagnose between hysteria and disease of the dorsal cord with descending sclerosis; the more so because such hysterical paraplegia may be a very permanent symptom, and may stand alone without other manifestations to guide us. Hysterical paralysis may spread from one limb to another, becoming more or less general. In such cases it is not unusual to find that the right hand is spared, so that the patient can feed herself and help herself to a certain extent. Paralysis of one limb only (monoplegia) is not unfrequent. This is often accompanied by contractures, and by anæsthesia of the limb. It is perhaps most frequent after some injury to the limb, and constitutes a kind of local hysteria.

Motor spasms of one kind or another are very

frequent in hysteria. Involuntary jerks of the limbs are often seen; sometimes there is a condition resembling chorea; sometimes one or both hands are affected with regular rhythmical flapping movements; sometimes there is a fine tremor seen when the hands are held out. The typical tremor of disseminated sclerosis, which begins when the patient endeavours to use her hand, causing large irregular wavy movements, and ceases again when the voluntary movement is completed, does not, according to Buzzard, occur in hysteria. This is an important observation, since the two diseases are often difficult to diagnose, and indeed may be commingled in one and the same patient. Hysterical contracture or rigidity is common. Thus we may have a paraplegia or a hemiplegia with rigidity; or one upper limb may become useless, anæsthetic, and rigid at all the joints, while the fingers (in particular) become tightly flexed into the palm. Or the fingers of one or both hands, and sometimes even the toes, may become thus flexed without any obvious palsy of other segments of the limbs. Fibrous adhesions about the joints may form when hysterical rigidity is long continued.

Affections of sensation.—Anæsthesia may occur in scattered patches, or, like motor palsy, be paraplegic, hemiplegic, or monoplegic in distribution. When it affects one limb (it often, as we have said, occurs along with paralysis and contracture of one upper limb), it involves the whole of the digits and the various segments from periphery to root, like a stocking or glove, according to the plan of cortical anæsthesia (*vide* p. 90). Hemianæsthesia involves one half the body, face, and scalp, with the limbs and special senses of the same side (as if from disease of the posterior part of the internal capsule).

Anæsthesia may exist in various degrees; its existence may be unknown till the physician has looked for it.

Analgesia occurs so that the patient does not feel faradisation, pricking, or even burns. Pain and

tenderness are frequent—pain in the forehead as if a nail were driven in, in the back, in a joint; tenderness along the spinal column, under the breast, and in the inguinal region. Tenderness to deep pressure in the left inguinal region has been referred to the ovary; such pressure may produce globus and other sensations, or even provoke an hysterical fit: some fits, on the other hand, can be cut short by this means. Inguinal tenderness may be witnessed even in hysterical men, so that its connection with the ovary may well be doubted.

Reflex actions may undergo modification. The tendon-reactions are often exaggerated, both where there is paralysis and where there is not; often there is a sort of attempt at ankle-clonus, less commonly a definite ankle-clonus.* But, so far as we know, the patellar tendon-reaction is never abolished by hysteria. As regards the skin-reflexes, the plantar reflex may be in abeyance, or it may take a great deal of tickling to obtain it; and the reflex normally obtainable by touching the back of the throat is often absent.

As to the organic reflexes, retention of urine is common enough in hysteria: involuntary or unconscious passage of the urine is far less common, but may yet take place in hysterical paraplegia (Buzzard).

All the *special senses* of one side may be affected, as we have said, in hysterical hemianæsthesia. Further, possible affections of the eyes are as follows: photophobia, amaurosis, or sometimes colour-blindness; or again concentric narrowing of the field of vision; monocular diplopia; ophthalmoplegia externa possibly in some few cases. Nystagmus is certainly uncommon; if it ever exists in hysteria, its presence suggests rather disseminated sclerosis, or multiple neuritis, or tumour; the pupils act normally in hysteria; there is never optic neuritis nor atrophy.

There may be a whole series of *abnormalities*

* Dr. Gowers, however, considers that a true ankle-clonus is a mark of organic disease.

referable to the internal organs; we will mention the principal of them. In hysterical aphonia, a very common and characteristic complaint, the patient cannot speak above a whisper; but the cough sounds natural, and on application of electricity to the larynx the voice will probably return suddenly. Further, on inspection the larynx is seen to be structurally normal, but there is paresis of the adductors or tensors, allowing a chink to remain between the cords, or the cords themselves to remain lax during attempts at phonation. Hysterical cough is a harsh monotonous bleat or bark, which may be repeated at intervals in the same key for hours together. "Globus" is the name applied to the choking feeling or lump in the throat, from which most hysterical women suffer at times; it may be associated with curious feelings ascending from the lower abdomen, or still more commonly from the epigastrium; or with palpitation of the heart. These often form the preludes to an hysterical fit. Dysphagia may reach such a degree that it can only be distinguished by the passage of a sound from organic disease of the pharynx. Hysterical vomiting generally occurs directly after taking food: the food returns unchanged, there is no epigastric pain, as in gastritis, and despite the apparent rejection of nutriment, the patient contrives to maintain her nutrition. Some hysterical women are troubled by constant eructations or borborygmi; and sometimes flatulent distension of the abdomen, with tenderness, simulating peritonitis, may be witnessed. Retention of urine, and the occasional incontinence of urine, we have noticed: a more rare and curious condition has been described by Charcot—viz., hysterical suppression of urine. In such exceptional cases there is usually vomiting, by which means possibly urea may be excreted. We must be careful to exclude malingering here.

Disturbances of nutrition form no part of hysteria as a rule; we will mention two exceptions. First,

as to general nutrition. There is a state, sometimes called "anorexia nervosa," in which the patient, a young woman who has usually been exposed to some nervous shock or strain, takes a dislike to all food, eats less and less, while she vomits apparently what little she does take. She emaciates so much that organic disease of the stomach or phthisis is feared; and her sunken flushed cheeks may strongly suggest phthisis. But nothing can be found by physical examination; and her recovery under forced feeding, massage, &c., may finally negative such suppositions. Secondly, as to the nutrition of a limb in hysterical paralysis. This is usually normal; but sometimes there is considerable wasting. The characteristics of such "hysterical atrophy" have been thus stated (Babinski): it develops somewhat rapidly, and when the motor power has returned it disappears rapidly; it may involve the whole limb or segments of the limb, but does not pick out particular muscles; there are no fibrillary twitchings; the muscle contracts normally when stimulated by a blow upon its substance; and the electro-contractility is either normal or else quantitatively diminished, but there is never reaction of degeneration.

Treatment.—It is evident not only that the diagnosis between hysterical and organic disease is all-important, both for purposes of prognosis and of treatment; but also that it may often be extremely difficult, and possible only to those who have a minute and accurate knowledge of the symptoms and course of organic nerve-disease. The treatment of an hysterical patient may be no less difficult; for it may require a more intimate knowledge of her circumstances and disposition, and a greater moral authority over her, than the physician has an opportunity of acquiring. For unless she entertain towards him a certain attitude of confidence and submission, things will probably not go well. In most severe cases it is advisable to remove the patient from the surroundings in which the disease

has been bred and fostered, and from friends whose attentions may be injudicious, or who may have lost control over her. She should be placed with some nurse or companion whom she can like and respect, and who must be firm yet kind and pleasant in her dealings with her. Every opportunity must be seized of suggesting* and demonstrating to her the fact that she can and will get well. Employment and interests should be found for her as soon as she is capable of them. And, not least, states of mal-nutrition or of anæmia must be rectified, the bowels and catamenia regulated, and any real bodily disease or source of irritation treated as efficiently and quickly as may be.

The plan of treatment advocated by Weir-Mitchell and Playfair is in accordance with these principles. It is most useful in cases of hysterical paralysis, and in women who by inaction and loss of appetite have become actually weak and emaciated. The patient is removed from home and friends, and placed in isolation under the charge of a suitable nurse. She is confined to bed, and at first her food consists solely of three or four ounces of milk every three hours. Then a course of massage is gradually begun, and with it the milk is increased in amount and solid food given, till within a fortnight or so she is taking three full meals daily, besides one or two quarts of milk and strong soup. The muscles are usually exercised by faradism as well as massage; and as she regains power and nutrition she is encouraged to use her limbs, and gradually to pass from invalidism to active habits. Isolation from her friends appears to be an essential to this treatment, and also the services of a competent nurse, and a masseuse who can properly exercise the muscles. Without proper massage, tissue change fails, and the overfeeding, the object of which is renovation of the tissues, cannot be carried on.

Other modes of treatment are often extremely useful

* We do not enter here into the recently revived question of treatment by "hypnotic" suggestion.

in hysteria, as, for instance, shower-baths and cold douches, static electricity, faradism to the muscles in cases of paralysis or contracture, faradism to the skin by means of the wire-brush in cases of anæsthesia. Bromides may be given for fits, in small doses, and combined either with tincture of digitalis or tincture of valerian. Valerianate of zinc is often useful; and, as general tonics, iron, quinine, and nux vomica. Alcohol, opiates, chloral and similar sedatives, are best avoided. We should not be too anxious to treat every symptom as it arises; it may be well to meet some of them with a judicious disregard. For instance, retention of urine is apt to be perpetuated by the use of the catheter. Hysterical fits may often be cut short by one or other of the following methods:—Cold water, applied either by slapping her face and neck vigorously with a wet towel, or as a profuse and sudden libation to her face and head; or, if this be inadvisable, by pouring a small stream from a height upon her face, and into her mouth if she opens it, her dress and body being protected by a towel or mackintosh; sharp faradism, applied by a wire-brush to the skin of the hand or neck; Hare's method of closing the nostrils and mouth with the hand till semi-strangulation is produced; Charcot's method of so-called ovarian pressure.

Trance.—To the rare conditions of trance and catalepsy we shall only just allude. In trance or lethargy (most frequently seen in hysterical subjects) the patient appears deeply asleep, and cannot be roused; in extreme cases the respirations become very shallow and infrequent, and the pulse and heart-beat very feeble, and the bodily functions are reduced to such a low ebb, as more or less to counterfeit death.

Catalepsy.—Catalepsy is most common in the insane, but may be seen sometimes in the hysterical, and a cataleptic condition of muscles may occur in the course of various organic cerebral diseases. A cataleptic attack generally comes on suddenly and under the

influence of some powerful emotion; the patient appears bereft of consciousness and of the power of spontaneous movement, yet muscular contraction is so far maintained that the limbs and trunk are immobilised in the posture wherein the attack found the patient; and if the limbs be manipulated by a bystander, the postures imprinted on them are similarly maintained. Thus the patient resembles a living statue which can be moulded to any position.

Hypnotism.—Similar conditions can be produced by the processes known as hypnotism or mesmerism, and not only these, but a further condition sometimes called somnambulism. In this the patient's mind and body seem to become capable of action again; but only of such action as is directed or suggested by the person who has hypnotised him. He does exactly what he is told to do, and appears to adopt any idea that is suggested to him. Neither does the influence always cease when he is awakened; commands and suggestions for future use, made to him during the hypnotic state, are sometimes obligatory upon him afterwards (post-hypnotic suggestion). Hypnotism has been largely recommended lately for therapeutical purposes, but it would appear to be a double-edged weapon.

Neurasthenia.—Neurasthenia is a name now frequently applied to a condition which appears to stand midway between hysteria and mental disease. The patient, generally a man, has few if any objective signs of disease, yet is tormented and preoccupied by a series of complaints. In his head he has "sensations," a giddiness, a burning, or still more often a "pressure" on the vertex, pain at the occiput, tenderness of the scalp. He has dimness of vision, spots before the eyes, indeed sometimes the visual field, as examined with the perimeter, is found to be concentrically narrowed. Tinnitus and a certain amount of deafness may be complained of. The speech in some cases is indistinct and drawling.

The spinal region is likewise the seat of aching, weariness, pain, or tenderness. In the limbs and trunk there may be tinglings, pains, districts of anæsthesia, possibly combined with hyper-algesia. The hands may be tremulous, and very commonly the legs become feeble, so that the patient walks in an uncertain straddling way. Palpitation is common; loss of appetite, loss of sexual desire, and above all sleeplessness or bad dreams, are complained of. With all this little is to be made out by physical examination, save general feebleness, anæsthetic patches possibly, slight exaggeration of tendon-reactions; and, according to Oppenheim, concentric narrowing of the visual fields; with optic atrophy and reflex iridoplegia in a very few cases. These last two factors, however, if present, should raise in our minds a very strong suspicion of organic nervous disease, such as spinal degeneration or general paralysis.

The mental condition of these unfortunate people is one of depression, of anxiety and alarms, not only about their symptoms, but often of vaguer kinds. They are irritable, unable to remember properly, and incapable of applying themselves to business. They say they are afraid of committing suicide, or of doing injury to others. In some cases attacks of downright insanity have supervened.

The causes are such as we have had to enumerate for several other neuroses—viz., domestic troubles and anxieties, business failures, over-application to business-work, perhaps sexual excess. The best treatment (which too often cannot be compassed) is relief from all anxieties, complete and prolonged holidays, outdoor life in a bracing air with agreeable companionship. In default of this, tonics and static electricity may do something to relieve symptoms. The prognosis "quoad vitam" is good, but the "restitutio ad integrum" is extremely slow, and often imperfect. The condition may be produced by one cause which deserves special mention—viz., bodily

injury combined with mental shock. The most perfect example is a railway accident. The patient has been stunned, or at most suddenly alarmed, bruised or shaken; then comes a period of anxiety for his own and others' safety, then worry with doctor's examinations and lawyer's cross-examinations. The nervous symptoms developing out of all these conditions have long received the name of "railway spine," but seeing that they may develop after any injury, and that the whole nervous centres (not the spinal cord only) are affected, the term "traumatic neurosis" has lately been proposed. Two cautions must, however, be given: (1) that we do not too hastily assume that such symptoms are a pure neurosis, for their persistency and some recent pathological observations suggest that there may be slight organic changes. (2) That when a lawsuit for compensation is impending, the possibility of malingering should be carefully and impartially weighed.

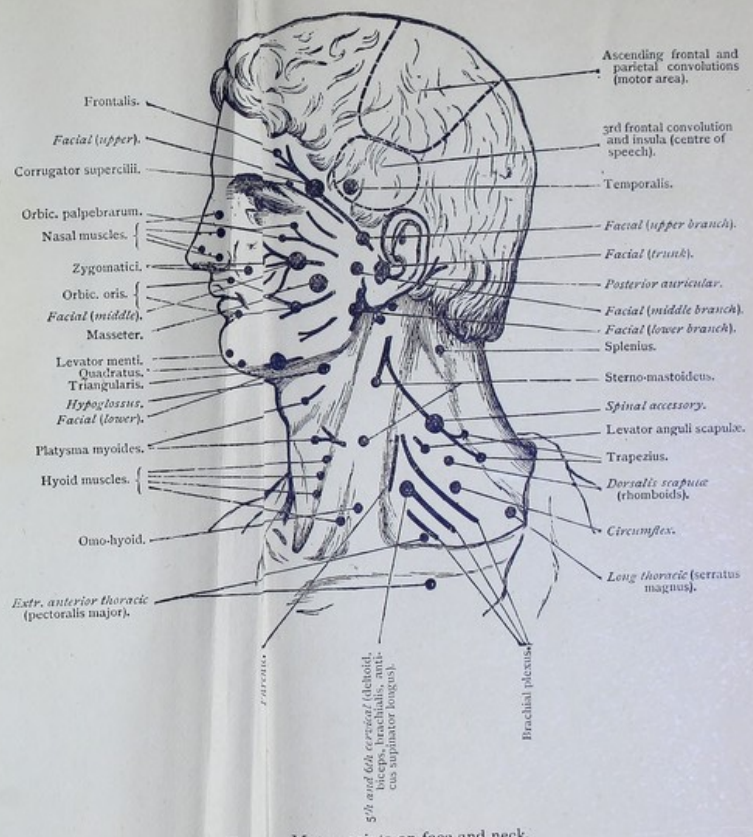


FIG. [illegible].—Motor points on face and neck. After Erb and De Watteville.)

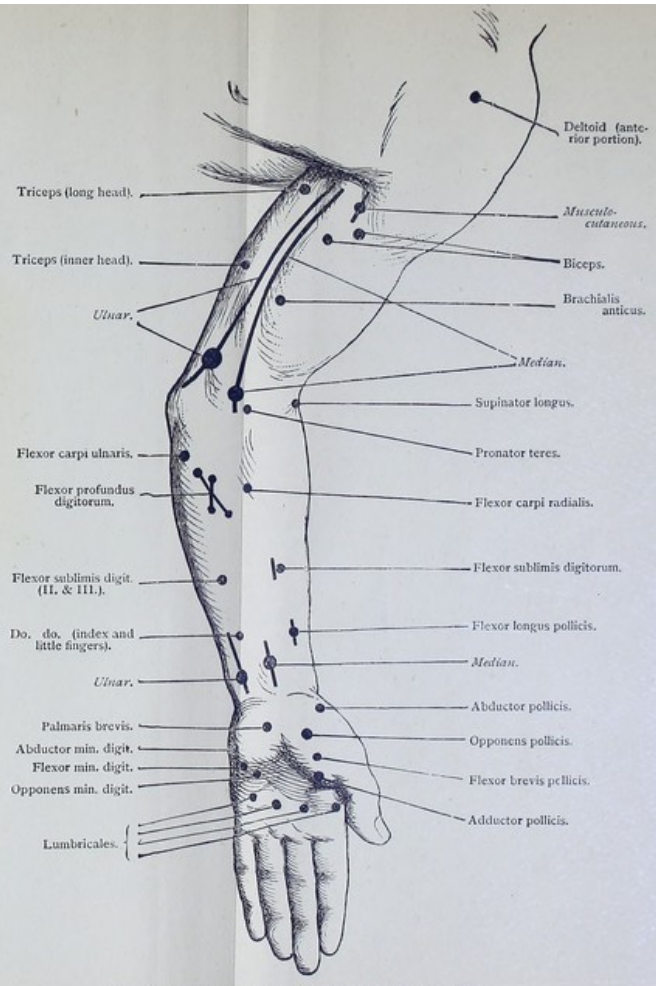


FIG. 62.—Motor points on upper limb, flexor surface.
(After Erb and De Watteville.)

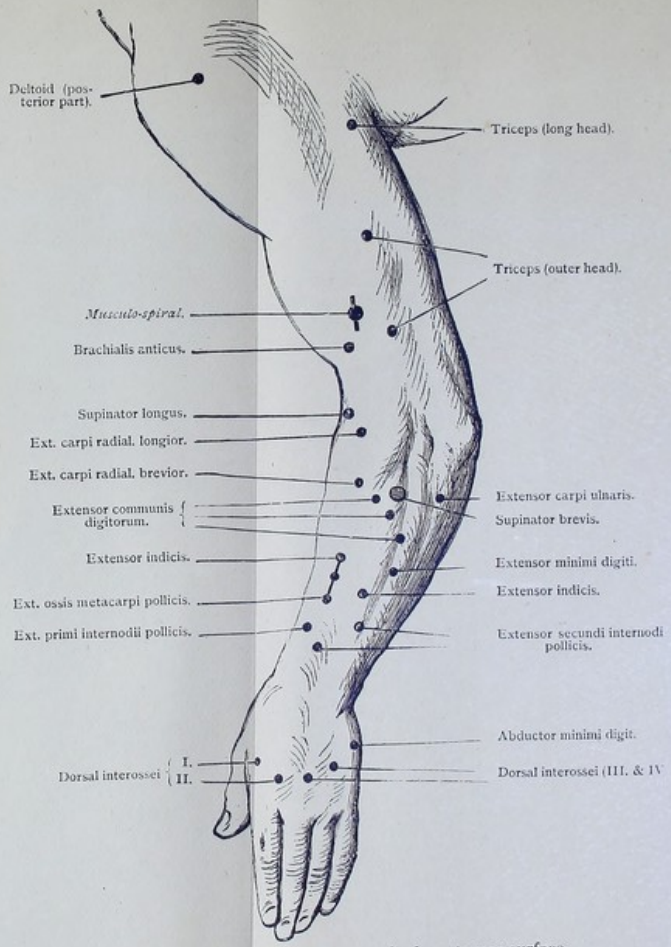


FIG. 63.—Motor points on upper limb, extensor surface.
(After Erb and De Watteville.)

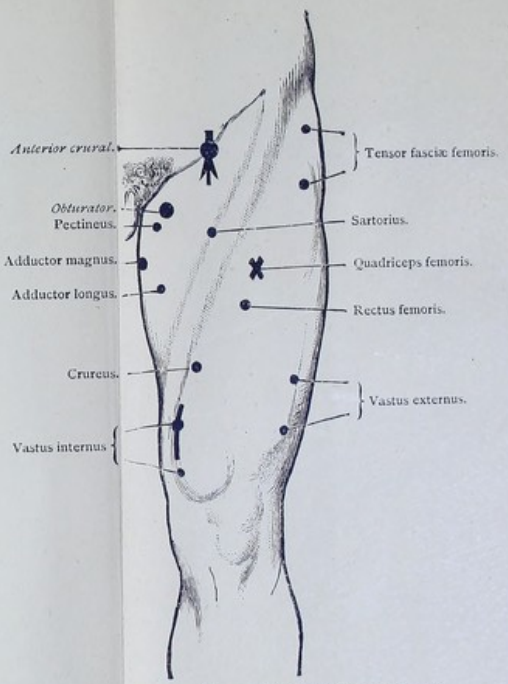


FIG. 64.—Motor points on thigh, anterior surface.
(After Erb and De Watteville.)

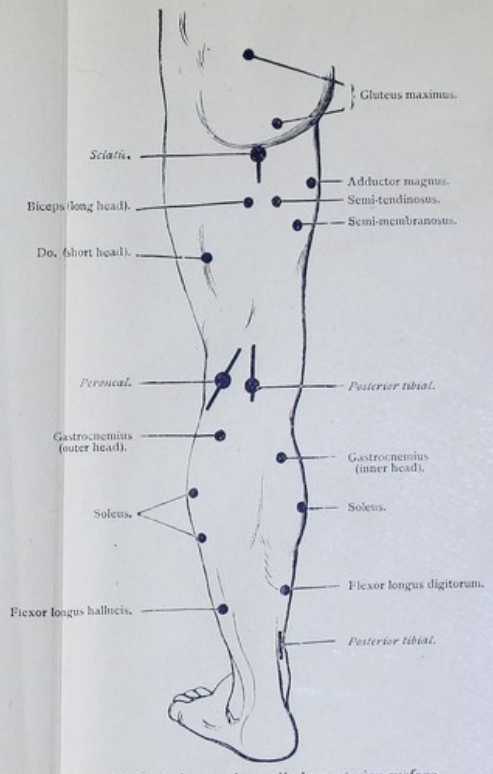


FIG. 65.—Motor points on lower limb, posterior surface.
(After Erb and De Watteville.)

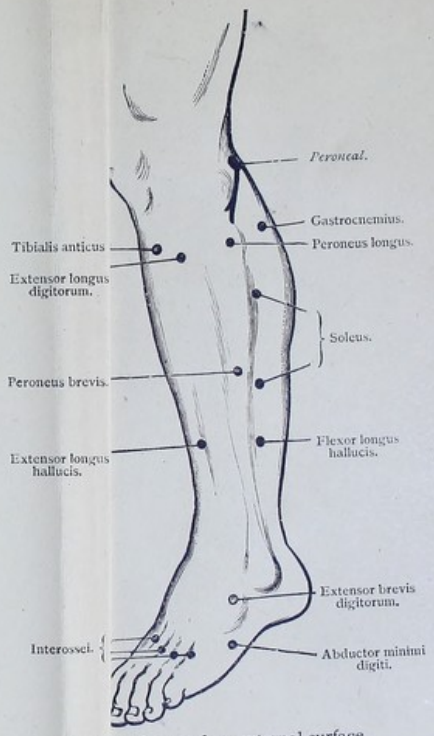


FIG. 6[illegible] points on leg, external surface.
Erb and De Watteville.)

INDEX.

PRINTED BY BALLANTYNE, HANSON AND CO.
LONDON AND EDINBURGH

[CATALOGUE C]

LONDON, *November*, 1891.

J. & A. CHURCHILL'S

MEDICAL CLASS BOOKS.

ANATOMY.

BRAUNE.—An Atlas of Topographical Anatomy, after Plane Sections of Frozen Bodies. By WILHELM BRAUNE, Professor of Anatomy in the University of Leipzig. Translated by EDWARD BELLAMY, F.R.C.S., Surgeon to Charing Cross Hospital, and Lecturer on Surgery in its School. With 34 Photo-lithographic Plates and 46 Woodcuts. Large Imp. 8vo, 40s.

FLOWER.—Diagrams of the Nerves of the Human Body, exhibiting their Origin, Divisions, and Connexions, with their Distribution to the various Regions of the Cutaneous Surface, and to all the Muscles. By WILLIAM H. FLOWER, C.B., F.R.C.S., F.R.S. Third Edition, containing 6 Plates. Royal 4to, 12s.

GODLEE.—An Atlas of Human Anatomy: illustrating most of the ordinary Dissections and many not usually practised by the Student. By RICKMAN J. GODLEE, M.S., F.R.C.S., Surgeon to University College Hospital; Teacher of Operative Surgery, and Assistant Professor of Clinical Surgery in University College; With 48 Imp. 4to Coloured Plates, containing 112 Figures, and a Volume of Explanatory Text, with many Engravings. 8vo, £4 14s. 6d.

HEATH.—Practical Anatomy: a Manual of Dissections. By CHRISTOPHER HEATH, F.R.C.S., Holme Professor of Clinical Surgery in University College and Surgeon to the Hospital. Seventh Edition, revised by RICKMAN J. GODLEE, M.S. Lond., F.R.C.S., Teacher of Operative Surgery, and Assistant Professor of Clinical Surgery in University College, and Surgeon to the Hospital. With 24 Coloured Plates and 278 Engravings. Crown 8vo, 15s.

ANATOMY—*continued.*

HOLDEN.—**A Manual of the Dissection of the** Human Body. By LUTHER HOLDEN, F.R.C.S. Fifth Edition, by JOHN LANGTON, F.R.C.S. and Member of the Court of Examiners; Surgeon to St. Bartholomew's Hospital. With 208 Engravings. 8vo, 20s.

By the same Author.

Human Osteology: comprising a Description of the Bones, with Delineations of the Attachments of the Muscles, the General and Microscopical Structure of Bone and its Development. Seventh Edition, by CHARLES STEWART, Conservator of the Museum, R.C.S., and R. W. REID, M.D., Professor of Anatomy in the University of Aberdeen. With 59 Plates and 75 Wood Engravings. Royal 8vo, 16s.

ALSO,

Landmarks, Medical and Surgical. Fourth Edition. 8vo, 3s. 6d.

MORRIS.—**The Anatomy of the Joints of Man.** By HENRY MORRIS, M.A., F.R.C.S., Surgeon to, and Lecturer on Anatomy and Practical Surgery at, the Middlesex Hospital. With 44 Plates (19 Coloured) and Engravings. 8vo, 16s.

TOOTH.—**An Atlas of the Central Nervous** System and Cranial Nerves, from the larger work of HIRSCHFELD and LÉVEILLÉ. Edited by HOWARD H. TOOTH, M.D., F.R.C.P., Assistant Physician to the National Hospital for the Paralysed and Epileptic. 37 Coloured Plates. Imp. 8vo, 40s.

WAGSTAFFE.—**The Student's Guide to Human** Osteology. By WM. WARWICK WAGSTAFFE, F.R.C.S., late Assistant-Surgeon to, and Lecturer on Anatomy at, St. Thomas's Hospital. With 23 Plates and 66 Engravings. Fcap. 8vo, 10s. 6d.

WILSON — BUCHANAN — CLARK. — **Wilson's** Anatomist's Vade-Mecum: a System of Human Anatomy. Tenth Edition, by GEORGE BUCHANAN, Professor of Clinical Surgery in the University of Glasgow, and HENRY E. CLARK, M.R.C.S., Lecturer on Anatomy in the Glasgow Royal Infirmary School of Medicine. With 450 Engravings, including 26 Coloured Plates. Crown 8vo, 18s.

BOTANY.

BENTLEY AND TRIMEN.—**Medicinal Plants:** being descriptions, with original Figures, of the Principal Plants employed in Medicine, and an account of their Properties and Uses. By ROBERT BENTLEY, F.L.S., and HENRY TRIMEN, M.B., F.R.S., F.L.S. In 4 Vols., large 8vo, with 306 Coloured Plates, bound in half morocco, gilt edges, £11 11s.

BENTLEY.—**A Manual of Botany.** By Robert BENTLEY, F.L.S., M.R.C.S., Emeritus Professor of Botany in King's College and to the Pharmaceutical Society. With nearly 1178 Engravings. Fifth Edition. Crown 8vo, 15s.

By the same Author.

The Student's Guide to Structural, Morphological, and Physiological Botany. With 660 Engravings. Fcap. 8vo, 7s. 6d.

ALSO,

The Student's Guide to Systematic Botany, including the Classification of Plants and Descriptive Botany. With 357 Engravings. Fcap. 8vo, 3s. 6d.

CHEMISTRY.

BERNAYS.—**Notes on Analytical Chemistry** for Students in Medicine. By ALBERT J. BERNAYS, Ph.D., F.C.S., F.I.C., Professor of Chemistry, &c., at St. Thomas's Hospital Medical School. Third Edition. Crown 8vo, 4s. 6d.

BLOXAM.—**Chemistry, Inorganic and Organic;** with Experiments. By CHARLES L. BLOXAM. Seventh Edition, by JOHN MILLAR THOMSON, Professor of Chemistry, King's College, London, and ARTHUR G. BLOXAM, Demonstrator of Chemistry, Royal Agricultural College, Cirencester. With 282 Illustrations. 8vo, 18s.

By the same Author.

Laboratory Teaching; or, Progressive Exercises in Practical Chemistry. Fifth Edition. With 89 Engravings. Crown 8vo, 5s. 6d.

BOWMAN AND BLOXAM.—**Practical Chemistry,** including Analysis. By JOHN E. BOWMAN, and CHARLES L. BLOXAM, late Professor of Chemistry in King's College. Eighth Edition. With 90 Engravings. Fcap. 8vo, 5s. 6d.

CHEMISTRY—*continued.*

CLOWES.—**Practical Chemistry and Qualitative Analysis.** Adapted for Colleges and Schools. By FRANK CLOWES, D.Sc. Lond., Professor of Chemistry at the University College, Nottingham. Fifth Edition. With 57 Engravings and Frontispiece. Post 8vo, 7s. 6d.

CLOWES AND COLEMAN.—**Quantitative Chemical Analysis.** Adapted for Colleges and Schools. By FRANK CLOWES, D.Sc. Lond., Professor of Chemistry in University College, Nottingham, and J. BERNARD COLEMAN, Assoc. R.C.Sci. Dub., Senior Demonstrator of Chemistry in University College, Nottingham. With 83 Engravings. Post 8vo, 7s. 6d.

FOWNES.—**Manual of Chemistry.**—*See WATTS.*

FRANKLAND AND JAPP.—**Inorganic Chemistry.** By EDWARD FRANKLAND, Ph.D., D.C.L., F.R.S., and F. R. JAPP, M.A., Ph.D., F.I.C., Professor of Chemistry in the University of Aberdeen. With Lithographic Plates and Wood Engravings. 8vo, 24s.

MORLEY.—**Outlines of Organic Chemistry.** By H. FORSTER MORLEY, M.A., D.Sc., Joint Editor of "Watts' Dictionary of Chemistry." Crown 8vo, 7s. 6d.

RAMSAY.—**Elementary Systematic Chemistry.** For Use of Schools and Colleges. By WILLIAM RAMSAY, Ph.D., F.R.S., Professor of Chemistry in University College, London. With Engravings, 4s. 6d. (or Interleaved, 5s. 6d.).

By the same Author.

A System of Inorganic Chemistry. With Engravings. 8vo, 15s.

VALENTIN.—**Chemical Tables for the Lecture-room and Laboratory.** By WILLIAM G. VALENTIN, F.C.S. In Five large Sheets, 5s. 6d.

VALENTIN AND HODGKINSON.—**A Course of Qualitative Chemical Analysis.** By the late W. G. VALENTIN, F.C.S. Seventh Edition, by Dr. W. R. HODGKINSON, F.R.S.E., Professor of Chemistry and Physics in the Royal Artillery College, and Royal Military Academy, Woolwich; assisted by H. CHAPMAN-JONES, F.C.S., Demonstrator in the Royal School of Mines, and F. E. MATTHEWS, Ph.D., of Cooper's Hill College. With Engravings and Map of Spectra. 8vo, 8s. 6d. [The Tables separately, 2s. 6d.]

CHEMISTRY—*continued.*

WATTS.—**Manual of Chemistry, Theoretical and Practical.** (Based on Fownes' Manual.) By HENRY WATTS, B.A., F.R.S.

VOL. I.—**Physical and Inorganic Chemistry.** Second Edition (Fourteenth of Fownes'). By WILLIAM A. TILDEN, D.Sc., F.R.S., Professor of Chemistry in the Mason College, Birmingham. With 122 Wood Engravings, and Coloured Plate of Spectra. Crown 8vo, 8s. 6d.

VOL. II.—**Chemistry of Carbon-Compounds,** or Organic Chemistry. Second Edition (Thirteenth of Fownes'). Edited by WM. A. TILDEN, D.Sc., F.R.S. With Engravings. Crown 8vo, 10s.

CHILDREN, DISEASES OF.

DAY.—**A Manual of the Diseases of Children.** By WILLIAM H. DAY, M.D., Physician to the Samaritan Hospital for Women and Children. Second Edition. Crown 8vo, 12s. 6d.

ELLIS.—**A Practical Manual of the Diseases** of Children. By EDWARD ELLIS, M.D., late Senior Physician to the Victoria Hospital for Sick Children. With a Formulary. Fifth Edition. Crown 8vo, 10s.

GOODHART.—**The Student's Guide to Diseases** of Children. By JAMES FREDERIC GOODHART, M.D., F.R.C.P., Physician to Guy's Hospital and Lecturer on Pathology in its Medical School. Fourth Edition. Fcap. 8vo, 10s. 6d.

SMITH.—**A Practical Treatise on Disease in** Children. By EUSTACE SMITH, M.D., F.R.C.P., Physician to H.M. the King of the Belgians, and to the East London Hospital for Children. Second Edition. 8vo, 22s.

By the same Author.

Clinical Studies of Disease in Children. Second Edition. Post 8vo, 7s. 6d.

Also,

On the Wasting Diseases of Infants and Children. Fifth Edition. Post 8vo, 8s. 6d.

STEINER.—**Compendium of Children's Dis-**eases; a Handbook for Practitioners and Students. By JOHANN STEINER, M.D. Translated by LAWSON TAIT, F.R.C.S., Surgeon to the Birmingham Hospital for Women, &c. 8vo, 12s. 6d.

DENTISTRY.

GORGAS. — **Dental Medicine: a Manual of** Dental Materia Medica and Therapeutics. By FERDINAND J. S. GORGAS, A.M., M.D., D.D.S., Professor of Dental Surgery and Science, &c., in the University of Maryland. Third Edition. 8vo, 16s.

HARRIS. — **The Principles and Practice of** Dentistry; including Anatomy, Physiology, Pathology, Therapeutics, Dental Surgery, and Mechanism. By CHAPIN A. HARRIS, M.D., D.D.S. Twelfth Edition, revised and edited by FERDINAND J. S. GORGAS, A.M., M.D., D.D.S. With over 1,000 Illustrations. 8vo, 33s.

STOCKEN.—**Elements of Dental Materia Medica** and Therapeutics, with Pharmacopœia. By JAMES STOCKEN, L.D.S.R.C.S., late Lecturer on Dental Materia Medica and Therapeutics and Dental Surgeon to the National Dental Hospital; assisted by THOMAS GADDES, L.D.S. Eng. and Edin. Third Edition. Fcap. 8vo, 7s. 6d.

TOMES (C. S.).—**Manual of Dental Anatomy,** Human and Comparative. By CHARLES S. TOMES, M.A., F.R.S. Third Edition. With 212 Engravings. Crown 8vo, 12s. 6d.

TOMES (J. and C. S.).—**A System of Dental** Surgery. By Sir JOHN TOMES, F.R.S., and CHARLES S. TOMES, M.A., M.R.C.S., F.R.S. Third Edition. With 292 Engravings. Crown 8vo, 15s.

EAR, DISEASES OF.

BURNETT.—**The Ear: its Anatomy, Physio-** logy, and Diseases. A Practical Treatise for the Use of Medical Students and Practitioners. By CHARLES H. BURNETT, M.D., Aural Surgeon to the Presbyterian Hospital, Philadelphia. Second Edition. With 107 Engravings. 8vo, 18s.

DALBY.—**On Diseases and Injuries of the Ear.** By Sir WILLIAM B. DALBY, F.R.C.S., Aural Surgeon to, and Lecturer on Aural Surgery at, St. George's Hospital. Third Edition. With Engravings. Crown 8vo. 7s. 6d.

EAR, DISEASES OF—*continued.*

JONES.—**Practitioner's Handbook of Diseases** of the Ear and Naso-Pharynx. By H. MACNAUGHTON JONES, M.D., M.Ch.; Examiner, and late Professor in the Queen's University; and Surgeon to the Cork Ophthalmic and Aural Hospital. Third Edition of "Aural Surgery." With 128 Engravings, and 2 Coloured Plates (16 Figures). Royal 8vo, 6s.

FORENSIC MEDICINE.

ABERCROMBIE. — **The Student's Guide to** Medical Jurisprudence. By JOHN ABERCROMBIE, M.D., F.R.C.P., Physician to Charing Cross Hospital. Fcap. 8vo, 7s. 6d.

OGSTON.—**Lectures on Medical Jurisprudence.** By FRANCIS OGSTON, M.D., late Professor of Medical Jurisprudence and Medical Logic in the University of Aberdeen. Edited by FRANCIS OGSTON, Jun., M.D., late Lecturer on Practical Toxicology in the University of Aberdeen. With 12 Plates. 8vo, 18s.

TAYLOR.—**The Principles and Practice of** Medical Jurisprudence. By ALFRED S. TAYLOR, M.D., F.R.S. Third Edition, revised by THOMAS STEVENSON, M.D., F.R.C.P., Lecturer on Chemistry and Medical Jurisprudence at Guy's Hospital; Examiner in Chemistry at the Royal College of Physicians; Official Analyst to the Home Office. With 188 Engravings. 2 Vols. 8vo, 31s. 6d.

By the same Author.

A Manual of Medical Jurisprudence. Twelfth Edition, revised by THOMAS STEVENSON, M.D., F.R.C.P. With 56 Engravings. Crown 8vo, 14s.

ALSO,

On Poisons, in relation to Medical Juris- prudence and Medicine. Third Edition. With 104 Engravings. Crown 8vo, 16s.

TIDY AND WOODMAN.—**A Handy-Book of** Forensic Medicine and Toxicology. By C. MEYMOTT TIDY, M.B.; and W. BATHURST WOODMAN, M.D., F.R.C.P. With 8 Lithographic Plates and 116 Wood Engravings. 8vo, 31s. 6d.

HYGIENE.

PARKES.—A Manual of Practical Hygiene. By the late EDMUND A. PARKES, M.D., F.R.S. Eighth Edition, by J. LANE NOTTER, M.D., Professor of Military Hygiene in the Army Medical School. With 10 Plates and 103 Wood Engravings. 8vo, 18s.

WILSON.—A Handbook of Hygiene and Sanitary Science. By GEORGE WILSON, M.A., M.D., F.R.S.E., Medical Officer of Health for Mid Warwickshire. Seventh Edition. With Engravings. Crown 8vo. [*In the press.*

MATERIA MEDICA AND THERAPEUTICS.

LESCHER.—Recent Materia Medica. Notes on their Origin and Therapeutics. By F. HARWOOD LESCHER, F.C.S., Pereira Medallist. Fourth Edition. 8vo, 2s. 6d.

OWEN.—A Manual of Materia Medica; incorporating the Author's "Tables of Materia Medica." By ISAMBARD OWEN, M.D., F.R.C.P., Lecturer on Materia Medica and Therapeutics to St. George's Hospital. Second Edition. Crown 8vo, 6s. 6d.

ROYLE AND HARLEY.—A Manual of Materia Medica and Therapeutics. By J. FORBES ROYLE, M.D., F.R.S., and JOHN HARLEY, M.D., F.R.C.P., Physician to, and Joint Lecturer on Clinical Medicine at, St. Thomas's Hospital. Sixth Edition, including addition and alterations in the B.P. 1885. With 139 Engravings. Crown 8vo, 15s.

SOUTHALL.—The Organic Materia Medica of the British Pharmacopœia, Systematically Arranged. By W. SOUTHALL, F.L.S. Fourth Edition. Crown 8vo, 5s.

THOROWGOOD.—The Student's Guide to Materia Medica and Therapeutics. By JOHN C. THOROWGOOD, M.D., F.R.C.P. Second Edition. With Engravings. Fcap. 8vo, 7s.

WARING.—A Manual of Practical Therapeutics. By EDWARD J. WARING, C.I.E., M.D., F.R.C.P. Fourth Edition, revised by the Author and DUDLEY W. BUXTON, M.D., M.R.C.P. Crown 8vo, 14s.

MEDICINE.

CHARTERIS.—**The Student's Guide to the Practice of Medicine.** By M. CHARTERIS, M.D., Professor of Therapeutics and Materia Medica, University of Glasgow. With Engravings on Copper and Wood. Sixth Edition. Fcap. 8vo, 9s.

FAGGE.—**The Principles and Practice of Medicine.** By the late C. HILTON FAGGE, M.D., F.R.C.P., Edited by PHILIP H. PYE-SMITH, M.D., F.R.S., F.R.C.P., Physician to, and Lecturer on Medicine in, Guy's Hospital. Third Edition. 2 Vols. 8vo. Cloth, 40s., leather, 46s.

FENWICK.—**The Student's Guide to Medical Diagnosis.** By SAMUEL FENWICK, M.D., F.R.C.P., Physician to the London Hospital. Seventh Edition. With 117 Engravings. Fcap. 8vo, 7s.

By the same Author.

Outlines of Medical Treatment, including Foreign as well as English Methods. Third Edition. Crown 8vo, 10s.

FOWLER.—**A Dictionary of Practical Medicine.** By Various Writers. Edited by JAMES KINGSTON FOWLER, M.A., M.D., F.R.C.P., Senior Assistant Physician to, and Lecturer on Pathological Anatomy at, Middlesex Hospital. 8vo., cloth, 21s.; half-calf, 25s.

HARRIS.—**The Student's Guide to Diseases of the Chest.** By VINCENT D. HARRIS, M.D., F.R.C.P., Physician to the Victoria Park Hospital for Diseases of the Chest. With 55 Engravings, plain and Coloured. Fcap. 8vo, 7s. 6d.

NIXON.—**Handbook of Hospital Practice and Physical Diagnosis.** By CHRISTOPHER J. NIXON, M.D., LL.D. Professor of Medicine in the Catholic University, Dublin; Examiner in Medicine, K.Q.C.P.I., and for the Conjoint Examinations of K.Q.C.P. and S. Irel. With Plates and Engravings. 8vo, 9s.

TAYLOR.—**A Manual of the Practice of Medicine.** By FREDERICK TAYLOR, M.D., F.R.C.P., Physician to, and Lecturer on Medicine at, Guy's Hospital. Second Edition. With 24 Illustrations. Crown 8vo, 15s.

WEST. — **How to Examine the Chest: a Practical Guide for the Use of Students.** By SAMUEL WEST, M.D., F.R.C.P., Assistant Physician and Medical Tutor to St. Bartholomew's Hospital. Second Edition. With 46 Engravings. Fcap. 8vo, 5s.

MIDWIFERY.

BARNES.—**Lectures on Obstetric Operations,** including the Treatment of Hæmorrhage, and forming a Guide to the Management of Difficult Labour. By ROBERT BARNES, M.D., F.R.C.P., Consulting Obstetric Physician to St. George's Hospital. Fourth Edition. With 121 Engravings. 8vo, 12s. 6d.

BURTON.—**Handbook of Midwifery for Mid-wives.** By JOHN E. BURTON, M.R.C.S., L.R.C.P., Surgeon to the Liverpool Hospital for Women. Second Edition. With Engravings. Fcap 8vo, 6s.

GALABIN.—**A Manual of Midwifery. By Alfred** LEWIS GALABIN, M.A., M.D., F.R.C.P., Obstetric Physician and Lecturer on Midwifery, &c., to Guy's Hospital, Examiner in Midwifery to the Conjoint Examining Board for England. Second Edition. With 249 Engravings. Crown 8vo, 15s.

REYNOLDS.—**Notes on Midwifery: specially** designed to assist the Student in preparing for Examination. By J. J. REYNOLDS, L.R.C.P., M.R.C.S. Second Edition. With 15 Engravings. Fcap. 8vo, 4s.

ROBERTS.—**The Student's Guide to the Practice** of Midwifery. By D. LLOYD ROBERTS, M.D., F.R.C.P., Lecturer on Clinical Midwifery and Diseases of Women at Owen's College, Physician to St. Mary's Hospital, Manchester. Third Edition. With 2 Coloured Plates and 127 Engravings. Fcap. 8vo, 7s. 6d.

SWAYNE.—**Obstetric Aphorisms for the Use of** Students commencing Midwifery Practice. By JOSEPH G. SWAYNE, M.D., Lecturer on Obstetric Medicine at the Bristol Medical School Ninth Edition. With 17 Engravings. Fcap. 8vo, 3s. 6d.

MICROSCOPY.

CARPENTER.—**The Microscope and its Revela-tions.** By WILLIAM B. CARPENTER, C.B., M.D., F.R.S. Seventh Edition. Edited by Rev. Dr. DALLINGER, F.R.S. With 21 Plates and 800 Woodcuts. 8vo, 26s.

LEE.—**The Microtomist's Vade-Mecum; a** Handbook of the Methods of Microscopic Anatomy. By ARTHUR BOLLES LEE, Assistant in the Russian Laboratory of Zoology at Villefranche-sur-Mer (Nice). Second Edition. 8vo, 12s. 6d.

OPHTHALMOLOGY.

GOWERS.—**A Manual and Atlas of Medical** Ophthalmoscopy. By W. R. GOWERS, M.D., F.R.S., F.R.C.P., Physician to the National Hospital for the Paralysed and Epileptic. Third Edition. Edited with the assistance of MARCUS GUNN, M.B., F.R.C.S., Surgeon to the Royal London Ophthalmic Hospital; Ophthalmic Surgeon to the National Hospital for the Paralysed and Epileptic. With 12 Autotype Plates and 83 Engravings. 8vo, 16s.

HARTRIDGE.—**The Refraction of the Eye. By** GUSTAVUS HARTRIDGE, F.R.C.S., Surgeon to the Royal Westminster Ophthalmic Hospital. Fifth Edition. With 98 Illustrations, Test Types, &c. Crown 8vo, 6s.

By the same Author.

The Ophthalmoscope: A Manual for Stu- dents. With 2 Coloured Plates and 63 Engravings. Post 8vo 4s.

HIGGENS.—**Hints on Ophthalmic Out-Patient** Practice. By CHARLES HIGGENS, F.R.C.S., Ophthalmic Surgeon to, and Lecturer on Ophthalmology at, Guy's Hospital. Third Edition Fcap. 8vo, 3s.

MACNAMARA.—**Diseases and Refraction of the** Eye. By CHARLES MACNAMARA, F.R.C.S., and GUSTAVUS HARTRIDGE, F.R.C.S., Surgeons to the Royal Westminster Ophthalmic Hospital. Fifth Edition. With 156 Engravings. Crown 8vo, 10s. 6d.

NETTLESHIP.—**The Student's Guide to Diseases** of the Eye. By EDWARD NETTLESHIP, F.R.C.S., Ophthalmic Surgeon to St. Thomas's Hospital, Surgeon to the Royal London Ophthalmic Hospital. Fifth Edition. With 164 Engravings, and a Coloured Plate illustrating Colour-blindness. Fcap. 8vo, 7s. 6d.

POLLOCK.—**The Normal and Pathological** Histology of the Human Eye and Eyelids. By C. FRED. POLLOCK, M.D., F.R.C.S.E., and F.R.S.E., Surgeon for Diseases of the Eye, Anderson's College Dispensary, Glasgow. With 100 Plates, containing 230 Original Drawings by the Author, Lithographed in black and colours. Crown 8vo, 15s.

WOLFE.—**On Diseases and Injuries of the Eye:** a Course of Systematic and Clinical Lectures to Students and Medical Practitioners. By J. R. WOLFE, M.D., F.R.C.S.E., Senior Surgeon to the Glasgow Ophthalmic Institution, Lecturer on Ophthalmic Medicine and Surgery in Anderson's College. With 10 Coloured Plates, and 120 Wood Engravings, 8vo, 21s.

PATHOLOGY.

BOWLBY.—**The Student's Guide to Surgical** Pathology and Morbid Anatomy. By ANTHONY A. BOWLBY, F.R.C.S., Surgical Registrar and Demonstrator of Practical Surgery and Surgical Pathology at St. Bartholomew's Hospital. Second Edition. With 158 Engravings. Fcap. 8vo, 9s.

JONES AND SIEVEKING.—**A Manual of Pathological Anatomy.** By C. HANDFIELD JONES, M.B., F.R.S., and SIR EDWARD H. SIEVEKING, M.D., F.R.C.P. Second Edition. Edited, with considerable enlargement, by J. F. PAYNE, M.B., Physician to, and Lecturer on Pathological Anatomy at, St. Thomas's Hospital. With 195 Engravings. Crown 8vo, 16s.

LANCEREAUX.—**Atlas of Pathological Anatomy.** By Dr. LANCEREAUX. Translated by W. S. GREENFIELD, M.D., Professor of Pathology in the University of Edinburgh. With 70 Coloured Plates. Imperial 8vo, £5 5s.

MOORE.—**Pathological Anatomy of Diseases,** arranged according to the Nomenclature of Diseases of the R.C.P. Lond. By NORMAN MOORE, M.D., Assistant Physician and Lecturer on Pathological Anatomy to St. Bartholomew's Hospital. With 110 Illustrations. Fcap. 8vo, 8s. 6d.

SUTTON. — **An Introduction to General Pathology.** By JOHN BLAND SUTTON, F.R.C.S., Sir E. WILSON, Lecturer on Pathology, R.C.S.; Assistant Surgeon to, and Lecturer on Anatomy at, Middlesex Hospital. With 149 Engravings. 8vo, 14s.

WYNTER AND WETHERED.—**A Manual of Clinical and Practical Pathology.** By W. ESSEX WYNTER, M.D., Medical Registrar and late Demonstrator of Anatomy and Chemistry at the Middlesex Hospital; and FRANK J. WETHERED, M.D., Assistant Physician to the City of London Hospital for Diseases of the Chest. With 4 Coloured Plates and 67 other Illustrations, 8vo, 12s. 6d.

PHYSIOLOGY.

CARPENTER.—Principles of Human Physiology. By WILLIAM B. CARPENTER, C.B., M.D., F.R.S. Ninth Edition. Edited by Henry Power, M.B., F.R.C.S. With 3 Steel Plates and 377 Wood Engravings. 8vo, 31s. 6d.

DALTON.—A Treatise on Human Physiology: designed for the use of Students and Practitioners of Medicine. By JOHN C. DALTON, M.D., Professor of Physiology and Hygiene in the College of Physicians and Surgeons, New York. Seventh Edition. With 252 Engravings. Royal 8vo, 20s.

FREY.—The Histology and Histo-Chemistry of Man. A Treatise on the Elements of Composition and Structure of the Human Body. By HEINRICH FREY, Professor of Medicine in Zurich. Translated by ARTHUR E. BARKER, Surgeon to the University College Hospital. With 608 Engravings. 8vo, 21s.

SANDERSON.—Handbook for the Physiological Laboratory: containing an Exposition of the fundamental facts of the Science, with explicit Directions for their demonstration. By J. BURDON SANDERSON, M.D., F.R.S.; E. KLEIN, M.D., F.R.S.; MICHAEL FOSTER, M.D., F.R.S., and T. LAUDER BRUNTON, M.D., F.R.S. 2 Vols., with 123 Plates. 8vo, 24s.

SHORE.—Elementary Practical Biology. Vegetable. By THOMAS W. SHORE, M.D., B.Sc. Lond., Lecturer on Comparative Anatomy at St. Bartholomew's Hospital. 8vo, 6s.

YEO.—A Manual of Physiology for the Use of Junior Students of Medicine. By GERALD F. YEO, M.D., F.R.C.S., F.R.S., late Professor of Physiology in King's College, London. Second Edition. With 318 Engravings (many figures). Crown 8vo, 14s.

PSYCHOLOGY.

BUCKNILL AND TUKE.—A Manual of Psychological Medicine: containing the Lunacy Laws, Nosology, Ætiology, Statistics, Description, Diagnosis, Pathology, and Treatment of Insanity, with an Appendix of Cases. By JOHN C. BUCKNILL, M.D. F.R.S., and D. HACK TUKE, M.D., F.R.C.P. Fourth Edition, with 12 Plates (30 Figures). 8vo, 25s.

CLOUSTON. — Clinical Lectures on Mental Diseases. By THOMAS S. CLOUSTON, M.D., and F.R.C.P. Edin.; Lecturer on Mental Diseases in the University of Edinburgh. Second Edition. With 8 Plates (6 Coloured). Crown 8vo, 12s. 6d.

SURGERY.

BELLAMY.—**The Student's Guide to Surgical Anatomy**; an Introduction to Operative Surgery. By EDWARD BELLAMY, F.R.C.S., Surgeon to, and Lecturer on Surgery at, Charing Cross Hospital. Third Edition. With 80 Engravings. Fcap. 8vo, 7s. 6d.

BRYANT.—**A Manual for the Practice of Surgery.** By THOMAS BRYANT, F.R.C.S., Consulting Surgeon to Guy's Hospital. Fourth Edition. With 750 Illustrations (many being coloured), and including 6 Chromo-Lithographic Plates. 2 Vols. Crown 8vo, 32s.

DRUITT AND BOYD.—**Druitt's Surgeon's Vade-Mecum**; a Manual of Modern Surgery. Edited by STANLEY BOYD, M.B., B.S. Lond., F.R.C.S., Assistant Surgeon and Pathologist to the Charing Cross Hospital. Twelfth Edition. With 373 Engravings. Crown 8vo, 16s.

HEATH.—**A Manual of Minor Surgery and Bandaging.** By CHRISTOPHER HEATH, F.R.C.S., Holme Professor of Clinical Surgery in University College and Surgeon to the Hospital. Ninth Edition. With 146 Engravings. Fcap. 8vo, 6s.

By the same Author.

A Course of Operative Surgery: with Twenty Plates (containing many figures) drawn from Nature by M. LÉVEILLÉ, and Coloured. Second Edition. Large 8vo, 30s

ALSO,

The Student's Guide to Surgical Diagnosis. Second Edition. Fcap. 8vo, 6s. 6d.

JACOBSON.—**The Operations of Surgery: intended especially for the use of those recently appointed on a Hospital Staff, and for those preparing for the Higher Examinations.** By W. H. A. JACOBSON, M.A., M.B., M.Ch. Oxon., F.R.C.S., Assistant Surgeon to Guy's Hospital, and Lecturer on Anatomy in the Medical School. Second Edition. With 235 Engravings. 8vo, 30s.

SURGERY—*continued.*

MOULLIN.—Surgery. By C. W. Mansell Moullin, M.A., M.D.Oxon., F.R.C.S., Surgeon to, and Lecturer on Physiology at, the London Hospital; formerly Radcliffe Travelling Fellow and Fellow of Pembroke College, Oxford. With 497 Illustrations. 8vo, 34s.

WALSHAM.—Surgery: its Theory and Practice (Student's Guide Series). By WILLIAM J. WALSHAM, F.R.C.S., Assistant Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital. Third Edition. With 318 Engravings. Fcap. 8vo, 10s. 6d.

TERMINOLOGY.

MAXWELL.—Terminologia Medica Polyglotta: a Concise International Dictionary of Medical Terms (French, Latin, English, German, Italian, Spanish, and Russian). By THEODORE MAXWELL, M.D., B.Sc. Lond., F.R.C.S Edin. Roy. 8vo, 16s.

MAYNE.—A Medical Vocabulary: being an Explanation of all Terms and Phrases used in the various Departments of Medical Science and Practice, giving their Derivation, Meaning, Application, and Pronunciation. By R. G. MAYNE, M.D., LL.D. Sixth Edition, by W. W. WAGSTAFFE, B.A., F.R.C.S. Crown 8vo, 10s. 6d.

A Short Dictionary of Medical Terms, 2s. 6d.

TREVES AND LANG.—A German-English Dictionary of Medical Terms. By FREDERICK TREVES, F.R.C.S., Surgeon to the London Hospital, and HUGO LANG, B.A. Half-bound in calf, 12s.

WOMEN, DISEASES OF.

BARNES.—A Clinical History of the Medical and Surgical Diseases of Women. By ROBERT BARNES, M.D., F.R.C.P., Obstetric Physician to, and Lecturer on Diseases of Women, &c., at, St. George's Hospital. Second Edition. With 181 Engravings. 8vo, 28s.

DUNCAN.—Clinical Lectures on the Diseases of Women. By J. MATTHEWS DUNCAN, A.M., M.D., LL.D., F.R.C.P., F.R.S., late Physician Accoucheur to, and Lecturer on Midwifery at, St. Bartholomew's Hospital. Fourth Edition. 8vo, 16s.

WOMEN, DISEASES OF—*continued.*

GALABIN.—**The Student's Guide to the Diseases of Women.** By ALFRED L. GALABIN, M.D., F.R.C.P., Obstetric Physician to Guy's Hospital, Examiner in Obstetric Medicine to the University of Cambridge, and to the R. C. P. Lond. Fourth Edition. With 94 Engravings. Fcap. 8vo, 7s. 6d.

REYNOLDS.—**Notes on Diseases of Women.** Specially designed to assist the Student in preparing for Examination. By J. J. REYNOLDS, L.R.C.P., M.R.C.S. Third Edition. Fcap. 8vo, 2s. 6d.

SAVAGE.—**The Surgery of the Female Pelvic Organs.** By HENRY SAVAGE, M.D., Lond., F.R.C.S., one of the Consulting Medical Officers of the Samaritan Hospital for Women. Fifth Edition. With 17 Lithographic Plates and 52 Woodcuts. Royal 4to, Coloured Plates, 35s.; Uncoloured, 15s.

WEST AND DUNCAN.—**Lectures on the Diseases of Women.** By CHARLES WEST, M.D., F.R.C.P. Fourth Edition. Revised and in part re-written by the Author, with numerous additions by J. MATTHEWS DUNCAN, M.D., F.R.C.P., F.R.S., late Obstetric Physician to St. Bartholomew's Hospital. 8vo, 16s.

ZOOLOGY.

CHAUVEAU AND FLEMING.—**The Comparative Anatomy of the Domesticated Animals.** By A. CHAUVEAU, Professor at the Lyons Veterinary School; and GEORGE FLEMING, C.B., late Principal Veterinary Surgeon of the Army. Second Edition. With 585 Engravings. 31s. 6d.

HUXLEY.—**Manual of the Anatomy of Invertebrated Animals.** By THOMAS H. HUXLEY, LL.D., F.R.S. With 156 Engravings. Post 8vo, 16s.

By the same Author.

Manual of the Anatomy of Vertebrated Animals. With 110 Engravings. Post 8vo, 12s.

WILSON.—**The Student's Guide to Zoology:** a Manual of the Principles of Zoological Science. By ANDREW WILSON, Lecturer on Natural History, Edinburgh. With Engravings. Fcap. 8vo, 6s. 6d.