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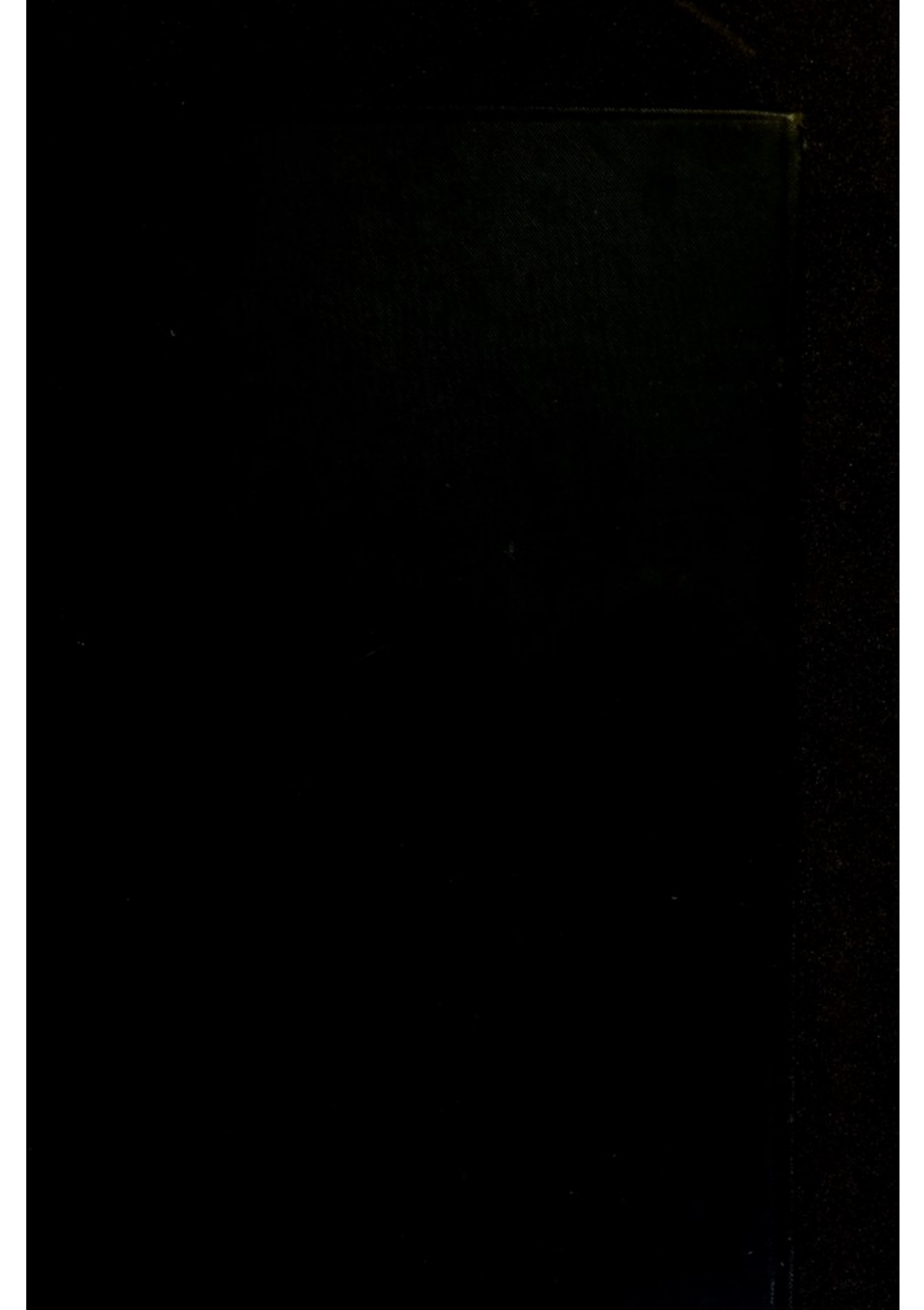
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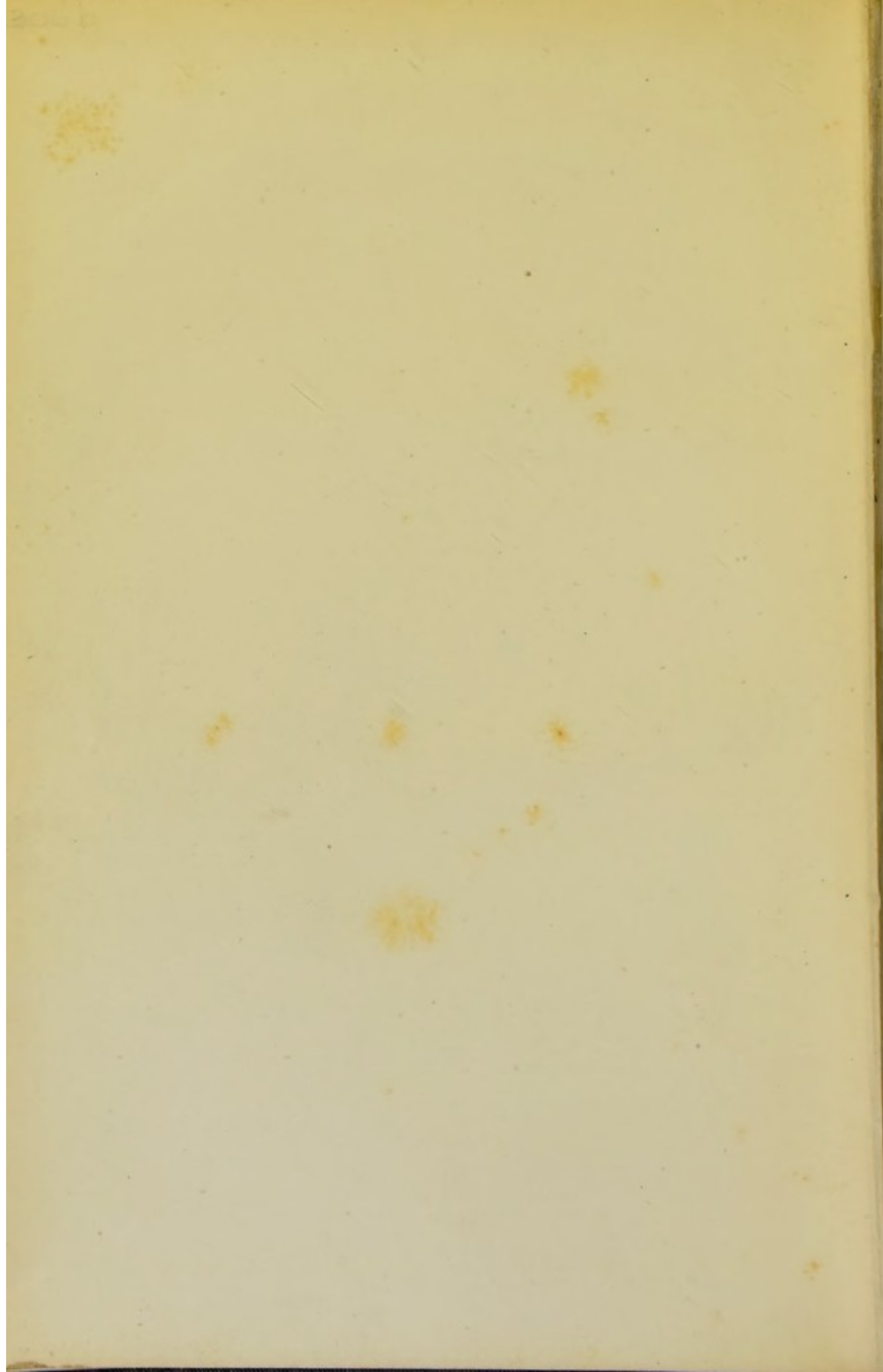
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ON THE DIAGNOSIS OF DISEASES
OF THE BRAIN, SPINAL CORD, AND NERVES.

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ON THE DIAGNOSIS OF
DISEASES OF THE BRAIN,
SPINAL CORD, AND NERVES.

BY

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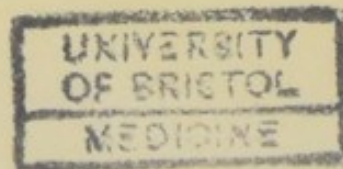
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P R E F A C E .

By the courtesy of the Council of Queen's College I have recently been privileged to deliver a Course of Post-graduate Lectures at that Institution on the "DIAGNOSIS OF DISEASES OF THE NERVOUS SYSTEM." Many gentlemen who attended my lectures have expressed a hope that I would publish them. From conversations with numerous professional friends I am led to believe that busy practitioners still feel the want of a small and plainly written work on the subject. This little volume, then, is chiefly intended for them and for students. I hope, too, that it may serve as an introduction to the more valuable and standard works of Ross, Gowers, Bramwell, Buzzard, and others. I must express the great obligation under which I lie to my friend Mr. Jordan Lloyd, for his kindness in reading over and correcting the proof sheets.

108 NEWHALL STREET,
BIRMINGHAM.

February 1887.



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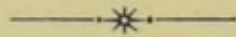
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CHAPTER I.

THE CEREBRAL CIRCULATION.



FOR much of our knowledge of the blood-supply of the different portions of the encephalon we are indebted to Charcot and Duret.

The Cerebral Arteries.

The brain is supplied by four large arteries, the two internal carotid and the two vertebral arteries, which together form an anastomosis at the base of the cerebrum called the circle of Willis.

The branches of these arteries form two systems :—

- I. The cortical system, comprising the nutrient arteries to the cortex and underlying white matter and
- II. The central or ganglionic system, comprising the arteries which supply the basal ganglia.

The cerebral arteries are terminal ; that is, each branch supplies its own area and does not anastomose freely with neighbouring vessels.

The *cortical* arteries ramify in the pia mater, and are distributed to the grey matter of the convolutions and subjacent white matter. They

vary in the extent of their inter-communication in different individuals, but this inter-communication is never a free one.

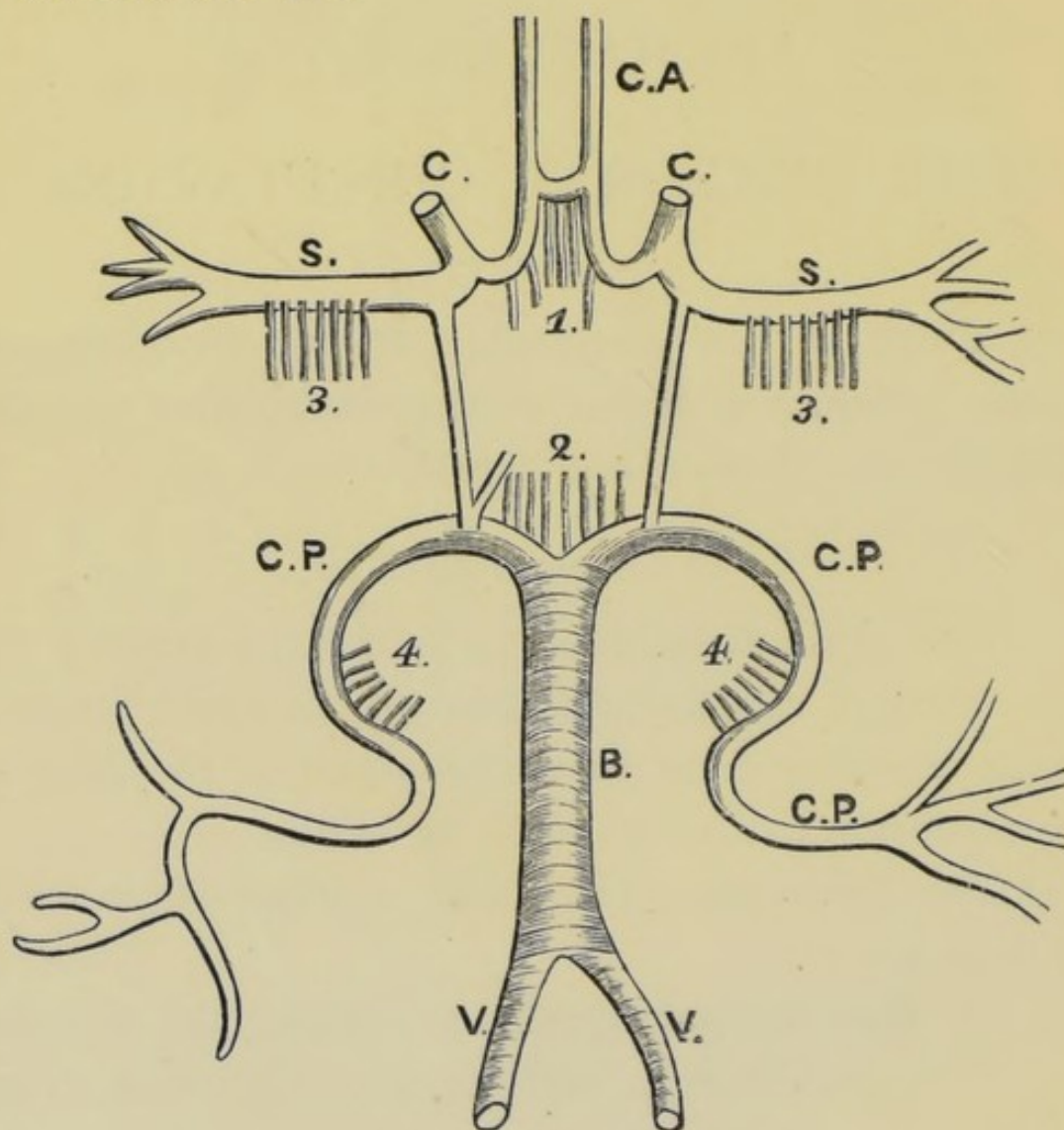


FIG. I.—(CHARCOT.)

DIAGRAM OF THE DISTRIBUTION OF THE ARTERIES AT THE BASE OF THE CEREBRUM.

C., the internal carotid artery. C.A., anterior cerebral artery. S., the middle cerebral or Sylvian artery. V., the vertebral artery. B., the basilar artery. C.P., the posterior cerebral artery. 1, 2, 3, 4, the central or perforating arteries.

Their branches are divisible into two sets—the long or medullary branches, which pass a considerable distance into the white matter, and the short or

cortical arteries proper, which supply the grey matter of the convolutions.

It is essential to know the distribution of the cerebral arteries in order to understand the effects of their rupture or obstruction. The anterior cerebral artery runs forwards over the corpus callosum along the longitudinal fissure; besides giving off the "anterior median group" of ganglionic vessels it gives off four cortical branches:—The first to the orbital convolutions, the second and third to the marginal and adjacent convolutions, the fourth to the quadrate lobe and corpus callosum. The middle cerebral or Sylvian artery is the largest and most important. It gives off five cortical branches. The first, or inferior frontal branch, supplies the third frontal convolution. The second, or ascending frontal, supplies the posterior part of the middle frontal and the chief portion of the ascending frontal convolution. The third, or ascending parietal artery, supplies part of the ascending frontal convolution, and nearly the whole of the ascending parietal convolution, together with the superior parietal lobule. The fourth supplies the inferior parietal lobule; and the fifth the superior temporo-sphenoidal convolution. Thus the middle cerebral artery supplies the convolutions especially connected with aphasia of all varieties, both motor and sensory, as well as the great motor centres of Ferrier. Obstruction of the middle cerebral artery at its origin by a thrombus or an embolon causes necrosis of those portions of the central ganglia and

internal capsule supplied by it. The cortex may or may not be affected, according to the extent of the anastomosis between the cortical arteries, and in fact it frequently escapes altogether. The posterior cerebral artery supplies the under surface of the occipital lobe, the lower portion of the temporo-sphenoidal lobe, and the sensory portion of the internal capsule.

The *central* or *ganglionic system* of arteries are small branches given off from the trunks of the main vessels. They form six groups:—an anterior and posterior median, right and left antero-lateral and right and left postero-lateral, and are all terminal arteries.

The vessels derived from the middle cerebral, the antero-lateral group, are of the most importance. They pierce the anterior perforated space and ascend to supply the corpus striatum, the internal capsule, and a portion of the optic thalamus. The largest of these branches is the *lenticulo-striate* artery (called by Charcot the artery of cerebral hæmorrhage). It ascends along the outer surface of the lenticular nucleus, crosses the internal capsule, and then passes forwards into the caudate nucleus.

These arteries are prone to rupture, arising as they do directly from a large vessel, and thereby being subjected to high pressure. Hence cerebral hæmorrhage is most frequent in this situation.

The anterior median group of vessels derived from the anterior cerebral and anterior communi-

cating arteries supply the caudate nucleus. They are important in that hæmorrhage from them may rupture into the ventricles and rapidly cause death.

The postero-lateral group of vessels supplies the hinder part of the optic thalamus. Hæmorrhage from them usually damages the posterior portion of the internal capsule and often extends into the crus.

The posterior median vessels supply the inner surfaces of the optic thalami. Hæmorrhage from these vessels finds its way into the ventricular cavity.

It is necessary to bear in mind the following facts as to the distribution of the cerebral arteries:—

The middle cerebral artery supplies the motor region of the brain, both central and cortical, also the cortical auditory centre and visual centre, and all the cortical area concerned in speech.

The anterior cerebral artery supplies only a small part of the motor region, viz., part of the leg centre (paracentral lobule) and the centre for the trunk muscles.

The posterior cerebral artery supplies the sensory portion of the internal capsule.

The region of the ganglia is especially subject to hæmorrhage. The cortical region especially to softening from obstruction of the vessels.

Obstruction of the artery to the superior temporo-sphenoidal convolution on the left side causes word deafness; the patient being unable to understand what is said to him, but not being deaf to sound.

Obstruction of the artery to the left third frontal convolution will cause motor aphasia simply.

Obstruction of the basilar artery *at its lower end* causes paralysis of all four extremities and of both sides of the face. It causes also severe respiratory trouble, dyspnœa, cyanosis, and death from asphyxia, the respiratory centre in the medulla being deprived of blood. It is generally attended with profound coma.

Obstruction of *the upper end* of the basilar artery is much less dangerous, respiration not being arrested.

The superior cerebellar artery crosses and supplies the crus cerebri and the third nerve. Occlusion of this branch causes paralysis of the third nerve on the side of the lesion and hemiplegia on the opposite side. I have met with a case in which this artery was probably obstructed.

A gentleman, aged 55, who had suffered severely from headache and vertigo, was seized with left hemiplegia and paralysis of the right third nerve, with anæsthesia of the right side of the face, the paralysis developing gradually in the course of a few hours without coma. In a week all the paralytic symptoms had passed off, and neuralgic pains were experienced in all three divisions of the fifth nerve. These pains finally ceased. There is little doubt that in this case there was thrombosis of the right superior cerebellar and upper portion of the basilar artery, the branch to the fifth nerve being also occluded.

Obstruction of *both* vertebral arteries would produce the same results as thrombosis of the basilar.

Obstruction of *one* vertebral artery produces hemiplegic symptoms.

In atheroma and syphilitic disease of the cerebral arteries, headache, vertigo, and numbness are usually present and precede hemiplegia.

The Cerebral Veins and Sinuses.

The arrangement of the cerebral veins and sinuses, while it tends to render the cerebral circulation equable, yet greatly favours the occurrence of local coagulation or thrombosis. The veins of the surface of the cerebrum pass from behind forwards to open into the superior longitudinal sinus, the direction of the blood stream in the veins being opposed to that in the sinus. The sinuses, moreover, are rigid tubes unable to collapse, and traversed by fibrous bands (chordæ Willisii), which also favour thrombosis. The cerebral veins do not anastomose freely with one another, hence thrombosis of them is a serious accident, occasioning grave damage to the cortex of the brain. The sinuses, on the other hand, have a free intercommunication. Thrombosis may occur either in the sinuses or in the veins.

Dr. Gowers believes that thrombosis of the cerebral veins apart from thrombosis of the sinuses is of frequent occurrence, and that this is probably the lesion in those cases where young children are attacked with hemiplegia, some form of mobile spasm and frequently also partial epilepsy occurring in the affected extremities.

Hemiplegia following the specific fevers is probably due to thrombosis of the cerebral veins in the motor area of the cortex.

Thrombosis of the cerebral veins and sinuses, like thrombosis elsewhere, is due either—

- I. To alterations in the blood, rendering it more prone to coagulate, or
- II. To inflammation of the walls of the vessels.

Marasmus, prolonged diarrhœa, the specific fevers, and carcinoma belong to the first group. Thrombosis from these causes usually occurs in the superior longitudinal sinus.

Disease of the adjacent bones, a blow on the head, or erysipelas and other inflammatory affections of the face and scalp belong to the second group.

In thrombosis of a sinus there is occasionally swelling of the veins outside the skull in communication with it. The occurrence of epistaxis in thrombosis of the superior longitudinal sinus is explained by the existence of a vein passing from the nose to this sinus through the foramen cœcum. The vein from the mastoid cells passes into the lateral sinus, and this sinus also communicates with the veins of the scalp behind the ear through the mastoid foramen. The occurrence of thrombosis of this sinus in necrosis of the temporal bone or in suppuration of the mastoid cells is thus explained. Obstruction of the cavernous sinus does not affect the veins of the retina, for the ophthalmic vein,

although it empties into this sinus, has a free anastomosis with the facial vein.

The veins of Galen as they pass over the corpora quadrigemina and middle lobe of the cerebellum are readily compressed by tumours in this situation, dropsy of the ventricles resulting.

THE CEREBRAL CONVOLUTIONS.



The Motor and Sensory Tracts in the Brain.

IT is unnecessary to describe here the convolutions of the brain fully, but a knowledge of their topographical anatomy is essential for the proper

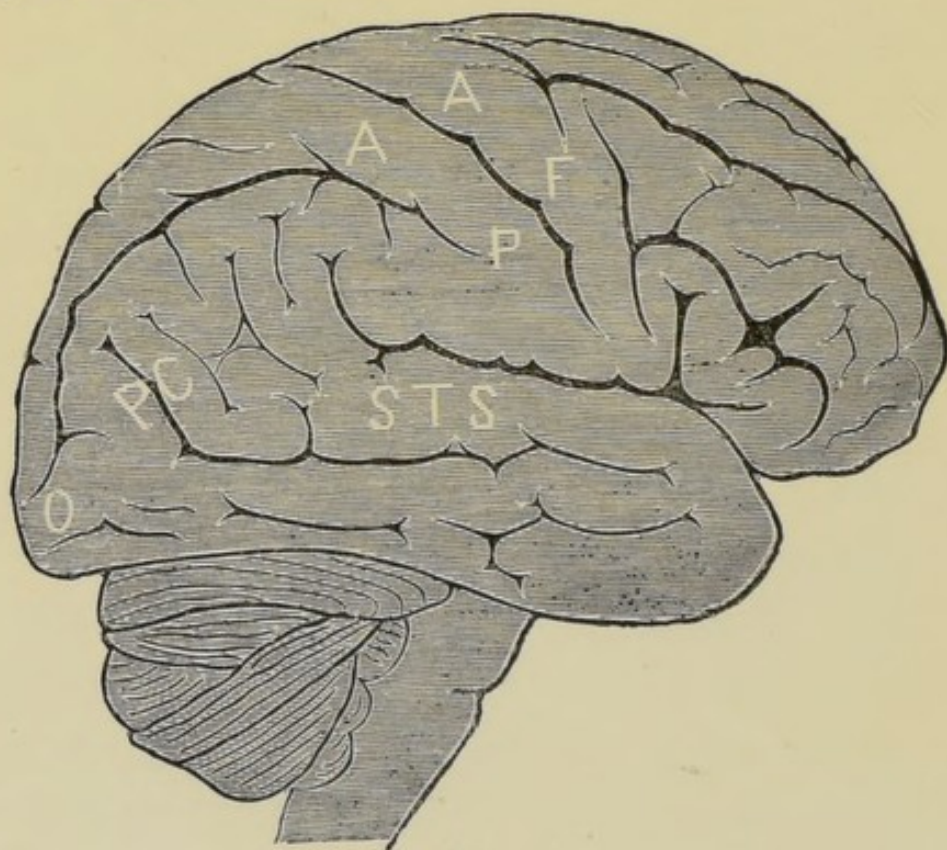


FIG. 2.

OUTER SURFACE OF THE RIGHT CEREBRAL HEMISPHERE.

A.F., ascending frontal convolution. A.P., ascending parietal convolution. S.T.S., superior temporo-sphenoidal convolution. P.C., pli courbe. O., occipital lobe.

diagnosis and localisation of cerebral lesions. We owe the greater part of our knowledge of the functions of the cerebral convolutions to Ferrier,

Hitzig, and Munk. The motor centres are situated in the convolutions bounding the fissure of Rolando, the so-called central convolutions, the ascending frontal and ascending parietal convolutions.

The tentorial surface of these convolutions is called the paracentral lobule.

Schäfer and Horsley have recently discovered that in monkeys the centres for the trunk muscles are situated in the paracentral lobule and precuneus or quadrate lobule.

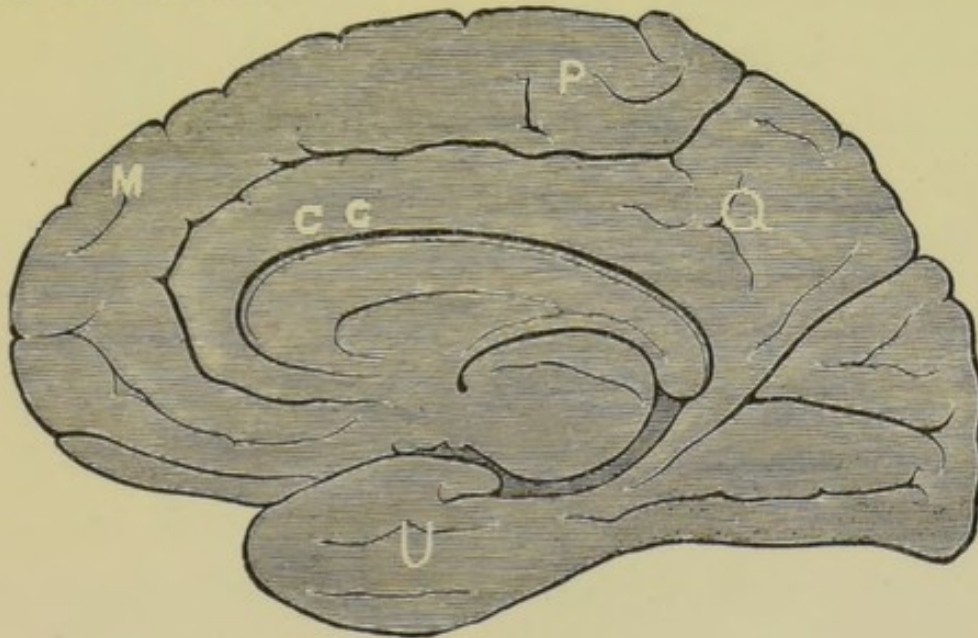


FIG. 3.

INNER SURFACE OF THE RIGHT CEREBRAL HEMISPHERE.

M., the marginal convolution. P. marks the situation of the paracentral lobule (the inner surfaces of the ascending frontal and ascending parietal convolutions). Q., the quadrate lobe. U., the uncinus gyrus. C.C., the convolution of the corpus callosum.

The leg is represented in the upper third of the central convolutions and superior parietal lobule. The arm in the middle third. The face, lips, and tongue, in order from above downwards, in the lower third.

The lower extremities of the central convolutions together with the posterior extremity of the third frontal constitute the operculum, which, besides containing the centres for the face, lips, and tongue, on the left side also is concerned with voluntary speech.

Dr. Beevor and Professor Horsley have recently proved by experimental investigation that in the cortical centre for the upper extremity, as determined by Ferrier, stimulation of the upper portion causes movements commencing in the shoulder; stimulation of the lowest portion movements commencing in the thumb; and stimulation of intermediate portions movements commencing in the wrist.

Stimulation of these areas excites convulsion in the limb represented; destruction causes paralysis. The motor regions, however, are not exclusively motor; destruction of them causes loss of muscular sense in the limbs represented by the area destroyed; moreover, in cortical disease of these regions an aura is constantly present.

The order of the motor centres from above downwards is trunk, leg, arm, face, lips, tongue.

From these areas the motor fibres converge to the *internal capsule*, a band of white matter placed between the caudate nucleus of the corpus striatum and the optic thalamus on the inner side, and the lenticular nucleus of the corpus striatum on the outer side.

It consists of an anterior and posterior half bent on each other at an angle, the concavity being directed outwards.

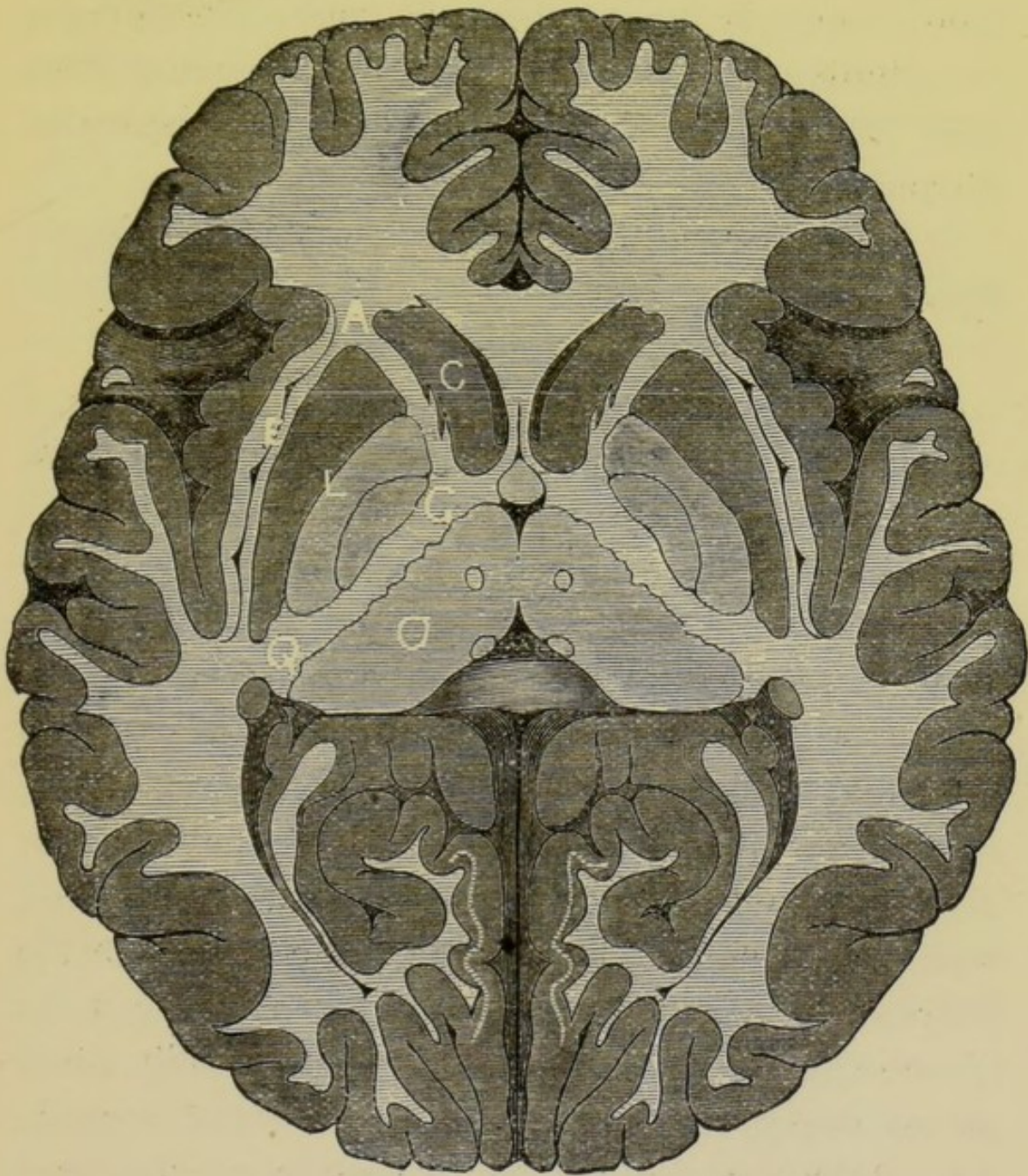


FIG. 4.—(FLECHSIG).

HORIZONTAL SECTION OF THE CEREBRUM THROUGH THE
INTERNAL CAPSULE.

C., the caudate nucleus of the corpus striatum. O., the optic thalamus. L., the lenticular nucleus of the corpus striatum, showing its three divisions. A.G.Q., the internal capsule. E., External capsule.

The function of the anterior half is unknown, beyond that it contains fibres from the cerebellum. The bend is called the knee or genu, and it contains fibres from the tongue, lips, and face. The anterior two-thirds of the posterior half contain motor fibres from the motor centres around the fissure of Rolando.

The order here from before backwards is tongue, lips, face, arm, leg, trunk.

The posterior third of the posterior half of the capsule is sensory. Destruction of it causes loss of sensation of the opposite side of the body with hemianopia, and loss also of the special senses on the opposite side.

The *motor tract* can be traced continuously from the motor convolutions to the muscles.

Thus, from the convolutions the fibres pass to the internal capsule, then to the crus cerebri forming the superficial portion or crust. Here the fibres lie in order from within outwards, tongue, lips, face, arm, leg, trunk. Thence they pass through the pons to the medulla, decussating there between the anterior pyramids to a variable degree, usually about 97 per cent. of the fibres passing down the opposite side of the cord forming the crossed pyramidal tract of the lateral column, and about 3 or 4 per cent. down the same side of the cord forming the direct pyramidal tract of the anterior column. (The pyramidal tracts of the cord are so called because they are continuous with the anterior pyramids of

the medulla.) From these tracts the fibres pass to the motor cells in the anterior cornua and thence into the motor nerve roots.

The motor tract is not interrupted by the nuclei of the corpus striatum. Disease of these nuclei alone does not cause hemiplegia except by pressure upon or involvement of the internal capsule.

In passing through the internal capsule the fibres from each limb centre keep together in bundles, so that it is possible to get a monoplegia produced by a small lesion in the capsule. Such a case is recorded in "Brain," Vol. VIII., p. 78, by Dr. Hughes Bennett.

The fibres from the tongue and face centres leave the motor tract in the pons and cross to the nuclei, from which the hypoglossal and facial nerves proceed.

Our knowledge of the *sensory tract* is much less certain and complete than that of the motor tract. Brown-Séquard taught that sensation is chiefly conducted in the grey matter, but there is evidence to show that the posterior median columns of the cord and a portion of the lateral columns in front of the crossed pyramidal tract are concerned with the upward transmission of sensory impressions.

Between the posterior roots of the spinal nerves and the internal capsule we are unable to localise accurately the sensory tract.

We know that the fibres cross to the opposite side soon after entering the cord, and that the tract

then passes up on the opposite side through the medulla, pons, crus, and posterior third of the posterior half of the internal capsule to the cerebral convolutions.

As to the exact termination of the sensory tract in the convolutions our knowledge is very imperfect. There is evidence pointing to the convolutions on the outer surface of the cerebrum about its centre, being those concerned in sensation; but Ferrier, from his experiments on monkeys, finds that the convolutions of the internal temporo-sphenoidal region, the hippocampal, and other gyri are the centres for touch, taste, and smell.

The superior temporo-sphenoidal convolution is the centre for hearing.

The occipital lobe and possibly the pli courbe or angular gyrus (Ferrier) are the centres for sight.

The centres for sight and hearing are bilaterally associated, and the centres on both sides must be destroyed to cause blindness or deafness.

The *sensory tract* in the cord and brain being much less defined than the motor, we find that hemianæsthesia is much less common than hemiplegia. Still it does occur, not from cortical mischief, but from damage to the internal capsule. I recently had a case in the Queen's Hospital of hæmorrhage into the internal capsule, in which there was complete hemiplegia and hemianæsthesia with hemianopia. The muscular sense in this case was also completely abolished on the paralysed side, the patient

six months after the attack being quite unable to recognise the position of his limbs, or to perceive even heavy weights. But the hemianæsthesia in these cases is usually much less complete and persistent than the hemiplegia as it usually results from pressure, and is an indirect symptom. Since the lenticulo-striate artery passes through the anterior portion of the internal capsule the clot resulting from its rupture will compress from before backwards the fibres from the face, lips, tongue, arm, leg, and trunk centres, and lastly the sensory tract. Hence we can readily understand why sensation is rarely affected, and if affected, why it is so quickly recovered.

In the cord, also, the diffusion of the sensory tract explains the fact that sensation usually first recovers in a myelitis.

Descending and ascending degeneration of the motor and sensory tracts respectively :—

Waller many years ago showed that nerve fibres severed from their nerve cells, experimentally or pathologically, undergo degeneration in one direction—that of functional conduction. The portion left connected with the centre remaining unaffected that dissevered degenerating; every nerve fibre depending for its nutrition upon a nerve cell.

This discovery has been of the greatest service to the anatomist and physiologist in the elucidation of the structure and functions of the brain and cord.

Thus, after hæmorrhage in the centrum ovale or in the motor region of the internal capsule, descending degeneration of the motor tract occurs; and sclerosis of the crossed and direct pyramidal tracts

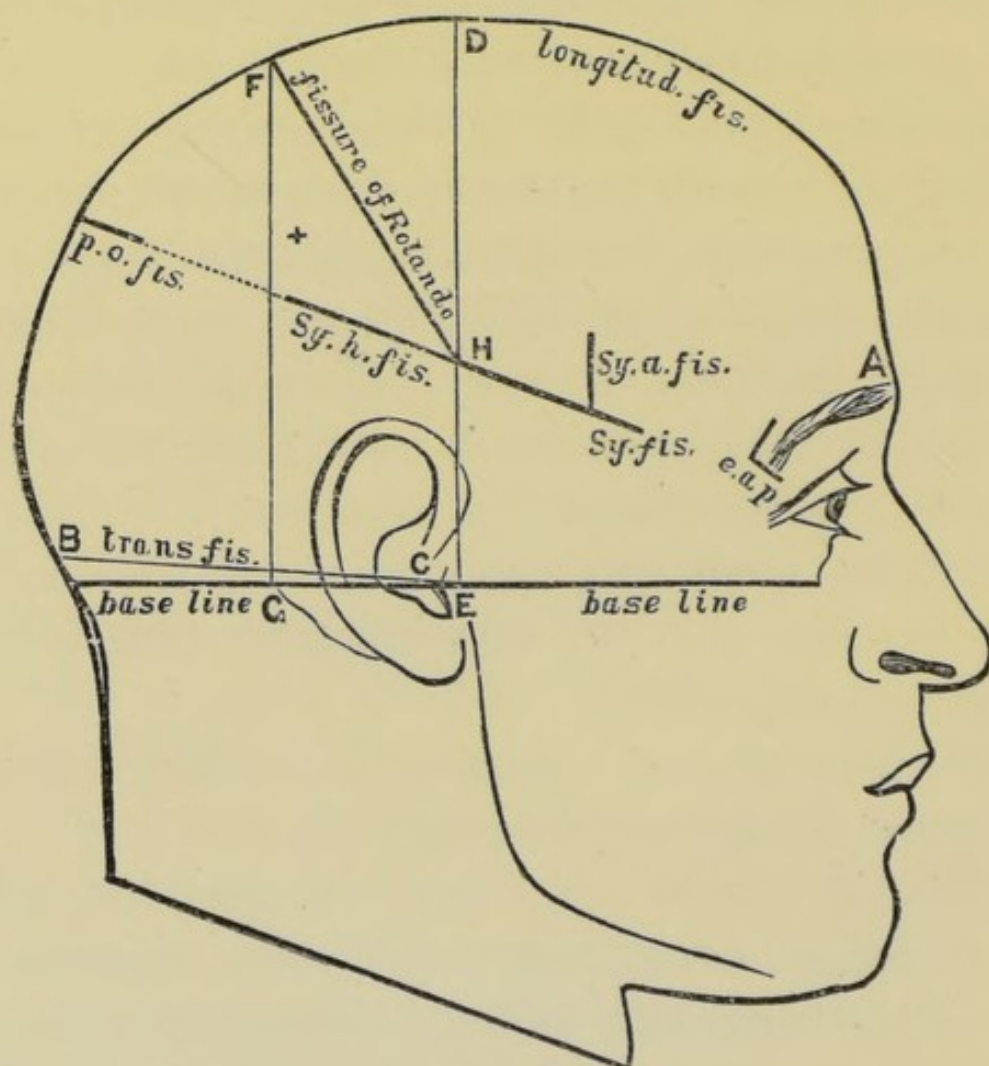


FIG. 5.

DIAGRAM TO SHOW THE RELATION OF THE CEREBRAL FISSURES AND CONVOLUTIONS TO THE SCALP. (REID.)

A, glabella. B, external occipital protuberance. *e.a.p.*, external angular process of frontal bone. BC, transverse fissure. AB, longitudinal fissure. *Sy. fis.*, Sylvian fissure. *Sy. a. fis.*, ascending limb of fissure of Sylvius. *Sy. h. fis.*, horizontal limb of fissure of Sylvius. DE, perpendicular line from depression in front of external auditory meatus to middle line of top of head. FG, perpendicular line from posterior end of base of mastoid process to middle line of top of head. FH, fissure of Rolando. *p. o. fis.*, parieto-occipital fissure. +, most prominent part of parietal eminence.

is found in the cord, the trophic cells for the motor fibres being situated in the central convolutions. This descending degeneration explains the late rigidity and exaggeration of reflexes observed in hemiplegia. If a transverse myelitis occurs, four tracts of degeneration are found descending the cord, the two crossed and the two direct pyramidal tracts, accounting for the rigidity and exaggeration of deep reflexes seen in these cases.

The Wallerian maxim applies also to the sensory tracts. In transverse lesions of the cord ascending degeneration is found in the postero-median columns, in a portion of the lateral tracts and also in the direct cerebellar tracts, the trophic cells for the sensory fibres being situated in the ganglia on the posterior roots of the nerves.

The relation of the cerebral fissures and convolutions to the scalp is of the highest importance in all operations on the brain.

Dr. R. W. Reid has recently published a practical method of ascertaining the relation of the primary fissures to the surface of the scalp (*vide* "Lancet," September 27th, 1884).

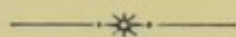
The fissure of Sylvius. To find this draw a line from a point one and a quarter inches behind the external angular process of the frontal bone to a point three-quarters of an inch below the most prominent part of the parietal eminence. Measuring from before backwards the first three-quarters of an inch of the line represents the main fissure and the

rest of the line represents the horizontal limb. The ascending limb starts from the posterior end of the line indicating the main fissure and runs vertically upward for about an inch.

The fissure of Rolando. This, and with it of course the situation of the ascending frontal and ascending parietal convolutions, is found in the following way :—First mark out on the surface of the scalp the longitudinal fissure (a line from the glabella to the external occipital protuberance) and the horizontal line of the fissure of Sylvius. Then from the base line, which runs through the lowest part of the infra-orbital margin and the middle of the external auditory meatus, draw two perpendicular lines to meet the line for the longitudinal fissure, one from the depression in front of the external auditory meatus, and the other from the posterior border of the mastoid process ; a four-sided figure is thus described on the surface of the head, bounded above and below by the lines for the longitudinal fissure and horizontal line of the fissure of Sylvius respectively, and in front and behind by the two perpendicular lines. A diagonal line drawn from the posterior superior angle to the anterior inferior angle of this space is over the fissure of Rolando.

CHAPTER II.

THE CRANIAL NERVES.



THE cranial nerves in several instances have known connections with the cortex of the brain.

The olfactory nerves commencing in the olfactorial cells of the Schneiderian membrane pass to the olfactory bulb, thence to the roots and onward to the cortex of the brain. The centre for smell is not definitely known in man, but in monkeys it is localised by Ferrier in the hippocampal gyrus.

In testing the sense of smell, substances having pungent odours should not be employed, to avoid irritation of the nasal branch of the fifth nerve.

Hyperæsthesia of the olfactory nerves may occur in hysteria, and illusions or hallucinations of smell are common in the insane.

An olfactory aura may be present in epilepsy, and is caused by irritation of a portion of the cortex.

Anosmia, diminution or abolition of the sense of smell, is present in ozæna, and is occasionally present in locomotor ataxia.

Unilateral loss of smell has been observed in hysterical and organic hemianæsthesia.

Anosmia of the left nasal cavity is sometimes associated with right hemiplegia from embolism of the left middle cerebral artery. The aphasia is usually of the sensory variety and due to softening of the angular and superior temporo-sphenoidal convolutions. The anosmia is probably due not to cortical softening, but to softening of the external root of the olfactory bulb which is supplied by the middle cerebral artery. Congenital loss of smell is caused by arrest of development of the olfactory tracts.

The Optic Nerve.

The fibres of the optic nerve partially decussate in the optic commissure, and thence can be traced through the white substance to the cortex of the occipital lobe. There is no second decussation at the corpora quadrigemina.

Destruction of one optic tract causes a loss of vision in the opposite half of each field—*hemianopia*.

A similar hemianopia results from disease of the cortex of the occipital lobe. Permanent and complete blindness only ensues when the occipital lobes and angular gyri (?) are destroyed on both sides.

Ferrier has shown that lesion of the angular gyrus in monkeys causes temporary amblyopia of the opposite eye; hence he suggests that in the angular gyrus there exists a visual centre representing the whole of the opposite field. This crossed amblyopia is accompanied with a slight restriction of the field

of the other eye, and is temporary; the temporary nature of the amblyopia being explained by the substitutionary action of the angular gyrus of the opposite side. But it is by no means generally accepted that the angular gyrus is the centre for sight in man.

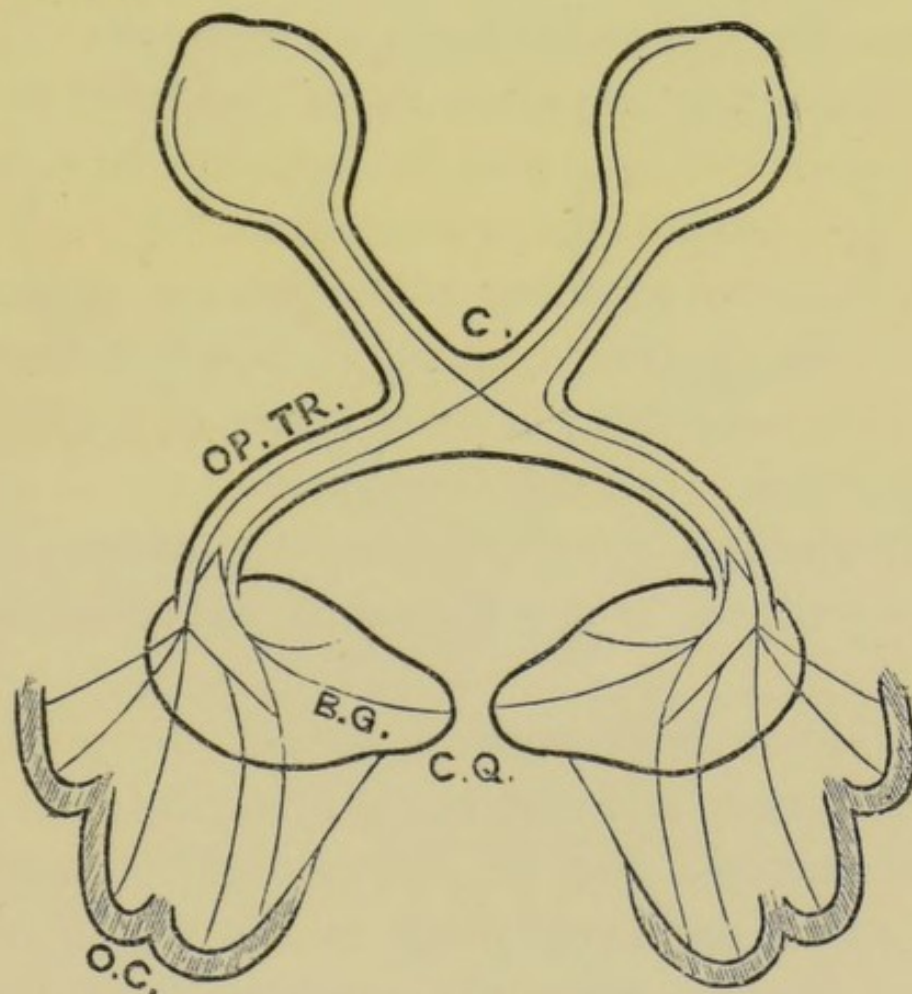


FIG. 6.

DIAGRAM OF THE DECUSSATION OF THE OPTIC TRACTS.

C. marks the semi-decussation in the chiasma. O P. T R., the optic tracts. B.G., the basal ganglia. C.Q., the corpora quadrigemina. O.C., the occipital convolutions.

Lesions of the centre of the anterior surface of the optic commissure cause bi-temporal hemianopia (blindness in the outer half of each field), the decussating fibres being damaged.

Meningitis at the base of the brain, chronic hydrocephalus causing distension of the third ventricle, or a cerebral tumour might cause bi-temporal hemianopia.

Nasal hemianopia, blindness in the inner half of each field, is rare ; it might be caused by a lesion of the outer fibres of the chiasma on each side.

Disease, then, anywhere in the optic tract beyond the commissure right into the occipital lobe causes hemianopia towards the opposite side.

When corresponding halves of the retinae are affected the resulting hemianopia is called homonymous ; when the inner or outer halves of both retinae are affected it is called heteronymous.

Hemianopia must be carefully looked for in cerebral diseases, as it is frequently unnoticed by the patient.

Persistent hemianopia is almost always due to organic disease, and very rarely hysterical ; but transient hemianopia may be due to organic disease, as, for instance, in hæmorrhage in the region of the internal capsule, when the hemianopia, if present, soon passes off, and is probably caused by compression of the optic tract.

A man with left hemiplegia informed me that at the time of the attack and shortly after he was blind with the left eye ; his was clearly a case of hæmorrhage, and he had blindness of the left half of each field of vision ; that is, he saw nothing on his left side, and he accordingly thought his left eye was blind.

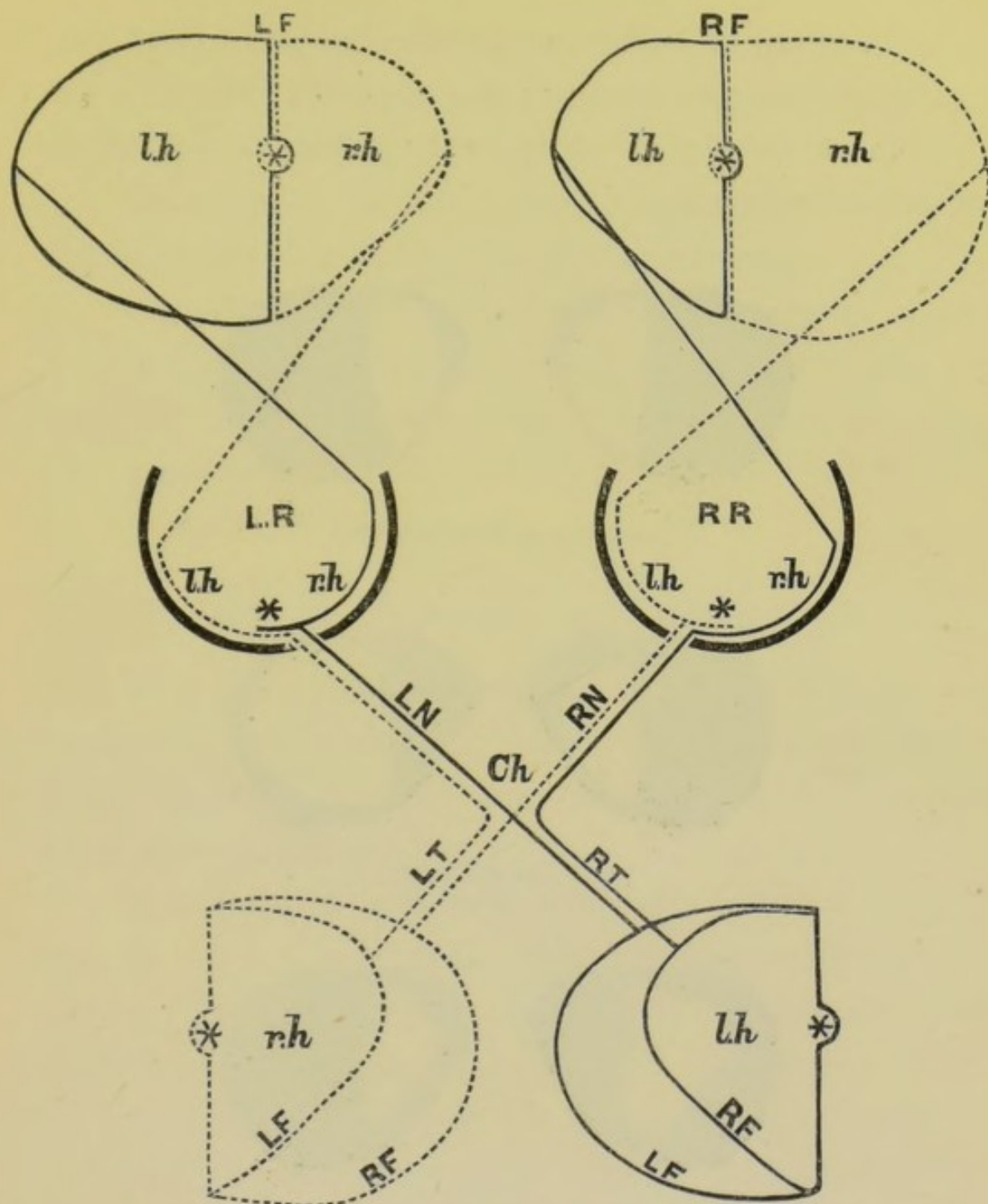


FIG. 7.

DIAGRAM OF THE RELATION OF THE FIELD OF VISION, RETINA, AND OPTIC TRACT ON EACH SIDE. (GOWERS.)

R.F. L.F., right and left fields—the asterisk is at the fixing point. R.R. L.R., right and left retina—the asterisk is at the macula lutea. *r.h.* *l.h.*, right half and left half of each retina, receiving rays from the opposite half of the field. R.N. L.N., right and left optic nerves. *Ch.*, chiasma. R.T. L.T., right and left optic tracts; below the halves of the fields from which impressions pass by each optic tract are superimposed.

Tumour or softening of the occipital lobe may cause hemianopia towards the opposite side.

Hemianopia is frequently present during an attack of migraine.



BITEMPORAL HEMIANOPIA.



LEFT HEMIANOPIA.



RIGHT HEMIANOPIA.

FIG. 8.

FIELDS OF VISION.

Impaired vision in one eye may be due to a lesion at either end of the visual tract, in the optic nerve or in the cortical centre.

If cortical or central, the pupil reflex to light is not affected, while disease of the nerve diminishes or abolishes this reflex.

In disease of the nerve, too, changes may be found in the disc with the ophthalmoscope.

Amblyopia of one eye with restriction of the field of vision and slight affection of the other eye may be caused by extensive disease of the cortex of the opposite hemisphere, but it is chiefly met with in hysterical hemi-anæsthesia.

Case of Amblyopia due to Cortical Disease.

The following interesting case was sent to me on the 10th of February by an old pupil, who diagnosed cerebral tumour, and thought the case would be of interest to me.

The patient is a boy aged 2. Several months ago he began to vomit; this continued for several weeks, the vomiting not seeming to distress him and occurring independently of food. At the same time the child frequently put his hand to the left side of the head as if in pain there. Three months ago he was attacked with convulsions, the spasm beginning in the right hand, then attacking the right side of the face and right leg, consciousness being lost late in the fit, and not being lost in every attack. The convulsions continued to occur daily for three or four weeks, when the right arm and leg and right side of the face were found to be paralysed. The leg and face have since recovered, but the arm has remained

the same. Lately the mother noticed that the child could not see, and this greatly alarmed her. Vision is not quite lost, for he runs about, though often running against obstacles. The child cannot be made to wink by passing the hand rapidly over the eyes, and cannot recognise any object presented to him.

There is a marked family history of phthisis on the mother's side, but no history of syphilis or cancer. The child is drowsy sometimes, at other times very irritable. Upon examination of the eyes no change in either fundus could be discovered, and there was no opacity of the cornea or lens. Mr. Priestley Smith kindly examined the eyes for me, and he agreed with me that there was nothing abnormal to be detected. The right hand is paralysed, the thumb being adducted into the palm and the fingers flexed over it; there is considerable rigidity, the bicipital reflex being exaggerated. The right leg seems a little weak, but the patient can walk well. The mother thinks the boy sees a little with the left eye.

As to the diagnosis.—The attacks of partial or Jacksonian epilepsy beginning in the right hand, and being followed at last by paralysis of the hand, indicate a lesion in Ferrier's motor region of the cortex. These seizures might result either from softening or tumour. The absence of optic neuritis prevents our diagnosing tumour with certainty; still the marked family history of phthisis, and the absence of any evident cause of softening, make the diagnosis of

cortical tumour almost certain. The amblyopia is evidently cortical too, for there is no change in either fundus, and the pupil responds well to light.

We may conjecture that the growth occupying the middle portion of the ascending frontal and of the ascending parietal convolutions (the centre for the hand and fingers), has extended backwards, invading the angular gyrus, and perhaps also the outer surface of the occipital lobe. Destruction of the angular gyrus on one side has been shown to cause amblyopia of the opposite eye, and partial blindness of the eye on the same side (in animals). The amblyopia is, however, said to be transitory. Destruction of the outer surface of the occipital lobe causes permanent hemianopia. It is of course impossible to map out the field of vision in an irritable restless child, but it is interesting that the mother thinks that the child sees a little with the left eye. The boy understands what is said to him and presents no other symptoms.

By *central scotoma* is meant blindness in the central portion of the field of vision.

It is usually due to (1) tobacco, (2) possibly to alcohol, and usually recovers with removal of the cause, there being no abnormal ophthalmoscopic appearances. It may, however, be due to (3) lesions near the macula lutea, as in syphilitic retinitis and the retinitis of Bright's disease. By means of the ophthalmoscope these lesions may readily be detected.

Peripheral contraction of the field of vision commonly results from atrophy of the optic nerve, either primary or following optic neuritis; it may also be due to chronic glaucoma or to retinal neurasthenia.

Subjective disturbances of sight in the form of colours or lights occur in epilepsy and migraine.

In migraine a spectrum of fixed or moving lines of a bright colour, the lines presenting a vandyked appearance, is commonly present. Sometimes a hemianopia, at other times a central scotoma or amblyopia.

Muscae volitantes—the spectrum of black objects before the eyes, fixed or moving, are frequently seen in functional disorders of the liver and in hypermetropic individuals.

Dimness of vision or *amblyopia* may be present in uræmia, hysteria, diabetes, epilepsy, diphtheria, and in all debilitating diseases, without retinal change.

Optic Neuritis.

Optic neuritis or papillitis is recognised by swelling and increased vascularity of the disc, with tortuosity of the vessels, and obscuration of its edge, partial or complete in extent. It is important to remember that vision may be unimpaired even with a considerable amount of neuritis present. Photophobia and pain in the eye are very rarely present.

Restriction of the visual field, diminution in acuity of vision, and defect of colour vision, are usually present in extensive papillitis.

The causes of optic neuritis are numerous :—

1.—Tumour of the brain being by far the most common, and with tumour we may include aneurism, hydatid, and abscess as causes.

We cannot localise or estimate the size of the tumour by the optic neuritis.

A large growth may be present without optic neuritis.

Tumours of the cerebellum are most constantly, of all tumours of the brain, attended with optic neuritis.

Generally all conditions which increase the intracranial pressure cause optic neuritis ; but in nearly all cases neuritis of the optic nerve is found, and the papillitis is due to descending inflammation.

2.—The next most frequent cause is meningitis at the base of the brain.

3.—Anæmia, amenorrhœa, and chlorosis are recognised causes and must be borne in mind, or the presence of optic neuritis in girls might cause you to wrongly diagnose the presence of organic brain mischief.

4.—Chronic Bright's disease. Occasionally it is impossible from the appearance of the fundus to diagnose between tumour cerebri and Bright's disease.

5.—Lead poisoning. At the present time I have in the workhouse infirmary a man completely blind from atrophy of both optic nerves, who was under Mr. Priestley Smith ten years ago for

neuritis from lead poisoning, the man at that time being a painter, and suffering from other symptoms of plumbism.

6.—Syphilis.

7.—Exposure to cold.

Optic neuritis occasionally occurs in the specific fevers, acute myelitis, and also in cerebral hæmorrhage.

In all these cases the neuritis is as a rule double; unilateral optic neuritis would suggest mischief in the orbit.

It is important to remember that hypermetropia predisposes to the occurrence of optic neuritis.

Optic Atrophy.

Optic atrophy may be primary or it may be secondary. In the latter case following optic neuritis, choroiditis, or occlusion of the vessels of the optic nerve.

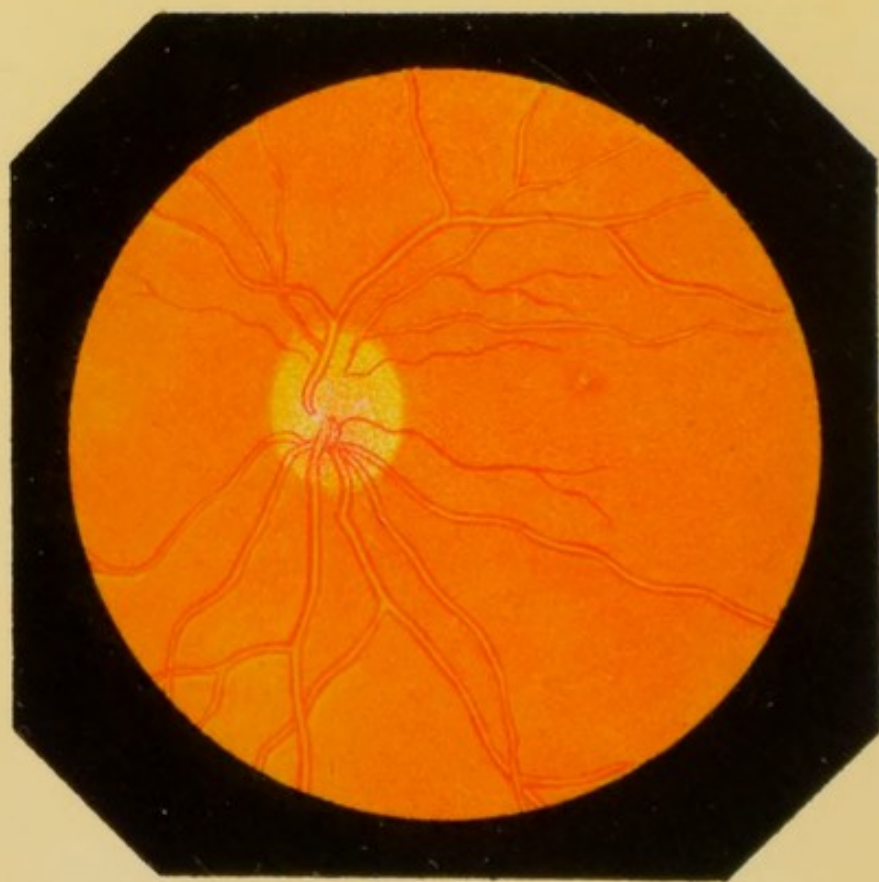
There is no diminution in the size of the disc as this depends on the size of the sclerotic opening; but the disc is excavated, the retinal vessels diminished in size, and the margin of the disc extremely well defined. The disc, moreover, is very pale, often of pearly whiteness, and at the same time vision is much impaired.

In primary atrophy the loss of sight is gradual and insidious, and progresses *pari passu* with the visible atrophy.

In secondary atrophy the loss of sight occurs first, and the atrophy is observed subsequently.



Plate 1.

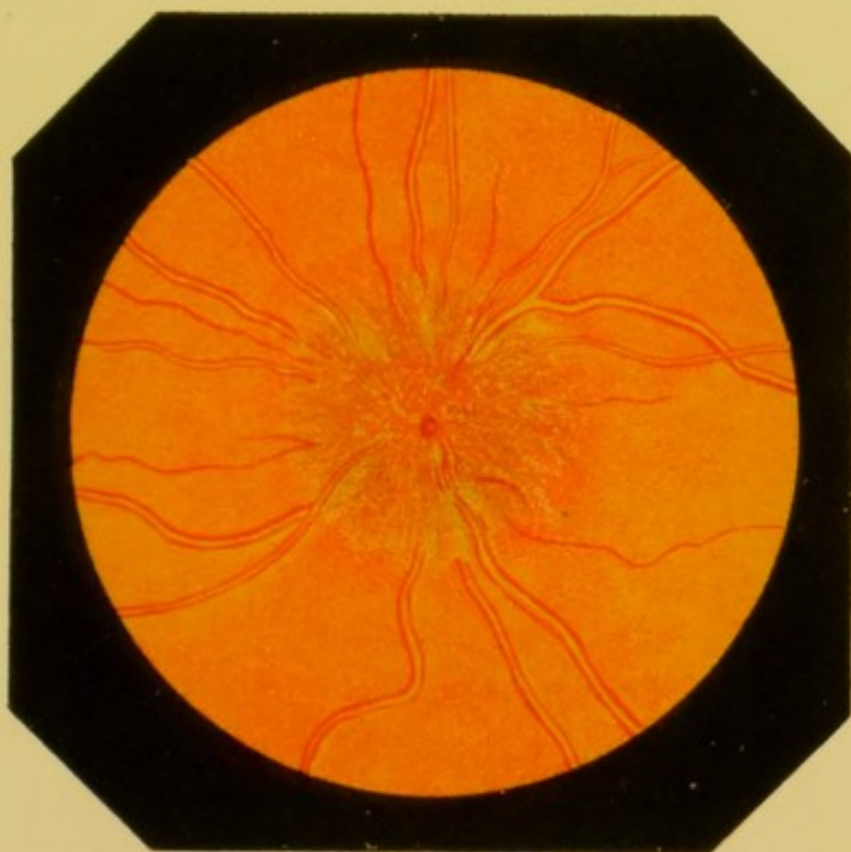


Normal fundus of Eye.

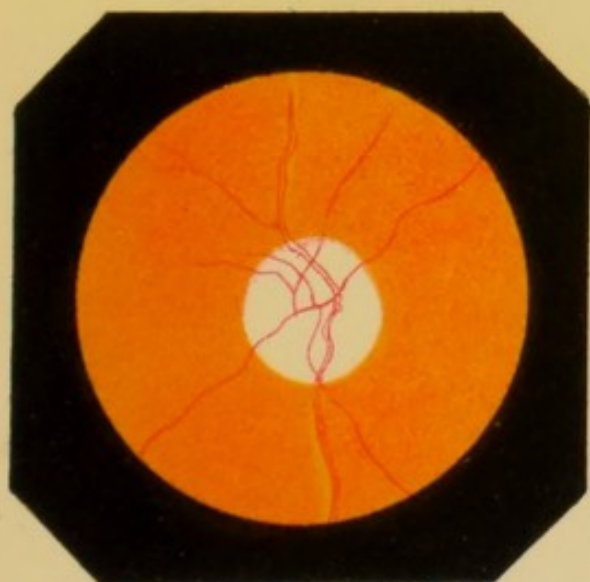
(After Ross.)



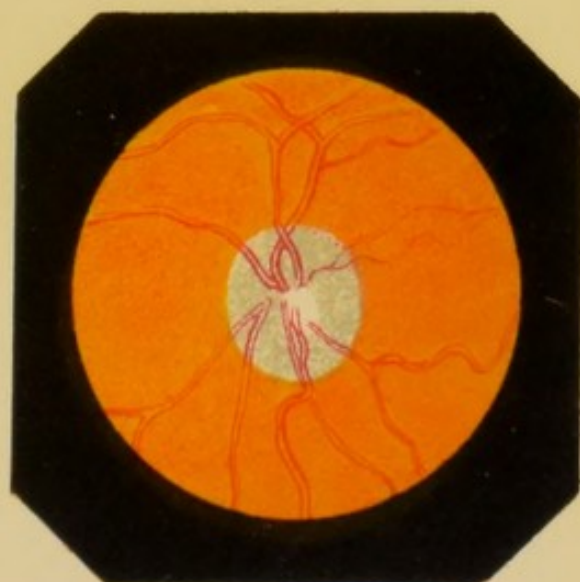




Optic Neuritis.



White disc Atrophy.



Grey disc Atrophy.

(After Ross.)



Primary atrophy occurs in the following conditions:—

1.—It may exist by itself, and without known cause; that is, it may be idiopathic.

2.—It is occasionally hereditary.

3.—It is common in locomotor ataxia, of which it may be the first symptom, and may exist for years without other symptoms; moreover, in this disease it is often of the grey variety.

4.—It may occur in other chronic degenerative diseases of the nervous system, *e.g.*—

General paralytic dementia,
Multiple sclerosis, and also in
Lateral sclerosis.

5.—It may be caused by syphilis, or

6.—By diabetes.

7.—Pressure on the optic nerves or on the optic commissure at the base of the brain may cause optic atrophy without neuritis; hence the frequency of blindness in children suffering from chronic hydrocephalus, the pressure being caused by the distended third ventricle.

8.—Unilateral optic atrophy occurring on the side of the headache is occasionally met with in migraine.

Hæmorrhage into the retina occurs in heart disease, especially in mitral regurgitation; in chronic Bright's disease, and after suppression of menstruation.

In gout, pernicious anæmia, leucocythæmia, and scurvy, whenever, in fact, the corpuscular richness of

the blood is below 50 pc, hæmorrhage into the retina is likely to occur. It also occurs in ulcerative endocarditis, and this fact might help us to diagnose this disease from simple endocarditis.

Diplopia often occurs in diseases of the cranial nerves and brain. It is usually binocular and a result of squint, due to recent paralysis of some of the ocular muscles. It may be uniocular in rare cases, such as early cataract.

The various movements of the eyes must be carefully tested in all cases.

Hemeralopia means that defect where vision is better in a dim than in a bright light. It is observed in commencing cataract.

Nyctalopia where vision is good in a bright light, but unduly defective in a dim light. It occurs in retinitis pigmentosa and in exhaustion of the retina. There is, however, great confusion as to the proper meaning of the terms hemeralopia and nyctalopia.

Asthenopia may be accommodative, muscular, or neurasthenic. *Accommodative asthenopia* is often present in hypermetropic individuals, and in them often appears suddenly during or after any illness. The symptoms, which are caused by enfeeblement of the ciliary muscle, are a sense of pressure in the eyes and headache after reading or sewing, or when near objects are looked at for any length of time, the objects soon becoming indistinct.

Muscular asthenopia, or insufficiency of the internal recti muscles, is common in myopic

individuals. The subjects of this form of asthenopia complain that after reading, &c., for a time, they find objects spreading and becoming indistinct, and often doubled; they also usually complain of headache.

Neurasthenic asthenopia, or *retinal anæsthesia*, is occasionally observed in children over-worked at school and underfed, in chlorotic and anæmic persons, and in all conditions in which the nervous system is debilitated. The field of vision is contracted, and acuity of vision diminished, but these symptoms are fluctuating. Objects looked at too long may disappear from view.

Colour vision is always affected in atrophy of the optic nerves.

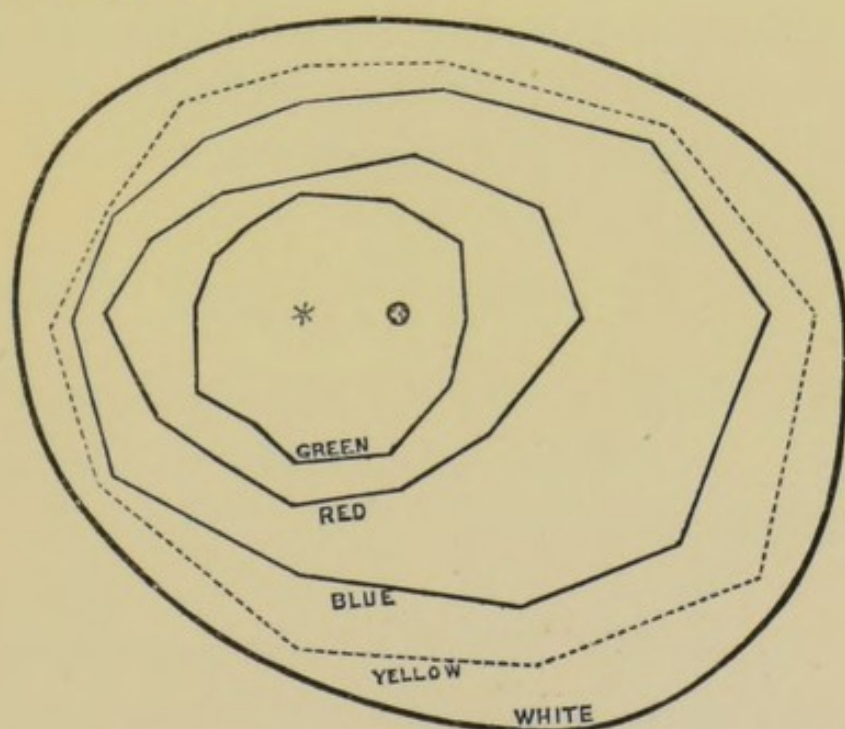


FIG. 9.

DIAGRAM SHOWING THE FIELDS OF COLOUR VISION IN A NORMAL EYE ON A DULL DAY. (GOWERS.)

The asterisk indicates the fixing point, the black dot the position of the blind spot.

In health the various colours are not equally perceived in different areas of the field of vision.

Blue is best seen at the periphery ; then comes yellow, red, and green from without inward.

Colour blindness is frequently congenital, but it may be present in locomotor ataxia and other organic lesions, or it may be due to functional causes, such as tobacco, alcoholism, hysteria, and migraine.

The pupil symptoms are of great importance in cerebral disease.

The third nerve supplies the circular muscular fibres or sphincter of the pupil ; the cervical sympathetic conveys impressions to the dilating muscular fibres from the cervical region of the cord. (It is denied by some that there is any dilatator muscle.)

Contraction of the pupil is brought about reflexly, the third nerve having no tonic action.

The sympathetic nerve exerts a tonic dilating influence upon the iris.

Myosis. Persistent contraction of the pupil occurs—

1.—In hypermetropia. In watchmakers and engravers, who habitually strain the muscles of accommodation.

2.—In paralysis of the cervical sympathetic nerve or destructive lesions of the cilio-spinal centre.

3.—In cerebral irritation.

4.—In photophobia.

5.—In hæmorrhage into the pons.

6.—After certain poisons, as calabar bean applied locally, opium internally.

7.—In certain movements of the eye, *e.g.*, convergence and accommodation for near objects.

Myosis is common in locomotor ataxia, from disease probably in the cervical region of the cord.

In destructive lesions of the brachial plexus contraction of the pupil on the side of the injury occurs.

The dilating fibres of the cervical sympathetic nerve pass from the cilio-spinal centre in the cervical region of the cord by the anterior roots of the first dorsal nerve. Ferrier has shown that the intrinsic muscles of the hand are supplied by this nerve, and a small pupil unable to dilate when shaded has been observed in the early stages of progressive muscular atrophy.

Mydriasis. Persistent dilatation of the pupil is observed—

1.—In optic nerve atrophy, except when due to locomotor ataxia.

2.—In paralysis of the third nerve.

3.—In glaucoma.

4.—In cerebral compression.

5.—In shock and conditions of low tone, from sexual excess or other cause.

6.—From the action of drugs, *e.g.*, atropia, cocain, &c.

Atropia acting on the third nerve.

Cocain on the sympathetic.

Argyll-Robertson pupil—this is that condition described by Dr. Argyll-Robertson, where the pupil responds to accommodation, but does not respond to

light. This condition is very commonly present in locomotor ataxia.

The break in the pupil reflex for light is probably situated near the floor of the aqueduct of Sylvius, for the Argyll-Robertson symptom is often present when sight is unaffected, showing that the lesion is not in the optic nerve, and when accommodation is normal showing that the third nerve is unaffected.

Loss of the pupil reflex for light may exist either with or without myosis, the latter being a spinal, the former a cerebral symptom.

This condition of the pupil is not limited to locomotor ataxia; it may be met with in general paralysis of the insane and in multiple sclerosis.

We can readily produce reflex dilatation of the pupils by pinching the skin and in many other ways, but reflex contraction only by exposing the eye to a bright light.

The size of the pupils after death is no reliable indication of their size during life.

Inequality of the pupils is observed most frequently in general paralysis of the insane, less frequently in multiple sclerosis and locomotor ataxia.

The pupils may be unequal or irregular from old iritis due to syphilis, and a pigmentary deposit about the circumference of the pupil is important evidence of syphilitic infection.

Clonic spasm of the iris I have observed in multiple sclerosis, in cerebellar tumour, and also in locomotor ataxia.

The conjugate movements of the eyes may be lost in certain directions. In cerebral hemiplegia from cerebral hæmorrhage, conjugate deviation of the eyes from the paralysed to the sound side is observed, the head being also rotated to the non-paralysed side. These deviations are, however, usually transitory. This shows that these movements are governed by the opposite side of the brain usually, but that they are also represented by the hemisphere on the same side. In epilepsy, spasmodic conjugate deviation to the convulsed side is observed.

Loss of the movements of the eyeballs either upwards, downwards, or to either side may be present in disease, the lesion being situated either in the corpora quadrigemina or in the cerebellum.

In coma the eyeballs will be found to have lost their parallelism—that is, they are divergent.

Loss of the conjugate movements of the eyeballs downwards is usually accompanied with loss of the power of convergence and accommodation. Paralysis of the internal muscles of the eye—namely, of the sphincter and dilatator pupillæ and the ciliary muscle, may exist without the external muscles being affected. This affection, named by Hutchinson *ophthalmoplegia interna* (the pupils neither responding to light or accommodation), may be caused by syphilis or diphtheria, and is occasionally met with in locomotor ataxia and multiple sclerosis.

Paralysis of the motor nerves of the eyeballs should at once cause you to look for locomotor

ataxia, syphilis, diphtheria, or some lesion at the base of the brain.

Bilateral progressive paralysis of the external muscles of the eyeballs was named by Hutchinson *ophthalmoplegia externa*. It often occurs in the course of chronic degenerative nervous diseases, such as locomotor ataxia or multiple sclerosis, but it may be due to syphilis or cold. Last year a case was sent to me by Dr. Wood White, which resulted probably from exposure, and which was quickly cured by large doses of iodide of potassium.

Case of Ophthalmoplegia Externa.

J. B., a man, aged 67, a japan worker, was admitted on November 17th, 1885, with almost complete paralysis of the muscles supplied by the right third nerve, and the left third and sixth nerves. He could tell nothing of his family history of importance. He himself had always been healthy, and had never suffered from rheumatism, gout, or syphilis. About five weeks before his admission he was much exposed to cold weather, after which he had conjunctivitis of the left eye and dropping of the left eyelid; the right eyelid also gradually dropped a few days later, but not to such an extent as the left.

Dr. White sent the following report:—"Vision in each eye = $\frac{5}{8}$. As the lenses were slightly hazy, this was considered normal. Both fundi quite healthy. Slight tortuosity of veins alone was noticed. Fields of vision normal." When admitted, there was nearly complete ptosis on the left side, and partial ptosis

on the right. The right eye could be moved outwards, and downwards and outwards to the normal extent, but the upward, inward, and downward movements were much restricted. The pupil was in a medium degree of dilatation, and responded normally to light and accommodation. The left eye was much restricted in its movements, in all directions except downwards and outwards. The pupil on this side was a little larger than the right, but responded normally. The muscles supplied by the third nerve on the right, and by the third and sixth nerves on the left, were affected. The plantar reflexes were absent; the knee-jerk on both sides was normal. He complained of double vision; and occasionally of slight vertigo, which caused him to stagger in his walk; and of frontal headache. The memory was defective. There was no albumen in the urine, no fever, and no change in either fundus oculi.

The patient was treated with iodide of potassium in increasing doses, with counter-irritation over both temples. He improved daily, and was discharged on December 7th, greatly improved, having no difficulty in keeping the eyelids open, and the movements of the eyeballs being almost normal. At the time of his discharge he was taking thirty-six grains of iodide of potassium thrice daily.

As to the diagnosis in this case:—The symmetry of the paralysis, the third nerve being affected on both sides; and the non-implication of the internal muscles of the eye, the iris responding normally

with preservation of the conjunctival reflex; indicated a central lesion. Mr. Hutchinson reported seventeen cases of "ophthalmoplegia externa," or "symmetrical immobility of the eyes with ptosis," in the "Medico-Chirurgical Transactions" (Vol. LXII., 1879). Incompleteness in the degree of paralysis was a marked feature. Inherited or acquired syphilis was present in ten out of his seventeen cases, rheumatism in a few; ten of the patients were males, some quite old men. Usually the onset was gradual, but occasionally rapid. The recovery was not complete in any one case. One *post-mortem* examination was made, and a degeneration of the nuclei of origin of the motor nerves to the eye, similar to that met with in the spinal cord in progressive muscular atrophy, was found. Dr. White pointed out, and I agreed with him, that the rapid onset, after exposure to cold, indicated in this case an inflammatory rather than a degenerative lesion. This was proved by his speedy improvement under iodide of potassium in large doses, and counter-irritation. Mr. Hutchinson advises iodide of potassium in large doses, and one patient of his took an ounce and a half in twenty-four hours with good results.

January 14th.—The patient had now completely recovered, the movements of both eyes being normal. He had taken half-drachm doses of the iodide thrice daily since he left the hospital.

Case of loss of the conjugate movement of the eyeballs downwards, of convergence, and of accommodation.—

M. O., a woman, aged 38, was sent to me in March last, complaining of giddiness. She said that she was taken ill after her last confinement twelve months previously, her illness coming on gradually. When standing she suffered from vertigo, which made her stagger, and frequently fall. The vertigo disappeared when the eyes were closed, and when she was lying down. She had had two miscarriages, two children died young, four were alive and well. No definite history of syphilis could be obtained. Her sight was not defective, but when reading the letters became blurred. The conjugate movement of the eyeballs downwards, the power of convergence, and of accommodation were lost. In monocular vision also these movements were lost. The movement of the eyeballs to either side was unaffected, and she could look up, but when doing so had great difficulty in bringing the eyeballs down again, and had no power at all of rotating the eyeballs downwards. There was nothing abnormal in either fundus, the pupils were of normal size, and responded normally to light and accommodation. The intellect was unaffected, the only psychical change being inability to bear the least noise. There was a slight general increase in the tendon reflexes, with slight ankle clonus, the increase being greater on the left side. In walking she held her neck and body stiffly, and seemed afraid of stepping out, evidently on account of vertigo. There was no nystagmus, no tremor, and no alteration of speech.

Loss of the movement of the eyeballs downwards is nearly always accompanied with loss of convergence and accommodation, the three defects having been often observed associated, and in the absence, too, of all other symptoms. The centre governing these movements probably resides in the cerebellum, and I think there is little doubt that the above patient has cerebellar disease, but as to its nature there is not sufficient evidence to justify a positive opinion. I am inclined to consider it a sclerosis.

Nystagmus or clonic spasm of the muscles of the eyeballs, causing oscillation, is always bilateral.

Its causes may be local or central.

Congenital defects, cataract, corneal opacity, and retinitis pigmentosa are some of the local causes.

Nystagmus of central origin arises from disease of the cerebellum and its peduncles.

It also frequently occurs in cerebellar tumours, multiple sclerosis, and more rarely in locomotor ataxia.

Albinos nearly always suffer from nystagmus. Miners' nystagmus is also well known.

The movements of the eyelids and the share taken by the globes in these movements must be understood to appreciate the alterations observed in exophthalmos. Dr. Gowers, in the "Medico-Chirurgical Transactions" (Vol. LXII.), has fully described and explained the lid movements.

In hysterical ptosis the upper lid is depressed by a gentle contraction of the orbicularis; if the

patient is directed to look upwards the levator contracts with the superior rectus, and the patient is obliged to contract the orbicularis strongly to prevent the lid being raised, the nature of the case being thus disclosed.

The *third and fourth* cranial nerves have not yet been traced to the motor convolutions.

The *third nerve* has its superficial origin on the inner side of the crus, immediately in front of the pons; hence its implication in tumours of or near the crus, a crossed paralysis being produced, the third nerve being paralysed on the same side as the lesion, the hemiplegia being on the opposite side. The nucleus of the third nerve is situated on the floor of the aqueduct of Sylvius, and is the seat of three centres. From before backwards these are—

- 1.—The centre for accommodation.
- 2.—The centre of the reflex contraction of the iris to light; and
- 3.—The centre regulating the external muscles of the eye.

Lépine localises the cortical centre of the *motor division of the fifth nerve* in the lower third of the ascending frontal convolution. Dr. Gowers is of opinion that the fifth nerve is the sole nerve of taste, and that the glosso-pharyngeal has nothing to do with this function.

The chorda tympani is the nerve of taste to the anterior two-thirds of the tongue, but it is probably derived from the fifth nerve by the vidian.

Dr. Gowers has published a case where there was paralysis of one fifth nerve only, due to disease at the surface of the pons, in which taste was entirely lost on that side, not only at the front of the tongue, but also at the back, on the soft palate, and on the palatine arches.

The cortical centre for taste is not yet known in man.

Certain trophic affections are met with in the area of distribution of the fifth cranial nerve.

1.—Herpes zoster frequently occurs, and when in the area of the ophthalmic nerve may destroy sight.

2.—Glaucoma probably in some cases depends upon irritation of the fifth nerve, increase of tension being frequently observed in trigeminal neuralgia.

3.—Neuro-paralytic ophthalmia takes place in irritation of the fifth nerve by intra-cranial tumours or meningitis, the cornea quickly becoming cloudy. In severe cases ulceration followed by perforation of the cornea and collapse of the globe occurs. These changes are not due to anæsthesia of the eyeball, rendering the organ liable to injury from dust, &c., for in complete anæsthesia of the conjunctiva ophthalmia may be absent, and, moreover, ophthalmia may be present without anæsthesia. It is most probable that the phenomena are due to lesion of trophic fibres, which descend from the Gasserian ganglion.

4.—Unilateral progressive facial atrophy is supposed to be due to a lesion of the trophic fibres of the fifth nerve.

The *sixth* nerves running on the under surface of the pons parallel to the basilar artery are very liable to be compressed by tumours of the pons. The sixth is close to the fifth, and both these nerves on one side are apt to be compressed by tumours of the lateral lobes of the cerebellum which grow forwards.

Lesions of the nucleus of the sixth nerve give rise to paralysis of the external rectus on the side of the lesion, and of the internal rectus on the opposite side, thus causing a conjugate deviation of the eyes away from the side of the lesion.

The *facial nerve* is connected, as Ferrier has proved, with the lower extremities of the ascending parietal and ascending frontal convolutions; the fibres pass from these convolutions through the internal capsule to the pons, where decussation occurs.

Dr. Gowers thinks it probable that those fibres of the facial nerve which supply the orbicularis oris muscle arise from the hypoglossal nucleus; in this way the implication of the lips in bulbar paralysis is readily explained. From clinical evidence, also, Dr. Gowers believes that the facial nerve has no real origin from the nucleus of the sixth.

Facial paralysis may be due to lesion of the nerve at its nucleus or beyond its superficial origin—that is, it may be peripheral, or it may be due to implication either of the cortical centre or of its path between this centre and its nucleus of origin.

Peripheral facial paralysis is characterised by the following symptoms:—

1.—The paralysis is complete, all its external branches being implicated.

2.—The conjunctival reflex and associated movements are lost.

3.—Qualitative electrical changes are present.

4.—There is inability to close the eyelids (lagophthalmos).

In cerebral facial paralysis the branches to the upper part of the face are not implicated, the associated movements being preserved; and there are no qualitative electrical changes.

Spasm may occur in the area of distribution of the facial nerve (convulsive tic). Any direct or reflex irritation of the nerve may occasion it, and the spasm may affect all the muscles or be limited to one or more. Trigeminal neuralgia and carious teeth are common causes. It may also result from cold and emotional disturbances, worry especially being a powerful factor in its production. The spasm may be tonic or clonic. The tonic form may be mistaken for facial paralysis of the opposite side, or for contracture resulting from previous paralysis. But the immobility is on the side to which the face is drawn, the opposite being the case in paralysis; moreover, the electrical reactions are normal in mere spasm.

The *auditory nerve* has its cortical centre in the superior temporo-sphenoidal convolution. Destruction of this convolution on both sides is necessary to

produce complete deafness, as they are bilaterally associated; destruction of the left convolution causes word deafness. The auditory nerve fibres can be traced to four nuclei in or close to the floor of the fourth ventricle. Decussation takes place beyond these nuclei, and fibres pass; some to the cerebellum which

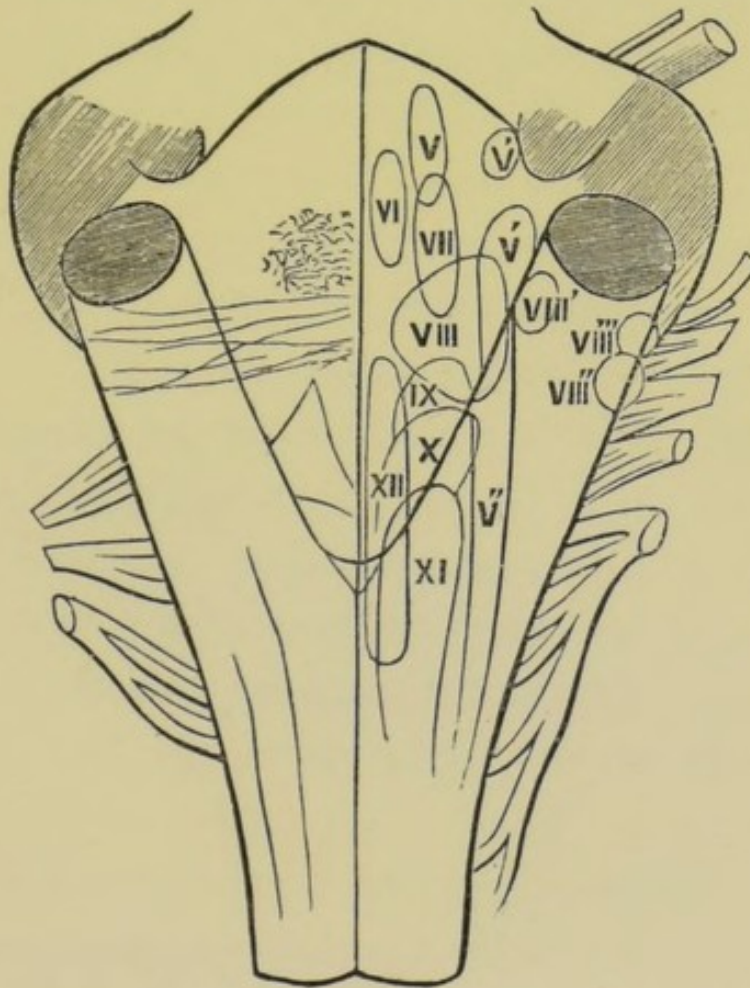


FIG. 10.

VIEW OF THE FLOOR OF THE FOURTH VENTRICLE. (ERB.)

The nerve nuclei being diagrammatically represented, the Roman numerals marking the situation of the corresponding nerve nuclei.

probably are concerned with equilibration, and others through the internal capsule to the superior temporo-sphenoidal convolutions.

The spinal accessory nerve.—Spasm of the muscles supplied by the spinal portion of the spinal accessory nerve constitutes wry neck. The spasm may be clonic or tonic, and the clonic form unilateral or bilateral. The bilateral variety (eclampsia nutans) is chiefly observed in children, the body and head being constantly flexed and immediately relaxed. There is a tendency for this variety to pass into epilepsy and idiocy. I have met with three cases of *unilateral clonic spasmodic wry neck*. One patient, a woman, became maniacal, and had to be sent to the asylum. In another case Mr. Jordan Lloyd excised a portion of the nerve, a cure resulting. The third was unrelieved by medical treatment, and refused to be operated on. The spasm frequently extends to the muscles of the face and upper extremity.

These cases, when of several months' duration, are most obstinate and difficult to remedy. Excision of a portion of the nerve offers the best chance of recovery.

It occasionally occurs in children, especially in girls, when it is usually amenable to treatment. Two cases recently under my care were cured by Faradisation.

The *pneumogastric nerve* has a more extensive distribution than any other cranial nerve.

It leaves the medulla in company with the glossopharyngeal and spinal accessory nerves, arising from a nucleus situated on the floor of the fourth ventricle, just external to the hypoglossal nucleus.

The cortical connections of this nerve are as yet unknown. It transmits sensory impressions from the larynx, lungs, heart, œsophagus, and stomach.

Its cardio-inhibitory fibres and probably all its motor fibres are derived from the spinal accessory nerve.

Paralysis of the pharynx is caused by disease at the base of the brain, by lesions of the pons and medulla, and is also observed as a sequel of diphtheria.

Laryngeal paralysis sometimes occurs from disease of the medulla, or in cases of hæmorrhage into this organ.

It occurs in bulbar paralysis, and occasionally in multiple sclerosis and locomotor ataxia.

In locomotor ataxia, paroxysms of coughing, something like those of pertussis, may occur (laryngeal crises), and may prove fatal. In severe cases, symptoms of asphyxia and unconsciousness occur.

These crises, which usually appear early in the course of this disease, are probably caused by irritation of the nuclei of the vagi, or of the spinal accessory nerves, in the medulla.

In all cases of locomotor ataxia the larynx should be examined, for bilateral paralysis of the abductors, which may give rise to sudden asphyxia, may be present.

The *hypoglossal nerve* is connected with the lower extremity of the ascending frontal convolution, lesion of this area causing paralysis of the opposite side of the tongue.

The fibres of the nerve pass from this centre through the internal capsule in the situation previously described ; thence to the nucleus of the hypoglossal nerve in the medulla, decussation taking place between the cortical centre and the nucleus in the medulla.

In hemiplegia, the tongue, when protruded, points to the paralysed side, being tilted over by the action of the sound genio-hyo-glossus ; when in the mouth the raphé is concave towards the sound side.

The tongue is paralysed in bulbar paralysis and may also be affected in locomotor ataxia.

Paralysis of the tongue by itself most commonly results from disease of its cortical centre ; but it may be caused by a small lesion in the internal capsule.

When paralysis of the tongue is complete, it falls back in the cavity of the mouth, and may close the glottis ; this accident frequently occurs in deep narcosis from inhalation of chloroform.

When one hypoglossal nerve or its nucleus of origin is diseased, atrophy of the half of the tongue occurs, in some cases being well marked.

CHAPTER III.

SYMPTOMS OF BRAIN DISEASE.



Paralysis—Hemiplegia, Monoplegia.

In the diagnosis of diseases of the nervous system, as in fact in the diagnosis of all diseases—

1.—You should observe the symptoms, and notice which are the most prominent.

2.—You should enumerate the different causes which might give rise to these symptoms, and

3.—Eliminating them one by one, determine the most probable cause in the case under notice.

Hemiplegia.

In hemiplegia the movements most paralysed are those most highly specialised or most volitional. The muscles of the trunk, and those muscles of the upper part of the face which are habitually bilaterally associated in their action, usually escape.

Dr. Broadbent explained the escape of the trunk muscles by supposing that when those on one side were shut off from the motor area of the cortex by a lesion in the internal capsule, they could still be set in action by the opposite side of the brain, there being a free communication between their

corresponding nuclei in the cord. But Mr. Horsley has recently determined the seat of the trunk centre on the tentorial surface of the convolutions, the fibres from this centre passing through the internal capsule behind the leg fibres. The order of the motor paths in the internal capsule from before backwards is tongue, lips, face, arm, leg, trunk; a hæmorrhage from the lenticulo-striate artery involves these fibres from before back, laceration occasioning a permanent, and mere pressure a temporary paralysis. The order of recovery is first the trunk, then the leg, then the arm as absorption of the clot takes place; the diminution of pressure occurring first at the part most removed from the seat of hæmorrhage. Thus the usual escape of the trunk muscles, and their rapid recovery when affected, in hæmorrhage into the internal capsule are readily explained by the anatomical arrangement of the fibres in the internal capsule. The trunk muscles are affected in hemiplegia if the hæmorrhage is large enough to implicate the trunk fibres. Hemianæsthesia is present when the clot compresses or destroys the posterior third of the posterior half of the internal capsule. This portion of the capsule is supplied by the posterior cerebral artery, so that while hemianæsthesia does occasionally occur in hæmorrhage from the lenticulo-striate branch of the middle cerebral artery, obstruction of this artery by an embolon or thrombus does not cause hemianæsthesia.

Hemiplegia occurring in early life is characterised by the following features:—(1) the leg nearly always

recovers ; (2) aphasia is transitory, and so is any sensory loss that may be present ; but (3) the limb may be stunted in its growth ; and (4) the intellect may be affected.

To perceive paresis in the lower facial muscles, we ask the patient to show his upper teeth by elevating the upper lip. One side may be elevated before the other, or to a greater extent. It would not do to tell the patient to smile, for emotional movements may be present while voluntary movement is lost. Emotional movements are probably innervated from either hemisphere. If deep coma is present, it is sometimes difficult to tell which side is paralysed. We must then look for conjugate deviation of the eyes, which is usually present and is away from the paralysed side. Or pinching the skin may cause movement on one side. The skin reflexes may be abolished or diminished on the paralysed side. The paralysed side may be rigid or flaccid.

Rigidity may come on immediately with the onset of hemiplegia ; this is called *initial* rigidity, and is due to irritation of the motor tract ; or it may come on within a few days—*early* rigidity, which is probably due to inflammatory changes. In a few weeks, if the lesion be a severe one, *late* rigidity due to descending degeneration of the crossed pyramidal tract supervenes. It is accompanied with exaggeration of all the deep reflexes, and usually also of the skin reflexes. The occurrence of ankle clonus within a few days of the onset of hemiplegia is a bad sign.

The flexor and adductor muscles being more powerful than the extensors and abductors, adduction and flexion prevails in the arm and leg. After late rigidity has lasted some time structural changes occur in the muscles—*structural* rigidity. The onset of late rigidity can be deferred, and its severity mitigated by appropriate Faradisation of the extensor and abductor muscles. In hemiplegia from syphilitic thrombosis, late rigidity is usually very marked ; and such a paralysis in a young man, if embolism is excluded, is almost invariably due to syphilitic thrombosis.

Various trophic and vaso-motor changes may be present in hemiplegia. Elevation of the temperature of the paralysed limb may be observed within the first few days ; œdema, blebs, acute bed sores, and occasionally joint lesions, may be present. These phenomena probably depend on the irritative character of the cerebral lesion.

The leg usually recovers first, but the extensors of the toes are especially affected, the feet being dropped, and the patient having difficulty in lifting the toes off the ground, scraping the floor as he walks. Cerebral paralysis may be partial, the arm, leg, face, or tongue being separately paralysed, the lesion being usually situated in the cortical centres. The arrangement of these centres in the cortex explains the grouping of the paralyses ; why, for instance, the tongue and arm cannot be affected by a single lesion without the face. In these cases the

paralysis is usually not absolute, and the extremities of the limbs are especially affected. Cortical lesions too, are liable to be attended with partial epilepsy.

A cerebral monoplegia may be due, not only to a lesion in the cortex, but occasionally, though rarely, to a lesion in the internal capsule, as I have already mentioned.

Various disorders of movement may appear as the paralysed limbs recover or mend, either tremor, choreiform movements, or slow continual spasm (athetosis).

Athetosis (*αθετος* without position or place), first described by Hammond, is in the great majority of cases a sequel of hemiplegia, and especially of hemiplegia occurring in early life and due to softening. It may, however, be bilateral and independent of hemiplegia.

Spastic hemiplegia of infancy is due to injury sustained by the motor areas of the brain during birth in difficult and tedious labours. It may, however, be due to thrombosis following specific fevers in early life. There is frequently in these cases idiocy or imbecility, and often epilepsy. The bones are shortened and the limbs atrophied. This disease is at once distinguished from paralysis due to polio-myelitis anterior acuta :—

- 1.—By the history of its presence from birth.
- 2.—By the presence of rigidity.
- 3.—By the increase of reflexes.

This hemiplegia may be bilateral and the child's four extremities rigid and paretic. The trunk

muscles being affected the child is unable even to sit up. I recently had a well-marked case at the Queen's Hospital of *bilateral spastic hemiplegia*, in which the patient was imbecile and unable to stand or sit up. These cases of bilateral spastic hemiplegia are somewhat confusing, but the history of the illness existing from birth, together with the presence of rigidity of the limbs and exaggeration of the deep reflexes, and generally also some degree of imbecility, enable us to make the diagnosis. *Spastic paraplegia* may be congenital and due to injury to the leg centres or to the spinal cord at the time of birth.

These conditions are frequently met with at workhouse infirmaries and at children's hospitals.

Cases of Spastic Hemiplegia.

CASE I.—Josiah Withers, aged 24, was admitted to the workhouse infirmary in April, 1875. He has one sister and one brother, who are in perfect health. His father says the paralysis came on after convulsions when a few months old. He is decidedly an imbecile, but can understand perfectly what is said to him, and can look after himself to the extent of eating and drinking, but little more. He has spastic hemiplegia on the right side. The right side of the face is slightly contracted, the angle of the mouth being elevated and the naso-labial furrow increased. The eyelids on the right side are more separated than on the left, so that the right eye seems to be slightly larger than the left. He cannot close this

eye completely. The right side of the tongue is not atrophied, and the movements of this organ are perfect. There is much wasting and rigidity of the right upper and lower extremities. The arm is abducted instead of being adducted as is usually the



FIG. II.

SPASTIC HEMIPLEGIA OF INFANCY.

case. The forearm is flexed and pronated, and the tendon of the biceps stands out prominently. The wrist is preternaturally extended. The fingers are flexed strongly over the thumb, which is adducted across the palm. The fingers are flexed at all

three joints, though usually in this disease the metacarpo-phalangeal joints are extended, the middle and distal joints flexed. He walks with the thigh and knee much flexed, but he can straighten them; the rigidity is more marked in the upper extremity than in the lower, and it increases towards the distal end of each extremity; thus the wrist and fingers are more rigid than the elbow and shoulder. Also the ankle is far more fixed than the knee or hip.

There is a condition of extreme talipes equinovarus, with partial dislocation of the astragalus, the patient walking on the balls of his toes, and being obliged to bend his thigh and knee to a considerable extent. There is a well-marked rotation inwards of the limb in progression, due chiefly to the gluteus medius. There is marked atrophy both of the muscles and bones on the right side. The bones are diminished both in circumference and in length. The circumference of the right arm is $7\frac{1}{2}$ ins.; of left, $8\frac{3}{4}$ ins.; of right elbow joint, 8ins.; of left, 9ins.; Around the centre of the forearm the measurement is, on the right side 5ins.; on the left, $6\frac{1}{2}$ ins. Circumference of centre of thigh on the right side is $13\frac{1}{2}$ ins.; of left, 16ins. Around centre of calf, on the right side, 8ins.; on the left side, $10\frac{1}{4}$ ins. The right collar bone is $5\frac{1}{2}$ ins. in length; the left, 6ins. Right humerus 10ins.; the left, $10\frac{1}{2}$ ins. The ulna on the right side is 8ins. long, on the left is $9\frac{1}{4}$ ins. The metacarpal bones of the right hand measure $2\frac{1}{2}$ ins.; of the left, $2\frac{3}{4}$ ins. There is also slight

shortening of the phalanges of the right hand. There is no appreciable shortening of the bones of the lower extremity. The reflexes, both superficial and deep, are much exaggerated. The olecranon reflex is much increased on the right side. The patellar reflex is much exaggerated, and there is a well marked front-tap contraction on the right side. The plantar, cremasteric, abdominal, and epigastric reflexes are all present. Ankle clonus cannot be obtained, from the great rigidity of the ankle joints. Sensation is perfect.

The patient, apart from his paralysis and imbecility, is in very good health. The atrophy is mainly due to disuse, partly also, most probably, from extension of degeneration of the pyramidal tracts to the multipolar nerve cells in the anterior cornua. The latter, however, are not much affected, or the rigidity and increase of the reflexes would not be present. No asymmetry of the skull can be made out, but the forehead is low and receding. The patient suffers from the severe form of epilepsy, and is very passionate at times.

CASE 2.—*Spastic hemiplegia, with epilepsy and imbecility, and with choreiform movements of the hand (athetosis).*—Patrick Lawley, aged 38. The patient states that his disease dates from early childhood. He has three brothers and two sisters in good health. His father and mother are both dead; he does not know anything of the cause of their death. He says he was always told that he was

paralysed when a baby. His brother tells me that he was paralysed after convulsions, when a few months old. There is spastic hemiplegia of the right side. The right side of the face appears smaller than the left, with the naso-labial fold more marked. The tongue when protruded is projected to the right. There is no shortening of the clavicle or humerus on the right side ; but the right ulna is $\frac{1}{2}$ in. shorter than its fellow. The metacarpal bones are slightly shorter than those of the left hand. The measurement around the centre of the right arm is $8\frac{1}{2}$ ins., on the left side $9\frac{1}{2}$ ins. The movements of the shoulder are a little stiff, but there is no marked rigidity ; the elbow is in the same condition. There is preternatural flexion of the right wrist, adduction of metacarpal bone of thumb, with extension of its phalanges. The voluntary movements of the fingers and thumb are slight, and the grasp of this hand is very feeble. There is a slow continuous rhythmical movement of the fingers and thumb, consisting of alternate flexions and extensions, the fingers also being separated from each other and then approximated ; he cannot control these movements. There is evident vasomotor disturbance on the right side of the body, for on drawing the finger nail along the skin a blush appears far more quickly and lasts much longer than on the left side. The hand is also very cold compared with the opposite one, and is livid. The right hand is decidedly atrophied, but the muscles

of the palm are hypertrophied from the continual motion. The olecranon reflex is much better marked on the right side than on the left. The right lower extremity is much weaker than the left. The bones do not seem to be shortened, although there is decided muscular atrophy. He can walk on the sole of the right foot, but there is a little talipes equino-varus. The patellar reflex is greatly exaggerated, but ankle clonus is obtained with difficulty owing to the rigidity of the muscles. The plantar reflex is exaggerated, and the cremasteric is present. The epigastric and abdominal cannot be obtained. The muscles respond pretty normally to Faradisation and Galvanisation, the alteration being only a slight diminution of response without qualitative changes. The *ophthalmoscope* shows nothing abnormal.

The patient is an epileptic, with imbecility, and is very passionate at times when irritated. Localisation of sensation in the affected hand is much impaired; although the patient can tell when he is touched, he cannot properly localise the point of contact.

CASE 3.—*Spastic hemiplegia of the left side, with imbecility and epilepsy, and choreiform movements of left hand.*—M. S., aged 34, a female, has been paralysed on her left side from birth. She suffers from the severe form of epilepsy, which she says she was frightened into when young. There is a great degree of imbecility, and the patient

has the characteristic expression of imbecility, with a low receding forehead. The left side of face is much smaller than the right, and she cannot close the left eye as well as the right, but there is not much rigidity. There is marked atrophy of the muscles of the left upper extremity, except of those of the hand. There is only slight shortening of the bones of the forearm. The left hand is much smaller than the right, although the muscles are hypertrophied. There is hyperextension of the middle-phalangeal joints, and there is a slow continuous movement of the fingers and hand. This movement is very similar to that observed in the case of Patrick Lawley, but is not so pronounced. The movement only comes on at times, and when it does she has no power of controlling it. The hand is cold and livid, and the grasp very feeble. There is slight rigidity of this extremity, and increase of olecranon reflex. Sensation is normal. The muscles of the left lower extremity are also much atrophied, but I cannot make out any shortening of the bones. The patellar reflex on this side is much exaggerated. There is no front-tap contraction, but ankle clonus is well marked, and the plantar reflex exaggerated. There is slight talipes equino-varus. Stiffness is present to a slight degree, and is exaggerated at times. There is diminished vaso-motor tone on the left side; on drawing the finger nail along the surface a blush quickly appears, and lasts some time. The electrical reactions are normal.

CASE 4.—*Hemiplegia of the right side, with epilepsy and imbecility, and peculiar movements of the hand.*—Ed. Webster, aged 16. His father, a very intelligent man, gives me the following history:—His son was a fine child till he reached the age of sixteen months; about this time he was seized with convulsions from teething. The fits were very severe and affected the right side especially. After the convulsions ceased the child was found to be paralysed on the right side of the face, and in the right arm and leg. And from being a bright boy he became, as his father expresses it, “a baby again,” and had to be carried about, and was dull and stupid. He was not able to speak for several months after the convulsions, although well able to before, and had to be taught over again. Epilepsy followed the fits, and he now suffers from the severe form. He is imbecile, but not to a great degree. His face looks intelligent and his memory is good, and he answers questions perfectly coherently; but his schoolfellows call him “silly Teddy.” Two years ago he took four or five sheep from a field, drove them to the Smithfield market, and sold them for half-a-crown; he was “let off” on account of his mental defect.

He has right hemiplegia, and there is marked atrophy. There is talipes equino-varus. The rigidity is not so marked as in the three previous cases, but it is present. There is increase of olecranon and patellar reflexes. No alteration of sensation. The right side of the face is atrophied as in the previous

cases, and the tongue points to the right side when protruded. He has almost entirely lost power in his right hand; slow movements are observed at times, but they are not so marked as in the case of Patrick Lawley. There is a remarkable illustration of the bilateral association of the movements of the extremities in this case. If the boy opens his left hand and straightens his fingers the right hand is observed to slowly open and the fingers to straighten, and if he closes his left hand the right follows suit. He has no power at all over the right hand by itself, but can control it through the medium of the left. His father says he observed this himself; in fact, he drew my attention to it. The recovery of speech, after what must have been a severe injury, is explained by the youth of the patient. The right side of the brain having been educated and taken on itself the duties of the left side. Apart from his epilepsy, the patient's general health is excellent. His father tells me that he is bad tempered and ready to fight on the most trivial provocation. He frightens the children by the expression of his face when angry.

In the diagnosis of the nature of a hemiplegia, as in the diagnosis of all varieties of paralysis, the first question to be answered is:—Is the paralysis functional, or is it due to organic lesion?

Hemiplegia may be present after epileptic attacks, or it may precede or follow chorea, or be hysterical or simulated.

In hysterical hemiplegia (1) there is nearly always loss or impairment of sensation, and the motor loss is rarely complete. Hemianæsthesia with implication of the special senses on the same side, there being amblyopia of the eye on the affected and slight visual impairment on the opposite side, is almost invariably due to hysteria.

(2) The face and tongue are not affected, and the leg frequently suffers more than the arm.

(3) Hysterical anæsthesia and paralysis are apt to vary and appear or disappear from trivial causes, as by the application of metals to the skin (Burq's treatment).

(4) In hysterical paralysis the sphincters are unaffected, and no trophic lesions or bed sores occur.

Having decided that the hemiplegia is due to organic causes, is it cerebral, spinal, or peripheral? Of course, in the vast majority of cases it is cerebral. A lesion of the brachial, lumbar, and sacral plexuses on one side would cause paralysis of the arm and leg, but there would be electrical changes proving damage to the peripheral nerves; also sensory disturbances with rapid wasting and trophic changes.

In spinal hemiplegia due to polio-myelitis anterior acuta, the electrical changes would at once show that the lesion was not cerebral. A tumour high up pressing on the spinal cord might cause a hemiplegia, but our attention would be arrested by the absence of implication of the face and tongue, and by sensation being blunted on the opposite side, not on the same side as the motor paralysis.

Having decided that the hemiplegia is cerebral, what is the exact situation of the lesion? The presumption is that it is in the internal capsule, this being the most common seat of the lesion in hemiplegia of the ordinary type. The presence of partial epilepsy would indicate that the lesion was cortical. If the third or facial nerves were affected on the opposite side we should localise the disease in the crus or pons.

Vomiting.—Vomiting is a reflex act, the centre being placed in the medulla and afferent impressions being conveyed by various nerves, the chief of which is the vagus. Undue irritation of the terminations of this nerve in the stomach is the cause of the vomiting in gastric ulcer and other diseases of the stomach. Irritation of the centre in the medulla may cause vomiting, the centre being affected either by the quality of the blood or by the presence of growths or inflammatory products. The vomiting of anæmia is caused by the action of the impure blood upon the centre.

Vomiting may be caused by psychical impressions, as is well known, and also by disease in any part of the encephalon, but is especially common when the disease is in or near the medulla.

Tumours of the cerebellum are very constantly attended with vomiting at some stage.

In hysteria the irritability of the vomiting centre is exalted and the reflex act is too readily brought about, food being at once rejected. This

increased irritability is present in cases of cerebral disease, and vomiting may occur independently of the ingestion of food. But it must be remembered that in cerebral disease vomiting frequently follows the taking of food, and so may simulate vomiting due to stomach mischief.

Cerebral vomiting is usually unattended with nausea, which is commonly present when the vomiting is due to stomach mischief. In cerebral disease we should probably have other symptoms pointing to brain disturbance, such as headache or vertigo, but these might be caused also by stomachic disorders.

Cerebral is usually much more intractable than stomachic vomiting, and in the latter a furred tongue and tenderness or discomfort at the epigastrium are usually present. Vomiting in children, frequently occurring, without a furred tongue or complaint of pain or discomfort at the stomach, should always excite suspicion of cerebral disease.

In all cases of obstinate vomiting examine the urine and the fundus oculi. The vomiting in cases of cerebral tumour may be frequently checked by counter-irritation by blisters or iodine at the nape of the neck or by a seton.

Temperature.—The temperature is frequently abnormal in cerebral disease. There is evidence which goes to prove that somewhere in the convolutions around the fissure of Rolando there is a heat-controlling centre, impressions from which pass through the crura, pons, and medulla to the cord.

Lesions in the pons are especially characterised by the remarkable variations which they may cause in the temperature. Probably four-fifths of cases of pontine disease, either acute or chronic, are attended with pyrexia. Lesions, also, below the pons in the medulla and cervical region of the cord may cause fever, which may amount to severe hyperpyrexia, the temperature rising even to 110° F. This hyperpyrexia is probably explained by loss of the controlling influence of the centre above mentioned over heat production. Towards the end of tubercular meningitis hyperpyrexia is occasionally observed.

In cerebral disease it must be remembered that fever may be absent in inflammation, that elsewhere would certainly raise the temperature, and also that the pulse rate may not be increased as it usually is with fever. For example, in an early stage of tubercular meningitis a slow pulse may be observed, with considerable fever; this is due to irritation of the vagi; the same phenomenon is observed to a less extent in enteric fever, probably also from irritation of the vagi, but of their peripheral, not of their proximal extremities. In enteric fever, however, though the pulse rate does not increase proportionately to the fever, yet the pulse is much more frequent than in health. The chronic degenerative diseases of the brain, *e.g.*, general paralytic dementia and multiple sclerosis, are apt to be attended with occasional pyrexia, which is not caused by any inflammatory

mischievous, but is probably due to interference with the nervous heat-controlling mechanism.

At the onset of cerebral hæmorrhage, except when pontine, the temperature is depressed, but afterwards there is an elevation due to surrounding inflammatory changes.

Respiration.—Respiration is often disturbed in cerebral disease. Lesions in or near the respiratory centre, or large hæmorrhages elsewhere, may cause quickening, slowing, or irregularity in the breathing, or the peculiar rhythmical variation called the “Cheyne-Stokes’” breathing may be present, in which the pulse is often quick during the period of apnœa, and slow during the period of dyspnœa. “Cheyne-Stokes’” breathing is a sign of great gravity, and is in the great majority of cases a forerunner of death. I have seen recovery where this modification of the breathing had been present both in uræmic and cardiac cases. A man was recently in the Queen’s Hospital suffering from aortic regurgitation and dyspnœa, the respiration being of the “Cheyne-Stokes’” type. His breathing became normal as his condition improved, and when he was discharged all respiratory trouble had disappeared. He died suddenly, however, a few weeks after. I have observed “Cheyne-Stokes’” breathing in cases of cerebral hæmorrhage, tumour, and tubercular meningitis. In various forms of heart disease and in uræmia it is frequently met with.

Coma.—Coma is a symptom frequently present in coarse brain disease. In coma the loss of function is from the highest to the lowest, the mental processes being first affected, the automatic and purely reflex centres continuing in action and being lost late. There is loss of power over the sphincters.

Stupor is incomplete coma, in which the reflex actions and the power of swallowing are preserved, and the patient may put out his tongue when told to, but is unable to carry on any conversation.

In deep coma the pupils may be widely dilated and immobile. Loss of the conjunctival reflex, or loss of the power of swallowing and the presence of stertor, are of grave omen. Any sudden lesion of the brain may occasion coma, but hæmorrhage is by far the most common cause; hence the term “apoplexy” is generally used to indicate hæmorrhage, although this condition, which strictly means the sudden onset of unconsciousness, is much more common in epilepsy, and rarely present in cerebral hæmorrhage. In the diagnosis of cerebral hæmorrhage as the cause of coma we must carefully examine for any paralytic symptoms, such as dilatation of one pupil, conjugate deviation of the eyes, alteration of the reflexes on one side of the body. The pulse is generally slow and full and the breathing slow, occasionally of the “Cheyne-Stokes’” type; this form of respiration and irregularity of the pulse are of fatal omen in cerebral hæmorrhage.

In the diagnosis of coma we shall have to bear in mind all the other causes of this condition, such as uræmia, diabetes, alcohol, opium, epilepsy, and hysteria. In all doubtful cases we ought to draw off and examine the urine, and use the stomach pump. The presence of albumen might lead you to consider the case as one of uræmia, while it really might be one of cerebral hæmorrhage; but uræmic coma is rarely complete, and is usually characterised by great restlessness. Hemiplegia may occur in uræmia, and, coinciding with coma, may exactly simulate cerebral hæmorrhage. Chauternes and Tenneson in the "*Revue de Médecine*," November, 1885, relate cases of partial epilepsy and hemiplegia in Bright's disease.

I recently had under my care in the workhouse infirmary a man, 40 years of age, with cardiac hypertrophy, a pulse of high tension, and with slight albuminuria. The albumen was not present constantly, but was often absent for weeks. He was admitted in a semi-comatose condition, with right hemiplegia, and after being bled he quickly recovered consciousness, the hemiplegia also quickly passing off. After his admission he had five similar attacks, which were always preceded by mental symptoms, delirium, and restlessness, and by violent convulsion of the right side of the face, right arm and leg, and conjugate deviation of the eyes to the right. Now, these attacks could not be due to hæmorrhage, for recovery was complete within a few hours of his

being bled, and they are best explained by the supposition that they were due to local œdema of the brain, as has been proved by Chauternes and Tenneson in their own cases.

In uræmia, convulsions usually precede coma, and recur frequently during its continuance. At the onset of apoplexy a convulsion may occur, but is usually single and not repeated. In uræmia the temperature is always depressed; in apoplexy the temperature falls at first, but there is usually some increase later on. Elevation of temperature would negative uræmia. The diagnosis is of the highest importance, since the treatment for uræmia is one of active interference, while the treatment for apoplexy is one of non-intervention.

In coma, as I have previously stated, the eyeballs are divergent; this I have found a constant symptom, and one that might be useful in detecting cases of hysteria and malingering.

Headache.—Headache is a symptom produced by many causes apart from organic disease of the brain. The pain of organic brain disease is usually very severe, constant, and diffused.

In migraine and hysterical headache the pain may often be, as the patients say, covered with a finger. Headache should always attract our attention to the eyes, errors of refraction being a common cause, especially hypermetropia and astigmatism. In obstinate cases always examine the urine for albumen, and the fundus oculi for optic neuritis or retinal changes.

The following forms of headache may be distinguished :—

1.—The anæmic headache; relieved by the recumbent, exaggerated by the upright posture.

2.—The congestive headache; aggravated by the recumbent posture.

3.—Clavus, or hysterical headache; the pain being frequently limited to one spot, so that it can be covered by the finger.

4.—The toxic headache; caused by alcohol, tobacco, uræmia, &c.; may be frontal or occipital.

5.—The febrile headache, occurring in typhoid and other fevers, usually frontal.

6.—The rheumatic headache, accompanied with tenderness of the scalp.

7.—The gouty headache.

8.—The dyspeptic headache, which is generally frontal.

9.—The syphilitic headache, which is very severe, especially at night, often causing transient mania at this time.

10.—The headache of organic brain disease, which is usually very severe, diffuse and constant.

11.—Hemicrania, migraine, periodical, or sick headache. In the attacks, of which we can distinguish three stages, in the first stage ocular disturbances occur, such as hemianopia and scintillating scotoma; occasionally also other sensory and motor affections are present, such as deafness, aphasia, transitory paresis of one limb or of some ocular

muscle ; the second stage of the attack is marked by headache, which is gradual in its onset, very severe and localised, and followed by the final stage of nausea and vomiting, after which the patient rapidly recovers. In the intervals, the subjects of this neurosis are usually quite free from pain and perfectly well.

Vertigo, or Giddiness.—In this condition the patient feels uncertain as to his position with regard to surrounding objects, sometimes seeming to move himself (active vertigo), sometimes feeling as if objects were moving round him (passive vertigo). Vertigo is constantly associated with impairment of equilibration, the patient usually falling or staggering in the direction in which there is a subjective sense of falling. The power of equilibration is guided and depends upon impressions derived from the periphery that give information as to the relation of the body to surrounding objects. The senses which subserve equilibration are touch, sight, the muscular sense, together with special organs, the semi-circular canals of the internal ear. Sight is especially important in guiding equilibration, a patient suffering from locomotor ataxy in an advanced stage being quite helpless when his eyes are shut. Recent ocular paralysis is attended with severe vertigo.

Flourens first discovered the function of the semi-circular canals, and showed that animals lost their power of equilibration after section of these organs. More recently Goltz and Cyon have

shown that it is the pressure of the endolymph within these canals that gives rise to sensory impressions which inform us as to the position of our heads in space. If any of these sources, namely, the ocular muscles and vision, the semi-circular canals, &c., from which we derive our information as to the position of our body in space, be disordered, vertigo results.

Before we conclude that vertigo is due to organic brain disease, we must exclude ocular defects and disease of the middle and external ear; we must also exclude stomach disorder, the pneumogastric nerve being intimately concerned with equilibration. Vertigo is common in uræmia and in disturbances of the cerebral circulation, and is frequent in minor epilepsy, sometimes constituting the entire attack.

Menière's disease, or labyrinthine vertigo, is characterised by three symptoms, viz.:—(1) Vertigo, which is severe and paroxysmal; (2) tinnitus, and (3) deafness; but it is important to remember that all these symptoms may be caused by accumulations in the external meatus. The symptoms of Menière's disease may be caused by a blow on the head, being probably due to hæmorrhage into the semi-circular canals. I had such a case recently in the Queen's Hospital. It is important also in all cases of vertigo to examine the nasal cavities for polypus or other disease. In locomotor ataxy, vertigo may be present, but usually only when there is paralysis of the ocular muscles.

Vertigo arises from a great variety of intracranial conditions, but it is most constantly associated with cerebellar disease, especially with disease of the middle peduncle of this body. In disease of the pons, of the corpora quadrigemina, or of the crura cerebri, vertigo is often present. It is a common symptom in cerebral anæmia and congestion. I find that vertigo is very common in old people, and in those affected with it the arteries are always markedly degenerated. The bromides and hydrobromic acid relieve such cases. Dr. Handfield Jones has recommended the bichloride of mercury in small doses for senile vertigo. Vertigo may be produced by psychical causes, and also from toxic causes, such as tobacco and alcohol.

A curious form of vertigo, called agoraphobia, is sometimes met with, the subjects of this affection experiencing giddiness whenever they attempt to cross a wide market place or a road, while they can walk quite well in a narrow passage, or when they keep on the footpath. Westphal described this variety of vertigo in 1872. I have met with it in myxœdema in one instance, and have known it to occur after childbirth where there had been considerable loss, the patient being seized with intense vertigo, and falling whenever she attempted to go out of doors, but being quite well while indoors.

Delirium.—Delirium, a perversion and excess of mental activity, in contrast with stupor in which there is diminution of mental activity, is frequently observed

in all conditions which cause defective nutrition of the brain. It is essentially the same as insanity, but the term delirium is used when the mental condition is evidently secondary to and depends upon some organic disease of the brain, or on some morbid state of the blood. We rarely get fixed delusions, and the patient is usually more incoherent than in insanity. Pyrexia is a far more common cause of delirium than organic brain disease. In all cases of delirium, then, take the temperature, and if there is fever carefully search the chest and abdomen for signs of disease.

It is well known that people addicted to alcohol are rendered delirious by slight causes. Pneumonia in drunkards is nearly always accompanied with early delirium, and occurs frequently without fever.

Hallucinations, illusions, and delusions are varieties of perverted consciousness. We may have hallucinations of hearing, of vision, of taste and touch, the patient hearing voices, or seeing things which have no objective existence whatever, in fact hallucinations are entirely subjective. Hallucinations of hearing are very dangerous, the patient hearing his neighbours abusing him is apt to resort to violence. Illusions are incorrect interpretations of objects which do exist, a patient mistaking the nurse for his wife, for example. Hallucinations and illusions, then, refer to the special senses; delusions are insane ideas of any kind.

Mental debility is markedly present in many acute diseases, as well as in softening and various degenerative diseases of the brain and after apoplexies.

The faculty of attention may be impaired, and memory may be lost; loss of memory for recent events is especially characteristic of general paralytic dementia.

Apoplectiform attacks occasionally occur in the course of the chronic degenerative diseases of the brain, namely, in general paralysis of the insane, and in multiple sclerosis. There is usually considerable fever during these attacks. They come on suddenly, and may last for days; weakness of one side of the body may remain for a few days after the attack, soon, however, passing off. Aphasia may also be present. The cause of these attacks is not known, but in all probability they are due to local œdema of the brain, similar to what Chauternes and Tenneson have described as the cause of uræmic epilepsy and hemiplegia.

Convulsions.—These spasmodic movements are frequent in diseases of the brain; they are supposed to depend upon an abnormal discharge from unstable grey matter.

Convulsions may be partial or general. In children, the common cause is reflex irritation, or the circulation of unhealthy blood through the brain, as in uræmia, or at the commencement of fevers. The prognosis is far more favourable in children than in adults. Convulsions in children

may be due to irritating lesions of the brain, such as tubercular meningitis, or to increase of the intracranial pressure, as in chronic hydrocephalus. Sudden lesions of the brain, such as hæmorrhage or softening, are apt to be attended with convulsions. General convulsions have no localising value. The partial or local convulsions indicate disease in or near the motor centres of the cortex. When organic brain disease is present a trivial cause may excite convulsions. Irritating and progressive lesions anywhere in the brain, but especially when in or near the cortex, may cause convulsions. Stationary lesions only cause convulsions when in or close to the cortex.

In all cases of convulsion we should carefully look for symptoms of organic brain disease and examine the fundus oculi, and also exclude uræmia, before concluding that the case is one of epilepsy or due to reflex irritation.

Deafness may be caused by disease of the auditory centres in the superior temporo-sphenoidal convolutions, but this is rare; more commonly it is due to lesions of the auditory nerve itself beyond its superficial origin. By means of the tuning fork we can generally distinguish between deafness due to lesions of the nerve and that due to mischief in the conducting apparatus.

In all cases of deafness or of tinnitus the external auditory meatus and the Eustachian tube should be examined. Deafness from disease of the

nerves of hearing is often observed in the subjects of inherited syphilis.

Occasionally hearing is lost in some people during periods of mental excitement. Deafness of nervous origin is observed in the course of and after specific fevers, as typhus, typhoid, and mumps.

Tremor.—The division of the various forms of paralysis into the atrophic and spasmodic varieties is an important one. In paralysis from cerebral lesions and in that due to lesions of the lateral columns of the cord, spasm is a prominent feature sooner or later. In paralysis due to lesions of the anterior cornua of the cord, or to lesions of the peripheral nerves, flaccidity with wasting is present. Tremor is the mildest form of clonic spasm; it consists of slight contractions of particular muscles or groups of muscles, causing a rhythmical oscillation of the limbs and trunk.

Tremor is observed in various pathological conditions, as for instance:—In (1) alcoholism, (2) debility, (3) mercurialism, (4) plumbism, (5) hysteria, (6) multiple sclerosis, (7) paralysis agitans, (8) senility, (9) post-hemiplegia, (10) in all cases in which the pyramidal tracts are the seat of disease or are compressed. A tumour anywhere in the region of the pyramidal tracts either in the brain or cord may cause tremor, so that this symptom is one of considerable localising value. (11) Tremor of the facial muscles and tongue is a constant feature of general paralytic dementia. The tremor of multiple

sclerosis is coarser than that of paralysis agitans and affects the head and trunk, the body during walking being frequently lunched to one side, while the head is held stiffly. The tremor in the former disease, moreover, ceases when the muscles are at rest, being present only during exertion.

Fibrillary Contractions consist of alternate contraction and relaxation of individual bundles of muscular fibres visible under the skin. These are usually present in progressive muscular atrophy, in which disease they are readily evoked by filliping the skin.

Fibrillary contractions are, however, by no means confined to this disease, but they are readily evoked in all wasting diseases, the irritability of the muscles in such diseases being exalted. In phthisis and in the typhoid state they are constantly present.

Cramp is a form of tonic spasm, causing painful contractions of a muscle or group of muscles. It most commonly affects the calf muscles after excessive exertion. The cramps of diarrhœa, cholera, and tetanus are well known; severe cramps are also present in extra-medullary growths, implicating the nerve roots. I have such a case in which the cramps can only be relieved by large and frequent hypodermic injections of morphia.

Contracture.—By this term is meant any persistent shortening of a muscle or group of muscles, occasioning persistent deformity.

Contracture may be due :—

1.—To changes in the muscles themselves—myopathic contracture.

2.—To paralysis of a group of muscles, their opponents contracting, and ultimately causing contracture—paralytic contracture.

3.—To spasm of a group of muscles, their opponents not being paralysed, but being overcome.

4.—To hysteria.

Whenever the origin and insertion of a muscle remain approximated for a considerable time the muscles become permanently shortened, causing contracture. The contracture accompanying the late rigidity of hemiplegia is well known. In hysteria, contracture may accompany paralysis, or may exist by itself, affecting any limb. These hysterical contractures may last for years, and then suddenly cease.

I have met with several cases of hysterical contracture, and have found them most difficult cases to cure.

In all cases a careful examination must be made, in order that no mistake as to its hysterical nature occurs.

Affections of Speech.—Aphasia.

Mental processes may be expressed (1) by speech, (2) by writing, and (3) by gesture.

The expression of mental processes by gesture is the most simple and elementary, and is the first

to be acquired by the child; but it has little value in expressing propositions, being more especially used in expressing the emotions.

The faculty of speech is not innate, but is acquired with difficulty and after much trouble.

A child understands what is said to him long before he can speak, the sensory processes being developed earlier than the motor.

Language possesses a subjective or sensory aspect, and an objective or motor aspect.

Impressions derived from the eye, from the ear, and the other senses; but more especially from the ear, are essential to the acquirement of speech.

If a child either before or shortly after it has learned to speak becomes deaf, it becomes mute also. Complete loss of speech, however, from defect in the peripheral sensory organs, is rare, for a deaf mute may be taught to speak by gesture.

The impressions derived from the organs of special sense are reduced to order in the cortex.

The following diagrammatic scheme (Fig. 12, page 86), used by Dr. Grainger Stewart, will help you to understand the main facts to be known about aphasia.

The seeing centre is situated in the occipital convolutions, and possibly in the angular gyri also.

The word-seeing centre is situated in these convolutions on the left side of the brain. A lesion, softening or tumour, of these parts causes *word-blindness*, the patient being unable to read a

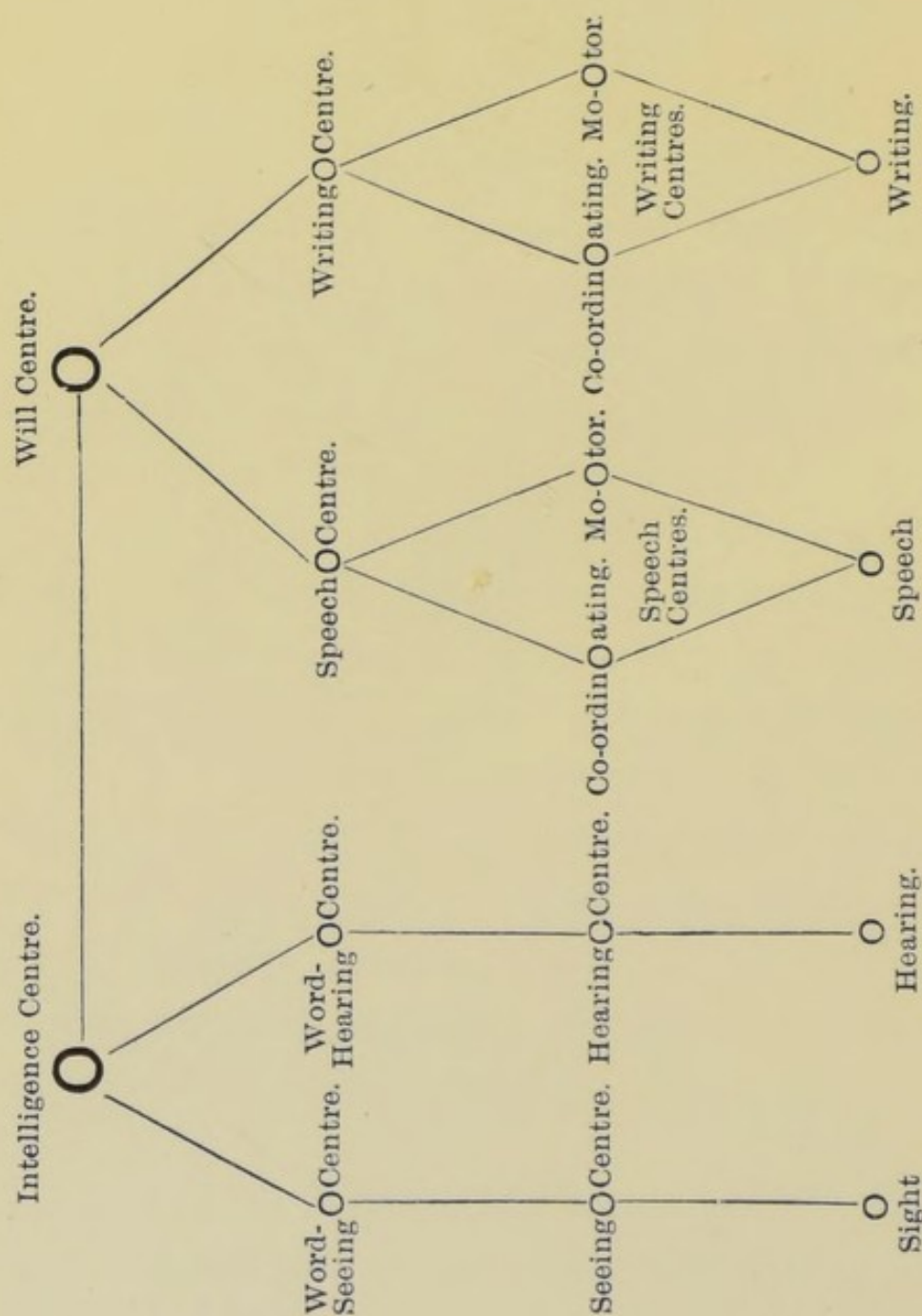


FIG. 12.

letter, or to name any object he sees; though he may be able to write and talk, but usually incorrectly.

The hearing centre is situated in the superior-temporo-sphenoidal convolutions, and the word-hearing centre in the same convolution on the left

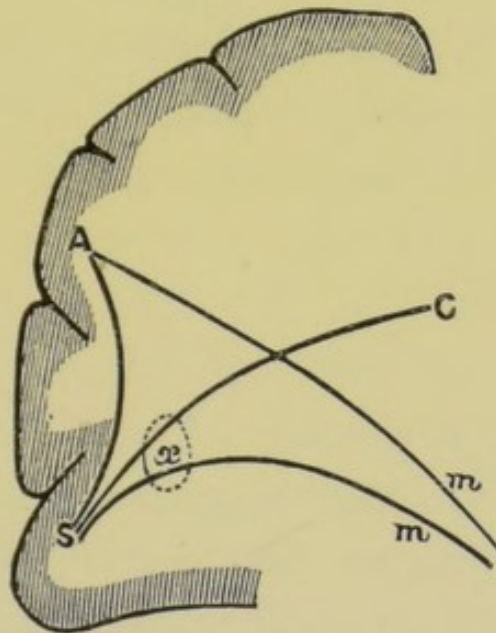


FIG. 13.

DIAGRAM OF PROBABLE COURSE OF FIBRES FROM MOTOR
SPEECH-CENTRE (GOWERS).

A., hand centre; A. *m.*, fibres from this to internal capsule; S., motor speech-centre; S. C., fibres from this to corpus callosum; and S. *m.*, to the internal capsule; S. A., fibres from speech-centre to hand centre. A lesion at *m. m.* causes transient aphasia only; the speech processes being able to pass by S. C. A lesion at *x* causes permanent aphasia, since it involves both the fibres to the corpus callosum and internal capsule, but would not abolish expression by writing, the fibres S. A. (connecting the speech and hand-centres) escaping.

side. A lesion in the superior-temporo-sphenoidal convolution on the left side causes *word-deafness*, the patient being unable to understand anything that is said to him, although he is not deaf to sounds.

The posterior portion of the third frontal convolution and the lower part of the ascending frontal convolution contain the centres for the muscles concerned in articulation. Motor processes for words leave the cortex in this region on the left side. The left side of the brain in right-handed people, and the right side in left-handed people being chiefly concerned with voluntary speech.

According to Hughlings Jackson the right hemisphere is concerned only with emotional and automatic utterances.

A lesion of the operculum causes motor aphasia, the patient being able to understand what is said to him and to read, but being unable to speak or to write. When the lesion is in the sensory part of the cortex, there being word-deafness and word-blindness, the aphasia is called *sensory*. Disease of the convolutions of the operculum, or immediately beneath them, causes permanent *motor* aphasia. But if the lesion is some distance below the cortex the aphasia may pass off, the left convolutions being then able to communicate with the nerves of articulation through the right hemisphere by the corpus callosum. If, however, a lesion damages the fibres of the corpus callosum which connect the third frontal convolutions of the two sides; as well as those which connect the third left frontal convolution with the internal capsule; then permanent aphasia, similar to that produced by destruction of the third left frontal convolution, is produced.

Occasionally in motor aphasia, a patient may be able to express himself by writing, though he cannot do so in words (aphemia). In this case the lesion has not destroyed the speech centre, or the fibres from it to the hand centre, but has interrupted the connection between the speech centre and the motor tract, and its connection with the opposite frontal convolution. Loss of memory of words (verbal amnesia), there being great difficulty in naming objects, is always present where there is word-deafness or word-blindness, and is much more frequent in sensory than in motor aphasia. Intelligence is nearly always affected in sensory, less commonly so in motor aphasia.

The term "dysarthria" is used when the defect in speech is due to defects of articulation, and must be carefully distinguished from true aphasia.

Cases of Aphasia.

CASE I.—*Sensory Aphasia due to Embolism (Word-blindness and Word-deafness).*—R. S., aged 19, a stamper, was admitted on September 26th, 1885, suffering from what was said to be "brain fever." Six years previously he had suffered from rheumatic fever, but had since remained well up to September 21st, except for occasional attacks of severe pain in the chest (angina). On this date, while at work, he was suddenly affected with pain in the head, and it was noticed that he talked nonsense. He was taken home, but his friends could not understand

him, neither could he understand them. He put up his hand to the left side of his head, and made signs of being in pain there. On examination, double aortic and mitral systolic *bruits* with considerable cardiac hypertrophy were found. The second sound was replaced by a diastolic bruit. Capillary pulsation was observed. The patient did not answer questions, but continually put his hand to the left side of his head. Verbal deafness was complete; when told to hold up his arm he put out his tongue, and answered questions by saying, "I can't hardly think of it, hardly," which he frequently repeated. He talked a great deal, but used wrong words, and little sense could be made out of what he said. At the same time he acted in a perfectly rational way, was quiet, and understood signs to take his food. He understood nothing printed but figures and letters; figures he easily recognised and could add them together correctly (the knowledge of figures being greatly automatic). He could spell words aloud, but evidently did not understand their meaning. He could write his name, but nothing else; could copy from a book, but did not understand what he wrote; could not copy from dictation. He had great difficulty in naming objects and always described them. When showed an egg he could not name it, but pointed to the fire and said "it takes about two minutes." He assented when the proper name was mentioned to him, but could not repeat the name. When shewn a leaf, he said, "It grows;" "Will do for a Sunday;"

and similarly with many other objects. He signed an I.O.U. for £100, and allowed me to take it without showing in the slightest that he understood what he had done. The temperature was raised to 101° Fahr. (due to softening). There was no hemiplegia, but slight paresis in the buccal region of the right side of the face. The case was a typical one of sensory aphasia, there being loss of understanding of spoken and of written language, loss of faculty of repeating words, of writing to dictation, and of reading aloud ; with preservation of power of writing, copying words, and of volitional speech. The lesion was in all probability in the area of distribution of the posterior terminal branches of the left middle cerebral artery, and due to embolism ; the convolutions affected being the supra-marginal or angular and the superior-temporo-sphenoidal. The patient improved daily, being able to name objects, which the day previously he had failed to name. It was noticed that when asked to name an object, he would look in a book till he came to the name in print and then would be able to say it. On October 29th the patient was much better. He understood all that was said to him ; could read short sentences, but failed to understand many words ; he was quite unable to copy from dictation, except monosyllables, and still suffered from considerable difficulty in naming objects. His mother informed me that before his illness he could read well and had taken prizes at his Board school.

CASE 2.—*Word-deafness*.—J. M., a man, aged 70, when seen in July, 1885, complained of tinnitus in the left ear, deafness, vertigo, and falling to the left side. He had had two slight attacks of hemiplegia on the right side, which had quickly passed off. There was no albuminuria, but the arteries were very extensively diseased. On October 15th he asked the nurse to let him see the doctor, as he did not feel well, and had pain in the left side of the head. When the resident medical officer saw him he found that the man could not answer any questions. I discovered that the patient suffered from complete word-deafness; he could not understand anything that was said to him, but spoke rationally and freely. He could read and understood what he read, and could copy from print, evidently understanding what he copied. He could hear when spoken to but could not understand. Verbal amnesia was also present; he could name none of the several articles shown him, but described them well. The next day the verbal deafness had passed off, and he understood what was said to him, and could name various articles. The case is interesting in connection with the symptoms of Menière's disease previously observed, and indicates that they were probably due to central mischief. The lesion was in all probability thrombosis of the branch of the left middle cerebral artery to the superior-temporo-sphenoidal convolution.

CHAPTER IV.

THE DIAGNOSIS OF THE SITUATION OF THE LESION IN DISEASES OF THE BRAIN.



In the diagnosis of the situation of the lesion in diseases of the encephalon, an intimate acquaintance with the anatomy and physiology of the brain is essential.

The brain is remarkably tolerant of disease; a large tumour or abscess may be present without symptoms, unless it be situated directly in or near the motor or sensory tracts.

In acute lesions, such as hæmorrhage and embolism, we must wait till indirect symptoms have passed off, before we can accurately localise the mischief.

The *præfrontal area* of the brain, that portion in front of the ascending frontal convolutions, is psychical in function; a lesion may exist here without causing any symptoms, but psychical changes are often present.

Lesions of the orbital surface of the frontal lobes are usually latent.

I recently had a man under my care with frontal headache and stupidity gradually deepening into coma, without any motor or sensory paralysis. Tenderness on percussion existed in one spot over the frontal bone, and the post-mortem examination revealed a gumma of the surface of the brain immediately under this spot.

In general paralysis of the insane the greatest changes are found in the præfrontal area.

A lesion of *the motor region of the cortex* causes motor paralysis, depending upon the centre affected. Convulsions are exceedingly common, the grey matter of the convulsions being less tolerant of disease than the white matter of the brain. Disease of one centre causes irritation in neighbouring centres, and convulsions in the limbs may result from disease near but not actually in the motor region.

Local paralysis is a more certain indication of the exact situation of the lesion than are convulsions, though usually, when convulsion attacks the whole of one side of the body, it commences in the limb in whose motor centre the lesion is situated. Irritative lesions of the motor area of the cortex are characterised then by unilateral convulsions or monospasms. The pathology of these spasmodic affections was first clearly recognised by Dr. Hughlings Jackson, who, from the study of these partial epilepsies, first suggested the existence of motor centres in the cortex ; hence the term "Jacksonian epilepsy."

There may be brachial, crural, facial, oculo-motor, or lingual convulsion. In the oculo-motor form there is conjugate deviation of the eyes and rotation of the head and neck.

These cases of Jacksonian or partial epilepsy are distinguished from ordinary epilepsy by the following features:—

1.—The convulsions are limited to one limb, or rather always begin in one limb, though they may ultimately spread all over the body.

2.—An aura is always present and is very distinct.

3.—Consciousness is either not lost or is lost late in the attack, after the convulsions have become general.

It is the local commencement of the convulsion that is especially characteristic. These partial epilepsies are of great localising value, they indicate organic disease in or near the motor region of the cortex, either softening or tumour. It is in these partial epilepsies that we can often check the progress of the attack by painting a ring of iodine or blistering fluid just above the extremity whence the aura starts.

For exact localisation of a tumour in the motor region of the cortex three factors are required:—

- 1.—Local persisting paralysis.
- 2.—Local epileptiform convulsions.
- 3.—Double optic neuritis.

It is only in this region of the brain that surgical interference with cerebral tumours is likely to prove successful.

Extensive disease of the outer surface of the hemisphere may cause hemianæsthesia, but this is never complete.

A lesion of *the angular gyrus* probably causes crossed amblyopia ; and if on the left side it causes word blindness also.

A lesion of *the occipital lobe* causes hemianopia towards the opposite side. Irritating lesions cause convulsions, which may commence with a visual aura referred to the opposite eye.

A lesion of *the superior-temporo-sphenoidal convolution* on the left side causes word-deafness, but there is not absolute deafness unless the convolutions on both sides are diseased.

A lesion of *the centrum ovale* causes symptoms like those brought about by disease of the corresponding portion of the cortex, except that Jacksonian epilepsy is not present, the symptoms varying with the seat of the lesion, and the fibres injured.

If aphasia is present, it is transitory unless the disease is immediately beneath the cortex, so that the fibres crossing by the corpus callosum from the third left frontal convolution to its fellow on the opposite side are interrupted.

A lesion of *the corpus striatum*, if the grey matter only is affected, causes no lasting symptoms. It is

only when the internal capsule is affected that hemiplegia is produced.

A lesion of *the optic thalamus* also causes no symptoms unless the capsule be affected, or the crus compressed. Cases of athetosis have been published where the lesion was located in the optic thalami.

In a child, who presented the usual symptoms of cerebral tumour, slight hemiplegia on the left side, with tremor of both upper limbs on effort was observed during life. At the autopsy caseous encapsuled masses were found in each optic thalamus; the paresis and tremor being due to pressure on the motor portions of the internal capsules.

A lesion of *the corpora quadrigemina* does not cause loss of sight, but loss of ocular movements, especially of the upward movement of the eyes and elevation of the lid. (Gowers.)

A lesion of *the pons* causes alternate or crossed paralysis. Since the facial nerves decussate about the centre of the pons, a lesion in the upper part of the pons will cause paralysis of the face and hemiplegia on the opposite side, but a lesion of the lower half will cause paralysis of the face on the same side and hemiplegia on the opposite side.

In tumours of the pons not only the facial but the fifth and sixth nerves may be paralysed on the side of the lesion, there being hemiplegia on the opposite side.

In hæmorrhage into the pons and other acute lesions of the upper portion of this body the pupils are often strongly contracted from irritation of the nuclei of the third nerves.

If the lesion be in the centre of the pons there is bilateral hemiplegia, but generally little loss of sensation, the sensory tract being deeper. In a lesion of the lateral half of the pons, causing hemiplegia on the opposite side, conjugate deviation of the eyes towards the paralysed side may be present from paralysis of the sixth nerve on the side of the lesion. While in lesions of the internal capsule, the conjugate deviation is away from the paralysed side.

A tumour outside *the medulla* may paralyse the hypoglossal, spinal accessory, and glossopharyngeal nerves on one side; and by pressure on the anterior pyramid cause hemiplegia on the opposite side.

Acute *bulbar* paralysis from hæmorrhage or softening is usually rapidly fatal; there being paralysis of the lips, tongue, palate, pharynx, and larynx.

A lesion of *the middle peduncle* of the *cerebellum* causes intense vertigo, nystagmus, and forced movement of the trunk on its longitudinal axis. According to Gowers, disease of the lateral lobes of the cerebellum, *per se*, causes no definite symptoms, and staggering does not occur unless the middle lobe is compressed. The gait of cerebellar ataxy is reeling like that of drunkenness, the feet being placed wide apart. If the lesion is in the right lateral lobe the

patient staggers to the right side, if in the left lobe to the left side, if in the median lobe he staggers either forwards or backwards. Vertigo, vomiting, and double optic neuritis are almost invariably present.

A tumour of the cerebellum is apt to compress the veins of Galen, or to press on the side of the pons and cause hemiplegia with or without paralysis of the fifth and sixth nerves. These symptoms are of value in localising the growth. Loss of the knee jerk has been observed in cases of cerebellar tumour. I have observed its loss in two cases.

Tumours of the middle lobe of the cerebellum are apt to cause tonic spasm of the spinal muscles, the head being drawn back and fixed, "the cerebellar rigidity" of Hughlings Jackson.

Convulsive seizures are common in cerebellar tumour, as also is tremor of the limbs.

Tremor or other form of spasm may be caused by pressure on the pyramidal tracts and is a sign of considerable localising value in tumours of the encephalon.

I recently saw a child with tremor of both upper extremities on exertion. Now both pyramidal tracts must have been compressed, and the only situation in which one lesion could cause bilateral tremor is either in the pons or in the cerebellum. On investigation I found that there had been vomiting and headache, and the child had a staggering gait, the case being one of cerebellar tumour.

In lesions of *the internal capsule* in its posterior half the hemiplegia which occurs is accompanied with paralysis of the face and tongue, and this is the case also when the lesion is in the crus or pons. If the face and tongue escape, the disease is probably in the cortex. In monoplegia due to brain disease the great probability is that the seat of the mischief is in the cortex, though as I have already stated it may be in the internal capsule, the fibres from each centre remaining separate. Hemiplegia with well-marked hemianæsthesia and hemianopia indicates disease of the posterior portion of the internal capsule, and damage to the corpus geniculatum or optic thalamus.

Paralysis of *both sides* of the *tongue* is usually due to disease of the nuclei in the medulla and the lips suffer with the tongue.

In paralysis of *the face*; if (1) only the lower part is affected and there are (2) no electrical changes, and (3) no loss of conjunctival reflex, the lesion is above the nucleus and probably cortical; otherwise the lesion may be in the nucleus, in the pons or anywhere in its course beyond its superficial origin.

Bilateral facial paralysis is sometimes met with but it is very rare. I lately saw a case under the care of Mr. Oakes, of this town, in which first one side of the face and then the other was paralysed, the lesion being evidently peripheral on both sides. A tumour at the base of the brain in the region of the pons might cause bilateral facial paralysis. But it may be

produced by exposure to cold or by the other causes of neuritis.

Paralysis of *the fifth and sixth nerves* generally indicates disease outside the pons; but I lately removed a brain with a glioma of the right half of the pons where the conjunctiva was insensitive on the right side and the right sixth nerve was paralysed.

Paralysis of *the sixth and facial nerves*, according to Gowers, means disease within the pons. Paralysis of one ocular nerve generally means disease at the base of the brain. Bilateral paralysis of the ocular nerves means disease of their nuclei.

Crus cerebri.—Lesions of or near the crus cerebri are apt to cause what is called a crossed paralysis, there being hemiplegia and facial paralysis on the opposite side with paralysis of the third nerve on the same side. Hemianæsthesia may also be present if the tegmentum or deeper portion of the crus be implicated. Paralysis of the third nerve with hemiplegia of the same side cannot result from a single lesion.

By crossed or alternate hemiplegia is meant hemiplegia on one side of the body with paralysis of a cranial nerve on the opposite side. It is apt to occur—

- 1.—In lesions of or near the crus cerebri.
- 2.—In lesions of the pons, or outside the pons but compressing it.
- 3.—In lesions near the medulla.

Cerebellar growths may compress and paralyse the hypoglossal and other nerves passing from the medulla and at the same time compress the pyramidal tract of the medulla. The body is thus affected on the opposite side ; the nerves on the same side. A cerebellar growth may also compress one lateral half of the pons causing hemiplegia on the opposite side ; and at the same time by compressing the fifth nerve paralyse it on the same side.

Cases of Alternate Hemiplegia.

CASE 1.—*Right hemiplegia, with complete facial paralysis on the right side, and paralysis of the third nerve on the left.*

George Wood, aged 51, a soldier, was admitted into the Infirmary on the 9th of January. His previous history cannot be obtained fully, the patient's memory being defective. He said that he had gone through twenty years' service in the Royal Artillery in India ; that he had had syphilis ; and that six months before admission he had had a stroke on the right side.

There was paresis of the right arm and leg, and the toes scraped the ground in walking. The muscles responded to both Faradic and Galvanic currents, but the intensity of the contractions was below normal. There was paralysis of the right half of the soft palate, the uvula pointing to the left, and the faucial pillars were broader on the right than on the left. There was also slight dysphagia from the affection of the

palate. The deep reflexes on the right side were increased. The patient retained his fæces but had no control over his bladder.

There was complete facial paralysis on the right side ; absence of conjunctival reflex ; and not the slightest response to either Galvanism or Faradism. Sensation was perfect. There was no alteration in hearing. There was paralysis of the third nerve on the left side, as shown by external strabismus, ptosis, and dilatation with immobility of the pupil.

There were no changes in either fundus. The patient was very quiet, in a somnolent condition, and answered questions decidedly slowly ; but there seemed to be no intellectual impairment except slight loss of memory and slowness in comprehension. The patient lived for a few weeks, gradually becoming more feeble and indifferent ; and before his death, conjunctivitis and sloughing of the cornea occurred. He died quietly from asthenia.

He was treated with iodide of potassium in large doses, and highly nutritious food. The disease was considered to be syphilitic. The facial paralysis was evidently peripheral. The right hemiplegia with paralysis of the left third nerve was undoubtedly due to some lesion in the neighbourhood of the left crus, implicating the lower and inner fibres (crust) of the crus, and the third nerve beyond its superficial origin.

Autopsy.—*The head only was examined.* The dura mater was tense and could not be pinched up.

Over the frontal and left parietal lobes it was thickened and blended with the other membranes, and could not be separated except by tearing away the brain substance. The dura was also adherent along the margins of the longitudinal fissure. The arachnoid was opaque, with excess of fluid beneath it; the veins of the pia turgid, and the cerebro-spinal fluid increased in quantity. A yellowish caseous mass was found around the internal auditory meatus, compressing the facial nerve, and apparently arising from the dura. A nodule about the size of a marble, yellow and soft, was also found on the left side on the edge of the tentorium, projecting from its under surface, and in a situation to compress the left crus (and left third nerve) as it passed through the opening in the tentorium. Two nodules were also found close together, situated in the posterior extremity of the right superior temporo-sphenoidal convolutions. There were two small cysts at the genu and posterior third of the internal capsule on the left side, with yellowish contents; these were probably the remains of small hæmorrhages.

The autopsy completely explained and corroborated the symptoms and diagnosis. Microscopical examination showed the growths to be gummata.

In this case it will be observed that the nodules in the right temporo-sphenoidal lobe were not suspected to be present, for they gave rise to no symptoms. Lesions of the temporo-sphenoidal lobe are often latent. Ferrier localises the auditory centre

in the superior temporo-sphenoidal convolutions, but a unilateral lesion would be unlikely to cause deafness ; hearing being bilaterally associated.

CASE 2.—*Left hemiplegia with right facial paralysis.*

E. B., aged 63, a widow. There is no history of paralysis in the family, but her father died from diabetes. The patient has been a healthy woman till three years ago, when she had a stroke which slightly affected her left side ; from this she entirely recovered. On the 9th of May, while standing, she was suddenly seized with giddiness ; she did not fall nor lose consciousness. On trying to move she found she had lost the use of her left arm and leg, but since then has gradually recovered the power in them. She did not notice that her face was affected.

She has marked facial paralysis on the right side, being unable to close her lids. The naso-labial furrow is obliterated, and the angle of the mouth on the left side drawn up. She has the usual difficulty in mastication, the food getting between the gums and cheek. There is paresis of the right half of the soft palate ; no alteration in her sense of taste ; nor is the fifth nerve in any way affected. The conjunctival reflex is abolished, and there is slight conjunctivitis, but the response of the muscles both to Faradism and Galvanism is perfectly normal. The sequence of the polar contractions and their intensity is normal. There is paresis of the left side of the body, and the left arm and leg are slightly wasted. There is

increased patellar reflex on the left side ; no front tap contraction, and no ankle clonus. Sensation on the left side is unaltered. The heart shows no signs of disease, but the arteries are markedly atheromatous. The patient suffers from vertigo, and there is a trace of albumen in the urine, but no casts. The ophthalmoscope shows that both fundi are normal.

The lesion is most probably a small hæmorrhage, and its situation is in the lower part of the right half of the pons just below the decussation of the facial nerves.

CASE 3.--*Cerebellar Disease with Crossed Paralysis.*

W. H., a man aged 48, a fitter ; has been a soldier, and has served in India. He had syphilis when a young man, but was otherwise quite well until about two years ago, when he suffered from headache and giddiness, with obstinate constipation. He suffered occasionally with shooting pains in the right arm, and also had deficient control over his rectum and bladder. The patient has been gradually getting worse up to the present time (Feb. 25th, 1884). He sleeps badly, and his memory is not as good as it was. His intellect appears to be clear. The bowels are confined. His appetite is good, but he has suffered from persistent vomiting for the last six weeks. He always vomits after he takes food ; but also, independently of food, especially in the morning. The vomiting is not attended with nausea. He suffers from headache, which at first was occipital but is now frontal. The headache is severe and

paroxysmal, and the patient has a wearied look indicative of suffering. He suffers from active vertigo; objects appearing to move to the right. He cannot walk without help, and his gait is reeling; the patient staggering and always falling to the right side. He has paresis of the right side; the grasp of the right hand being much weaker than that of the left. The triceps and biceps reflexes are exaggerated on the right side, clonus being obtained by tapping the lower end of the right radius. The patellar reflex is exaggerated on the right side, and there is also ankle clonus, but no front tap contraction. The gluteal, lumbar, and scapular reflexes are absent. The plantar, cremasteric, and epigastric well marked. Sensation is perfect. The pupils respond to accommodation freely, but not so readily to light. Both arms tremble, but especially the right, all the movements of which are attended with tremor. The tongue points to the opposite side, *i.e.*, to the left; whereas in ordinary hemiplegia it points to the same side. The deflection of the tongue is marked, and the left half is smaller than the right. The tongue also is tremulous. The optic discs are normal, only a few white lines along the vessels being observed.

The patient was admitted into the Queen's Hospital on the 25th of February. He suffered severely from cerebral vomiting, which was checked by hourly drop doses of vin. ipecac., and has not recurred since. He also was much constipated. The patient was treated with iodide of

potassium (gr. xxv. twice a day), and improved greatly. His gait became steadier; the headache ceased; and the grasp of the hand was stronger. It was noticed that there was a periodicity in the attacks of headache, and the stagger to the right was well seen when he was ordered to turn round quickly. The patient was kept in six weeks, and then discharged much improved. He is still under treatment as an out-patient, and still shows the slight hemiplegia and the deflection of the tongue.

As to the diagnosis:—The cephalalgia, vertigo, and vomiting, point to cerebral tumour; and were there double optic neuritis the diagnosis of tumour would be positive. The staggering gait indicates that the lesion is in the cerebellum. But he has also a crossed paralysis, for there is paresis of the right side of the body, and paralysis of the left half of the tongue. We can explain this by supposing that the lesion in the cerebellum has compressed or invaded the motor tract in the medulla on the left side above the decussation of the motor fibres, thus causing hemiparesis and descending degeneration of the crossed pyramidal tract in the right side of the cord, and has compressed the trunk of the left hypoglossal nerve also. There is no sign of any lesion of the vagus, glosso-pharyngeal, or spinal accessory nerves.

As to the nature of the lesion:—It is probably gummatous, for there is a distinct history of syphilis, and the man improved greatly under the influence of iodide of potassium.

CHAPTER V.

THE NATURE OF THE LESION IN BRAIN DISEASE.



Diseases of the Brain.

I.—ORGANIC DISEASES.

(A.) Of the Membranes	{	of the Dura—Pachymeningitis	{ Externa. Interna.
		of the Pia	{ Acute { Simple. Tubercular. Chronic.

(B.) Of the Brain proper—

(1) Diffuse Diseases.

Anæmia and Hyperæmia.

Cerebritis or Encephalitis { Diffuse.
Local.

Chronic Degenerative Diseases.

(2) Focal Diseases.

Tumour.

Hæmorrhage.

Softening.

II.—FUNCTIONAL DISEASES.

In diagnosing the nature of the lesion in diseases of the nervous system, we must carefully enquire as to the mode of onset of the symptoms ; whether the onset was immediate, rapid, or gradual.

In the first case, where the symptoms develop in a few minutes or hours, the lesion is probably vascular. A sudden onset, excluding functional disorders, means a vascular lesion.

Where the onset of the symptoms is rapid, occupying from a day or so to two or three weeks, the lesion is in all probability inflammatory.

Where the onset is chronic, occupying more than three or four weeks, the lesion may be a chronic inflammation, a degeneration, or a growth.

Meningitis.

The general rule that inflammation of a viscus is attended with little pain, while inflammation of its coverings or capsule is attended with great pain, holds good in the case of the brain and its membranes.

Encephalitis is accompanied with little pain, while in meningitis pain is the prominent symptom.

Inflammation of the membranes of the brain causes two sets of symptoms :—

1.—In an early stage those due to irritation of the cranial nerves and surface of the brain, and

2.—Later on those due to compression, and loss of function, of the cranial nerves and brain.

In meningitis, the phenomena of irritation are more marked and more lasting than in cerebritis—in the latter the phenomena of irritation are less marked, fever and pain are less, and mental loss is usually more profound.

The symptoms of meningitis vary with the situation of the inflammatory mischief, whether at the base or convexity of the brain.

In both varieties (1) headache is the prominent symptom, with (2) vomiting, (3) pyrexia, and (4) delirium.

If at the base, the cranial nerves are at first irritated and finally paralysed; so that at an early stage we find photophobia, spasmodic squint, contracted pupils, irritability of temper and restlessness.

Vomiting, a slow pulse, hyperæsthesia of the special senses, and optic neuritis may also be present.

Retraction of the head is very constantly present, especially in children.

Later on the inflammatory effusion which occurs compresses and paralyses the nerves, and we find paralytic squint, dilatation of the pupils, an irregular and perhaps rapid pulse (from paralysis of the vagus), with blindness and deafness. Irritability giving way to somnolence and coma.

Tubercular meningitis is, as is well known, often with difficulty distinguished from typhoid fever.

In one of my cases a young man, about 18 years of age, was admitted in a delirious condition. No

account could be obtained either of his family history or of the mode of onset of his illness. There was considerable fever, a pulse not increased in ratio with the fever, and a brown tongue. No spots could be found, but there was marked tenderness in the right iliac fossa, and enlargement of the spleen with signs of bronchitis. The stools were not examined, the patient only living a few hours after admission. I pointed out to the students with me that the case was either one of general tuberculosis or of typhoid, and summing up the pros and cons, concluded that the case was probably typhoid; at the autopsy, however, general tuberculosis was found, the tenderness in the iliac fossa being due to tubercular ulceration of the ileum.

In another case, that of a child, there was a history of vomiting, loss of flesh, and constipation. On admission the child was found to be emaciated; there was fever, no rash, a markedly retracted abdomen, and symptoms of cerebral irritation, the child lying curled up in bed with its head buried in the pillows, and crying out when disturbed. Photophobia was marked, and the spleen was not enlarged. The case was thought to be one of tubercular meningitis; the next day, however, after the application of an ice bag to the head, the child was perfectly conscious, all the irritative phenomena had disappeared, the retracted abdomen had become swollen, and diarrhœa with typhoid stools had supervened.

It is necessary, then, to be very careful in the diagnosis of the cause of delirium and cerebral irritation in children. In many cases it is simply the result of pyrexia.

Another condition which may simulate meningitis in children has been called the *hydrocephaloid* condition by Marshall Hall. It occurs in young children exhausted by diarrhœa. The patient may become somnolent or comatose. Convulsions, strabismus and dilatation of the pupils may be present, but after death no change is found in the brain. This condition may be distinguished from meningitis by the history of severe diarrhœa preceding the cerebral symptoms, and by depression of the fontanelle, whereas in meningitis the fontanelle bulges.

In distinguishing between tubercular meningitis and typhoid fever, it is of great importance to learn the history of the case. An enlarged spleen and diarrhœa point to typhoid fever. Spots, if present, are conclusive of typhoid. Optic neuritis or tubercles in the choroid are conclusive of meningitis. The diagnosis in some cases cannot be made with certainty.

Meningitis of the convexity of the brain does not, of course, affect the various cranial nerves, nor does it cause papillitis; but headache, vomiting, delirium, and pyrexia are present. Tremor of the limbs, followed by paralysis, may be present from implication of the cortical motor centres. The temperature is higher, and the disease more acute

and less liable to remission, than in tubercular meningitis, which is almost invariably basilar.

Pachymeningitis.

Pachymeningitis of the cerebral dura mater may be divided into the following varieties:—

1.—Pachymeningitis *externa*, which is produced by

(a.) Injuries which separate the dura from the skull and occasion extravasation of blood between them. The clot may compress the brain and cause death, or inflammatory changes may be set up.

(b.) Extension of inflammation from the neighbouring bones, the most common of such cases being caries of the petrous portion of the temporal bone.

External pachymeningitis probably always arises from causes such as those above mentioned. The symptoms vary with the seat of the mischief, but severe pain in the head is nearly always complained of, the dura mater being freely supplied by the fifth cranial nerve. If the brain is compressed convulsions followed by hemiplegia may result.

2.—Pachymeningitis *interna hæmorrhagica* occurs most commonly in old age, and always in adult life. It may be caused by injury or by any condition conducive to continued hyperæmia of the dura mater; such as alcoholism, sunstroke, and various cachectic conditions. The symptoms vary greatly with the seat of the lesion, but headache is the most constant. Motor disturbances are common. There may be

twitchings of one or both sides of the body, or paresis following a transitory loss of consciousness. The course of the disease is chronic; and the first symptom may be a transient loss of consciousness with hemiplegia, due to a slight hæmorrhage, which is usually followed by two or three others. These attacks resemble cerebral hæmorrhage, but are usually not accompanied by such profound coma, and the hemiplegia is usually transitory. In severe cases, however, the symptoms cannot be distinguished from those of ordinary apoplexy.

Encephalitis.

Encephalitis or cerebritis, except from injury or in connection with disease of the bones in the form of abscess, is rare; and can rarely be distinguished from meningitis, with which, indeed, it is frequently combined.

The phenomena of irritation and implication of the cranial nerves are less frequent and less pronounced than in meningitis, while the symptoms of mental impairment and loss of cerebral functions are more prominent. Headache, loss of memory, impairment of speech, imbecility, and coma are commonly observed, the symptoms depending, of course, upon the situation and extent of the inflammatory softening.

In cerebritis the headache is less violent and the fever not nearly so high as in meningitis.

Abscess of the brain is very commonly due to suppuration in the tympanum and necrosis of the petrous bone, it also frequently follows injuries to the head. The symptoms of abscess are very much the same as those of tumour of the brain. Optic neuritis and headache may be present in both. Abscess of the brain is usually attended with much greater emaciation and impairment of the general health than is the case in tumour. The focal symptoms will depend upon the situation of the abscess, and these alone enable us to localise the lesion.

Abscess of the brain is more often latent than tumour, and grave symptoms may supervene suddenly from rupture of the abscess, either into the ventricles or on the surface of the brain. Abscess, moreover, usually runs a more rapid course than tumour, and is accompanied with fever.

Caries of the mastoid cells causes abscess most frequently in the cerebellum. Caries of the tympanum causes abscess in the temporo-sphenoidal lobe; this rule as to the different situation of the abscess in disease of the mastoid and petrous portions of the temporal bone was laid down by Mr. Toynbee. The presence of paralysis or of spasm on one side of the body would indicate that the abscess was in the temporo-sphenoidal lobe near the motor convolutions.

In cases of ear disease with cerebral symptoms, look for pain and tenderness over the mastoid process, and if there is in addition redness or

pitting upon pressure, suppuration of the mastoid cells is certain, and a free incision is urgently called for, with subsequent trephining of the mastoid cells. It is not always easy to distinguish between cases of ear disease alone and those cases where purulent meningitis and abscess have been set up.

The subjects of chronic otorrhœa often suffer from symptoms of cerebral irritation, lasting a few days and then passing away, being due to changes in the dura covering the petrous bone. If paralytic symptoms are present, or if there is optic neuritis, we may be sure that either purulent meningitis or abscess, or both are present.

Tumour.

Tumour of the brain is more common than abscess, and may be of almost every variety. Tubercular tumours are the most common, then come syphilitic growths, and thirdly gliomata.

Syphilitic tumours are characterised by their multiplicity. Tubercular tumours also may be multiple. Gliomata occur singly, they invade the brain tissue, and are liable to be the seat of hæmorrhage from their highly vascular structure. A fall or a blow upon the head may cause the development of a glioma in the brain.

I recently removed the brain of a little boy who had injured his head in falling a few months previously, and since then had suffered from headache, other symptoms of cerebral tumour gradually supervening. I found a large glioma of the left side of the pons.

The characteristic symptoms of cerebral tumour are—(1) headache, (2) vomiting, and (3) double optic neuritis. But optic neuritis may be absent and so may vomiting. Stupor is a common symptom of tumour of the brain. Cerebral tumour, like abscess, may be latent for a considerable period. The situation of the tumour can only be diagnosed by the presence of paralysis or spasm in various situations, or by a staggering gait. In cerebellar tumour tonic convulsive seizures may occur in addition to the symptoms previously mentioned, and are due to pressure on the pons or medulla. The successive invasion of cranial nerves is very suggestive of cerebral tumour.

Case of Tubercular Tumours of the Optic Thalami.

W. P., a boy, aged 3 years and 9 months, was admitted into the Queen's Hospital on the 18th of June, with left hemiplegia and tremor of the right upper extremity. The father and mother are healthy, and there is no history of consumption on either side; but of the six children they have had, the eldest died from chronic hydrocephalus, the second died at the age of nine months from diarrhoea and convulsions, and one daughter is under treatment for strumous glands of the neck. The boy has always been weakly and ailing; when he was about three months old he had bronchitis, and since that time has never been really well. About two months before admission he complained of pain in the right side of the

head, and his mother noticed that his right arm and leg were occasionally convulsed, and that the left side was paralysed. The child used to vomit about every third day, and on those days would refuse all food and drink. The vomiting would come on suddenly without evident cause, and without retching. The vomiting has ceased since about the 4th of June. Constipation has been marked during the last two months. On admission the boy was found to have left hemiplegia, the face being affected, with rigidity and increase of the deep reflexes. Tremor of the right hand of a fine character was present on exertion. The boy was in a torpid, somnolent condition, and had the vacant expression of amaurosis. The right pupil was the larger, but both responded to light and accommodation. The mother gave a distinct history of fits, in which, she said, he always put his hand to the right side of his head, and then uttered shrill short screams, and his left arm would be convulsed and then the leg. Double optic neuritis was found to be present in a marked degree, with some hæmorrhages into the retina. There was no sensory disturbance, and no paralysis of the ocular muscles. No fits occurred while the patient was in the hospital, but the drowsiness gradually deepened into coma, and he died on the 26th June, the temperature rising before death to 105° F.

Autopsy.—The membranes and convolutions of the brain were healthy. On removing the corpus

callosum and fornix, two tumours were seen side by side, occupying the anterior portion of each optic thalamus; that in the right thalamus being the larger, and about as large as a pigeon's egg. The velum interpositum was separated with difficulty from these tumours. The third ventricle was almost obliterated, the growths being adherent in front. The tumour on the right side extended out into the internal capsule, and some circumscribed yellow softening was found around. On section each tumour was composed of a soft yellow material. The lungs contained caseous masses, and the bronchial glands were considerably enlarged and caseous. The mesenteric glands were caseous and enlarged.

This case was of course easily diagnosed as one of cerebral tumour. The nature also of the growth was foretold by the markedly scrofulous history in the other children. The boy's sister is still under my care with scrofulous enlargement of the cervical glands. The mother's account of the boy's fits led me to suppose they were of the Jacksonian type, and indicated mischief in the motor area of the cortex.

Tubercular tumours also far more frequently occur on the surface than in the interior of the brain.

I therefore imagined that there was a tubercular mass in the convolutions around the fissure of Rolando on the right side; this supposition, however, did not account for the tremor on the right side which was observed for a few weeks before death.

The hemiplegia in this case was due to pressure upon the internal capsule, the paralysis having supervened late in the course of the symptoms. The tremor of the right upper extremity was also probably due to pressure upon the left internal capsule. The movements were not at all like those of post hemiplegic chorea or athetosis, in which affections the lesion is nearly always situated in the posterior and external part of the optic thalamus.

Tumours of the optic thalami are by no means common.

Aneurism.

Aneurisms of the cerebral arteries give rise to the symptoms common to most cerebral tumours, and focal symptoms according to their situation.

Embolism is a cause of aneurism, so that the presence of heart disease in a case of tumour of the brain should excite suspicion as to its being an aneurism.

In rare instances a bruit has been detected over the cranium.

Atheroma, syphilitic, and fibroid degeneration of the vessels are common causes of aneurism.

Aneurism of the cerebral arteries may occur in youth and in both sexes.

The miliary aneurisms first described by Charcot give no symptoms until they rupture.

Softening.

The term softening is used in medicine in its literal sense, and ought not to be loosely applied to

CEREBRAL HEMIPLEGIA

FROM

	HÆMORRHAGE.	EMBOLISM.	THROMBOSIS.
Age—	Over forty years. Rarely in early adult life.	All ages.	In old age. Often occurs in early adult life from Syphilitic Arteritis. May occur in young children after the specific fevers or exhausting diarrhoea.
Prodromata—	Usually present.	Absent.	Usually present.
Onset—	Gradual.	Immediate.	Gradual.
Coma—	Usually present. Is the characteristic feature.	Absent. May be present in aged subjects.	Absent. May be present in aged subjects.
Recovery—	Rarely complete.	Rarely complete.	Often complete.
Recurrence—	Frequent.	May occur.	Very frequent.
Late rigidity—	Often very marked.	Often very marked.	Slight except in Syphilitic Thrombosis, when it is marked.
Arteries—	Atheromatous.	May be healthy. An aneurism may be the source of an Embolon.	Atheromatous.
Heart—	Often hypertrophied.	Endocarditis often present.	May be healthy. Often feeble.
Kidneys—	Bright's Disease often present.		

In Embolic Hemiplegia the right side of the body is more often paralysed than the left, and other Embolic phenomena may occur, *e.g.*, Embolism of the Spleen, Kidneys, or Central Artery of the Retina. Cerebral Hæmorrhage frequently occurs at night.

all cases where there is mental defect ; where, in fact, there is frequently no softening.

The two most common causes of softening are inflammation and occlusion of the blood vessels. The blood vessels may be occluded either by an embolon or by a thrombus.

Embolism and thrombosis of the cerebral arteries produce symptoms, of course, varying with the artery affected.

The middle cerebral artery is frequently the seat of these lesions, hemiplegia resulting.

Hemiplegia occurring suddenly may be due to (1) hæmorrhage, (2) thrombosis, or (3) embolism. Charcot and Bouchard showed long ago that miliary aneurisms are common on the small branches of the cerebral arteries of old people, and that these miliary aneurisms precede rupture of the vessels.

They give no symptoms themselves, and are most numerous on the antero-lateral group of perforating arteries.

The following are the chief features of cerebral *hæmorrhage*, cerebral *thrombosis* and cerebral *embolism*.

The commonest seat of hæmorrhage is the corpus striatum, the lenticulo-striate artery especially being the one that ruptures. Hæmorrhages are rare in the cortex, softening being more common in this situation, Cerebral hæmorrhage frequently occurs at night, the recumbent position not being so favourable to the return of venous blood from the brain as the erect.

The striking symptom of the apoplectic condition is coma, the loss of consciousness depending on the rapidity of the hæmorrhage, as well as upon the extent of hæmorrhage. The loss of consciousness is not immediate, but in the great majority of cases is gradual, being preceded by paresis of the side, headache, numbness, &c.

Deep coma is nearly always due to hæmorrhage, not to softening.

A small hæmorrhage, however, may not cause coma.

In old people softening may be attended with coma.

Hæmorrhage is rare under the middle period of life ; embolism occurs at all ages.

Thrombosis is very common in the aged, but its occurrence in young adults from syphilis is common, and it also occurs in children exhausted by specific fevers or diarrhœa.

In hæmorrhage and thrombosis prodromata are usually present in the shape of headache, vertigo, attacks of numbness and tingling in the limbs, which later on became paralysed.

Complete recovery is rare after hæmorrhage, common after thrombosis.

Coma is usually absent in thrombosis and embolism, except in a mild degree, but thrombosis of the basilar artery and of the sinuses is usually attended with coma.

In thrombosis (1) contractures are not so common as after hæmorrhage and embolism, except in syphilitic thrombosis; (2) the development of the paralysis is usually gradual and recovery often rapid; (3) there is also usually a history of several attacks.

High arterial tension and cardiac hypertrophy point to hæmorrhage.

Albuminuria also indicates hæmorrhage.

In embolism there are (1) no prodromal head symptoms; (2) there is usually valvular disease or aneurism.

In old cases of embolic hemiplegia the murmur may have disappeared, and we must rely on the history of rheumatism in diagnosing its cause.

Embolic hemiplegia is most common on the right side, the left common carotid coming off directly from the aorta, affording a more direct course for an embolon.

Embolism of the central artery of the retina, or of the spleen, or kidney, may occur at the same time as the hemiplegia, and thus positively indicate its nature.

Cerebral hæmorrhage occasionally occurs in early adult life. I have at present a young man, aged 21, of very fine physique, who is suffering from right hemiplegia. I find he has considerable cardiac hypertrophy and a very hard pulse. The hemiplegia occurred at night, and I have no doubt that it was due to hæmorrhage. He enlisted as a soldier at 18, and had a good deal of heavy work and athletic

exercise to perform, the strain causing cardiac hypertrophy and finally cerebral hæmorrhage.

Chronic Degenerative Diseases.

There are three chronic degenerative diseases of the brain—(1) general paralytic dementia, (2) multiple sclerosis, and (3) bulbar paralysis.

Cases of *general paralysis of the insane*, one of the most fatal of all diseases, are occasionally seen among the out-patients of hospitals. At the Birmingham Workhouse Infirmary there are invariably two or three cases in the wards.

This disease is frequently overlooked in its early stage, and several of my cases had attended at hospitals without their true condition being detected. As seen in the out-patient room, the patient is usually a young man, looking robust and in good health, but you are told by his friends that he has been unable to follow his employment for some time on account of his mental failure.

The symptoms in the early stage are :—

- 1.—Loss of memory and optimism.
- 2.—Inequality of the pupils.
- 3.—Tremor of the lips, tongue, and facial muscles, when put in action.

Maniacal outbreaks, apoplectiform attacks, and attacks of hemiplegia are common.

Epileptiform convulsions are of frequent occurrence.

Spinal disease may precede or follow the cerebral symptoms. The two most common forms of

cord disease associated with general paralytic dementia being locomotor ataxia and lateral sclerosis.

It must be remembered that there are cases of pseudo-paralytic dementia characterised especially by optimism, but the symptoms mentioned above are not all present.

When the disease has advanced the diagnosis is easy. The remarkable condition of well-being (*bien-être*), the patients generally expressing themselves as feeling well and happy, and the characteristic delusions, with debility and slurred speech, rendering the diagnosis easy.

The characteristic features of *multiple sclerosis* are :—

1.—The tremors, supervening on exertion ceasing during rest, and accompanied with paresis. The tremor implicates the head and trunk.

2.—When the brain is affected—an alteration in speech, the syllabic utterance, is frequently observed.

3.—Nystagmus is also often observed when the eyes are moved in any direction so that the muscles are strained.

4.—Mental changes are usually present, such as loss of memory for recent events, unnatural cheerfulness, and imbecility.

5.—Vertigo is also commonly present.

6.—The gait is unsteady, the head being held stiffly, and the patient is apt to lurch violently to one side.

7.—Apoplectiform attacks frequently occur.

This disease may be mistaken for paralysis agitans, general paralytic dementia, and cerebellar tumour.

1.—The tremor of disseminated sclerosis only occurs with voluntary movement ; that of paralysis agitans, while the muscles are at rest. The tremor of disseminated sclerosis is much coarser than that of paralysis agitans.

2.—Disseminated sclerosis occurs in early life, paralysis agitans is a disease of advanced life.

3.—Nystagmus, mental symptoms, vertigo, and alteration of speech at once distinguish disseminated sclerosis from paralysis agitans.

From cerebellar tumour, multiple sclerosis may be distinguished by the absence in the latter complaint of (1) optic neuritis, (2) vomiting, and (3) headache.

Tremor may occur in cerebellar tumour from pressure on the motor tracts, but is rare.

4.—Convulsive seizures are common in cerebellar tumour, rare in disseminated sclerosis.

In *bulbar paralysis* or *glosso-labio laryngeal paralysis* the tongue, palate, and vocal cords are paralysed bilaterally ; the lips and pharynx being also involved. Defective articulation is the first symptom, the lingual consonants being especially articulated with difficulty. The patient finally is unable to speak. Swallowing becomes difficult, liquids regurgitate into the nose, and food may get

into the larynx. The aspect of the patient is very characteristic. He is apt to attract attention in the streets by his open mouth and silly appearance. The tongue soon becomes completely paralysed and lies like a log in the mouth.

Glosso-labio laryngeal paralysis is a chronic progressive paralysis which may be primary or supervene on progressive muscular atrophy.

Bulbar paralysis may be acute, as when it is due to hæmorrhage into the medulla, or to plugging of its vessels.

Glosso-labio laryngeal paralysis in its initial stage simulates general paralysis of the insane, but the condition of the pupils and mental symptoms readily distinguish the latter, and, moreover, the tremor of the lips and tongue, which is usually well marked in general paralysis of the insane, is absent in bulbar paralysis.

Functional Affections of the Brain.

Among the functional disorders of the brain are included such diseases as (1) epilepsy, (2) chorea, (3) hysteria, (4) hypochondriasis, (5) delirium tremens, (6) neurasthenia, (7) catalepsy, (8) trance.

Hysteria.

Hysteria in women and *neurasthenia* in men is nearly always the outcome of failing health, especially from worry and mental strain. Cases of hysterical paralysis and contracture rarely recover suddenly, as is

usually supposed, but recovery is generally gradual and effected with difficulty, relapse being common. Isolation, good feeding, and Faradisation are the agents to be relied upon in the treatment of hysteria and its manifestations, but even these agents may fail entirely. A woman, aged 25, whose husband had deserted her, came under my care suffering from paraplegia with rigidity, but with no loss of sensation. I have no doubt that the paraplegia was hysterical, but I was unable to do any good. When the patient was isolated and Faradised, she commenced to have severe convulsive seizures of the left arm, and her condition became worse. Paralysis depending upon idea, on the other hand, may get well immediately. Neurasthenia in men takes the place of hysteria in women. In America especially, where competition is very keen, this disease is common, and it is getting more prevalent in our own large cities. It occurs amongst the brain-workers especially. I have met with a large number of such cases. The following is a typical illustration of the disease:—A young man, who had greatly overworked himself in the service of an important company, and had gained for himself a very responsible position by his hard work, gradually became nervous and irritable. When I saw him he was suffering from what is called claustrophobia—a dread of being in narrow and closed places. He could not travel in a railway carriage, and although a season ticket holder, was obliged to walk or ride a tricycle. The dread of being shut up

in a railway carriage was so great that he could not go to London on some important business, which occasioned him great loss. He was also afraid of going to any place of public meeting, and felt a constant dread lest he should do something which would disgrace and ruin him. These are very distressing symptoms and the patient is always depressed and miserable.

No drug will cure this neurasthenic state. Absolute rest from mental work and worry, with change of scene, especially a sea voyage, and good feeding are the remedies. The patient should be encouraged and should be told that there is no organic mischief present and that he will recover.

Such cases as the one I have recorded are very common and are on the increase. They are apt to pass into melancholia, and, in fact, are closely allied to insanity.

Mental depression should always be looked upon as a grave symptom ; this fact has been impressed upon me by two cases of suicide in which there was depression of spirits only, without delusion or mental loss.

I find that medical men are apt to take too little notice of this symptom, and thus the patients are left unattended and the friends unsuspecting. Whereas in all cases where mental depression is marked the patient ought to be watched, and the friends advised as to the danger of suicide.

Hypochondriasis.

Hypochondriasis is a chronic mental disorder far more frequently met with in men than in women. It is closely allied to and frequently terminates in melancholia. The patient is morbidly anxious about his health. He may always complain of the same trouble, either of his stomach or liver, &c., or he constantly changes his complaint; as soon as you convince him that his liver is all right, he complains of his heart, and so on. The symptoms in these cases are always subjective, and it is often impossible at one examination to come to the conclusion that the case is one of hypochondriasis.

At the Workhouse Infirmary I have had several cases under observation for some months at a time. In these cases the symptoms were subjective, and I could never find any cause for the loudness and persistence of their complaining. They always talked of their sufferings to their fellow-patients; were constantly writing long lists of questions for me to answer; and always wanted me to see them whenever I went in the ward.

All these cases finally showed unmistakable symptoms of melancholia, and were sent to an asylum.

Case of Hypochondriasis terminating in Melancholia.

W. P., a man aged 47, came to me in June, 1886, complaining of flatulency and of a sinking sensation at the epigastrium. He has always been of an anxious,

worrying disposition, but has been much worse since he was overworked a year ago, and was very frightened by spitting a little blood. His sister is in an asylum, but no other history of insanity in his family can be obtained. He told me that he felt he was going to die ; that food turned sour on his stomach ; and that he was going out of his mind. When assured that there was no organic disease whatever, and nothing of consequence was wrong with his stomach, he began to complain of his heart—was sure that it was seriously diseased. He was obliged to give up his employment for he could not settle to work. His companions said that he was continually talking to them of his ailments, and telling them that he was going to die. His wife said that it was wretched to live with him on account of his complaining. After seeing me he would be better for a few hours, but would shortly find something he had not asked me about, and would worry about it all night and come again the next day.

After leaving me he would frequently return in a few minutes to ask some other question, and always brought a paper full of written questions for me to answer. He had a great dread of catching cold, and was afraid to use a comb for fear it would injure him. He soon began to show distinct delusions, became very depressed, and talked of killing himself. Finally, his friends were obliged to send him to an asylum.

Chorea.

Chorea must be differentiated from multiple sclerosis, paralysis agitans, and hysteria.

The movements of chorea are disorderly, the great characteristic of the movement being the *jerk*; while in multiple sclerosis and paralysis agitans the characteristic movement is *tremor*, which is rhythmical and fine.

In hysteria the movements are more co-ordinated and regular than in chorea.

The term "habit chorea" has been used by Weir Mitchell for a light form of the affection, consisting of some grimace or shrugging of the shoulders only. Hemiplegia may precede or follow chorea, and muscular weakness is always present.

Chorea may be periodic, especially in malarious districts, and there is a great tendency to relapse either in the Autumn or Spring.

Chorea, occurring in men after rheumatism, is very fatal.

I have met with two cases:—One man was treated for acute rheumatism in the Queen's Hospital, and later on came up as an out-patient with chorea. He was admitted again and rapidly became maniacal and died.

The other case was sent into the Workhouse Infirmary. He had chorea and acute mania. I sent him to the asylum, but he died there a few days after admission.

Epilepsy.

The characteristic feature of epilepsy is loss of consciousness, attended or unattended with convulsive movements. We may distinguish four varieties :—

1.—*Epilepsia gravior*—in which the attacks are severe and convulsion is a prominent feature.

2.—*Epilepsia mitior*—in which the attacks are mild and convulsion absent or slight, there being transient loss of consciousness only.

3.—Abortive epilepsy—in which consciousness is not completely lost. These attacks usually occur in the course of ordinary epilepsy; the patient, being in a purely automatic condition, performs eccentric actions, sometimes running along the street or going up into a corner of a room and micturating, &c.

4.—Partial epilepsy (*Jacksonian epilepsy*). This variety has been previously described.

Epilepsy must be distinguished from *uræmia*, *hysteria*, *syncope*, and *malingering*.

Uræmic convulsions are usually preceded by drowsiness; dropsy may be present and the urine is albuminous. In all cases in which convulsions occur and in which the heart is enlarged and the arterial tension high, examine the *fundus oculi* and the urine.

Attacks of *syncope* may simulate *petit-mal*, but spasm is not present and the pulse is feeble.

Epilepsy must be carefully distinguished from *hysteria*; the two diseases frequently occur together in the same individual.

In epileptic women an hysterical attack frequently follows the fits, and such women are at all times liable to hysteria.

Biting of the tongue, incontinence of urine and fæces, and sudden loss of consciousness are peculiar to epilepsy. In hysterical attacks the patient is noisy, talks during the attack, and does not completely lose consciousness.

In *hystero-epilepsy*, a disease distinct from epilepsy, and which was first clearly recognised by Charcot, convulsive movements are markedly present.

There is a prodromal stage of the attack, in which ovarian hyperæsthesia, globus, and clavus are present.

In the actual attack the first stage is characterised by tonic convulsion—the entire body being rigid, and respiration suspended; in the next stage tetaniform and clonic spasms occur, respiration being irregular and stertorous.

Finally, delirium appears, the patient assuming various attitudes expressive of her emotional condition, and having hallucinations; hemianæsthesia and contracture are of frequent occurrence.

In these cases hysterogenic zones have been described, consisting of limited cutaneous areas, which when pressed upon, blistered, or Faradised, often give rise to or check an attack.

In epilepsy, when the fits are frequently repeated, the patient not recovering consciousness between

them, the temperature may rise to a high degree, even to 106°F. more ; this condition is called the "status epilepticus," and is very dangerous.

In hystero-epilepsy, even when the attacks are exceedingly frequent, the temperature, according to Charcot, rarely attains a height of 100°F., and this he considers to be a very important diagnostic sign of hystero-epilepsy as contrasted with epilepsy.

A malingerer cannot assume the pallor and the wide dilatation of the pupils present at the commencement of an epileptic attack.

CHAPTER VI.

THE USE OF ELECTRICITY IN DIAGNOSIS.



Three varieties of electricity are employed in medicine :—

1.—Static or friction electricity—Franklinism.

2.—The constant current—Galvanism, Voltaism ; the current always flowing in the same direction, from the positive to the negative pole, and being produced by chemical action.

3.—The induced or Faradic current, which is momentary in its action, constantly changing in its direction, and produced from voltaic or magnetic source.

The Galvanic and Faradic currents alone are useful for diagnostic purposes.

The motor points, or the exact spots where the nerves enter the muscles, must be borne in mind ;

irritation here causes totally different effects from stimulation of the muscular fibres themselves, the whole muscle in the former case being thrown into contraction.

Diagrams of these motor points are given in works on medical electricity, such as those of De Watteville or Hughes Bennett.

Electrical Reactions in Health.

In health, if the motor nerve be irritated with the Faradic current, each shock produces a contraction of the muscles supplied by the nerve, and as the interruptions rapidly follow one another a tetanus of the muscles is produced.

If the nerve be irritated with Galvanism, muscular contractions take place only at the make and break; the contractions are short, sudden (not tetanic), and quickly relax.

While the current is passing without any great variations in its strength there are no contractions.

The muscular contractions vary according to the direction of the current:—

With one electrode on the back or sternum and the other on the nerve (the polar method), it will be found that the cathode (negative pole) always predominates over the anode in its power of inducing contractions.

With currents of gradually increasing intensity, C C C, or the cathodic closure contraction (the anode

being placed over the sternum and the cathode over the nerve) is obtained with a weak current ; with a stronger current we obtain not only C C C, but also A C C (the anodic closure contraction), the cathode being on the sternum.

With still stronger currents we obtain three contractions, C C C, A C C, and A O C (A O C being the anodic opening contraction).

And with very strong currents four contractions, C C C, A C C, A O C, and C O C (C O C being the cathodic opening contraction).

The practical fact to be noted is that in health C C C exceeds A C C.

When the Galvanic and Faradic currents are applied to the muscles in health, contractions are produced exactly as when the nerves are irritated, but are less extensive ; only those portions of the muscles contracting which are in the immediate neighbourhood of the electrodes. If a muscle, however, be deprived of its nerve influence by curare, Faradism applied to the muscle causes no contraction whatever. Galvanism causes an increased contraction, a weaker current producing a contraction than is necessary to do so when the nerve is intact. But so long as the muscle is healthy the sequence of the polar contractions remains the same. The mode of the contraction is altered, for when the nerve is intact the contraction is sudden and short ; when the nerve is removed the contraction is delayed and prolonged.

Faradism acts on nerves alone. Galvanism acts on both nerves and muscles. There is no true farado-muscular irritability.

Electrical Reactions in Disease.

It is found that after section of a motor nerve, corresponding with the degenerative changes which occur in the nerve below the section and in the muscles supplied by the nerve, important modifications in the electrical reactions ensue.

First, as to the nerve,—the response to both currents is increased for a few days, then diminished without alteration in the sequence of the polar reactions, and in from one to two weeks the excitability of the nerve to both currents is abolished. A nerve may regain its power of conducting voluntary impulses long before it regains its electrical excitability below the seat of injury.

Secondly, as to the muscle,—the Faradic excitability rapidly diminishes, and is lost in about two weeks; this is due to the degeneration of the intramuscular nerve fibres.

With Galvanism we obtain the so-called “reaction of degeneration” (Erb), which indicates degenerative changes in the muscle. There are serial and modal changes in the contractions. During the first fortnight, galvanic irritability of the muscles is diminished; then their excitability to slowly interrupted currents is increased, and serial changes occur; instead of C C C predominating, A C C does,

the anode taking the place of the cathode ; and the order of the contractions with gradually increasing currents is

$$\left. \begin{array}{l} A \ C \ C \\ C \ C \ C \\ C \ O \ C \\ A \ O \ C \end{array} \right\} \text{ instead of } \left\{ \begin{array}{l} C \ C \ C \\ A \ C \ C \\ A \ O \ C \\ C \ O \ C \end{array} \right.$$

The modal changes are very important, and indicate degeneration of the muscle ; the contraction wave is sluggish and prolonged, and apt to become tetanic. If the galvanic irritability is lost then the muscle is destroyed.

The diminution or loss of Faradic irritability in a nerve indicates its degeneration ; in a muscle, degeneration of its nerve only.

Increased response of muscle to Galvanism with reaction of degeneration means degeneration of the muscle.

Total loss of galvanic reaction means complete destruction of the muscle.

In disease, if the lesion is above the nucleus of the motor nerve, then we either have normal electrical reactions or slight increase or diminution. But when the lesion suddenly destroys the nucleus, or damages the nerve trunk, then we get not only quantitative but also qualitative changes—the reaction of degeneration as just described—the qualitative changes being both modal and serial.

In *cerebral paralyses*, then, we have no qualitative electrical changes, no loss of Faradic irritability in the

nerves of the paralysed limbs, and no modal or serial alteration in galvanic reactions.

If the reaction of degeneration is present in the area of distribution of a cranial nerve we know at once that the lesion is either in its nucleus or in the trunk of the nerve.

In diseases of the *spinal cord* no qualitative changes occur unless the trophic nuclei of the nerves and muscles in the anterior cornua are affected. So that it is only in polio-myelitis anterior acuta, and in myelitis implicating these centres, that the reaction of degeneration occurs ; when the lesion in the anterior cornua is a chronic one the reaction of degeneration is not usually present, *e.g.*, progressive muscular atrophy.

In lesions of the *peripheral nerves* the reaction of degeneration occurs in its typical form. But in these cases, if the lesion is slight, there may be only slight diminution in the Faradic irritability without qualitative changes. If then, in paralysis due to peripheral neuritis from any cause, the reaction of degeneration is not present, the injury is slight and recovery will quickly ensue. If the reaction of degeneration is present then the injury is severe ; the case will be a tedious one, but may recover. If the galvanic irritability of the muscles is lost, then the case is hopeless.

We can at once see the great use of electricity in the diagnosis of the seat of the lesion. The reaction of degeneration at once tells us that the

lesion is either in the nerves, or in the anterior cornua of the cord, or in the trophic nuclei in the case of the cranial nerves.

Electricity is also of immense service in enabling us to form a prognosis as above indicated. If we find qualitative electrical changes we are sure of organic disease, but of course organic disease may be present without any alteration in the electrical reactions. We can distinguish trance from death by the use of electricity, for in the latter condition Faradic irritability is lost in two or three hours.

Qualitative electrical changes may precede paralysis, and exist without it ; in cases of plumbism electrical changes are often present before drop wrist occurs.

The reaction of degeneration (R D) is characterised both by qualitative and quantitative alterations. The following changes constitute it :—

1.—As the nerve undergoes degeneration its Faradic and Galvanic excitability diminishes, and at the end of about fourteen days is entirely lost.

2.—The Faradic current applied to the muscle causes no contraction, the intra-muscular nerves having degenerated, and there being no idio-muscular Faradic irritability.

3.—During the first ten days from the onset of the disease, or from the time of injury, the galvanic irritability of the muscles is diminished ; then it is increased to slowly interrupted currents ; and

0 1 2 3 4 5 6 7 8 9 10 11 12 weeks.

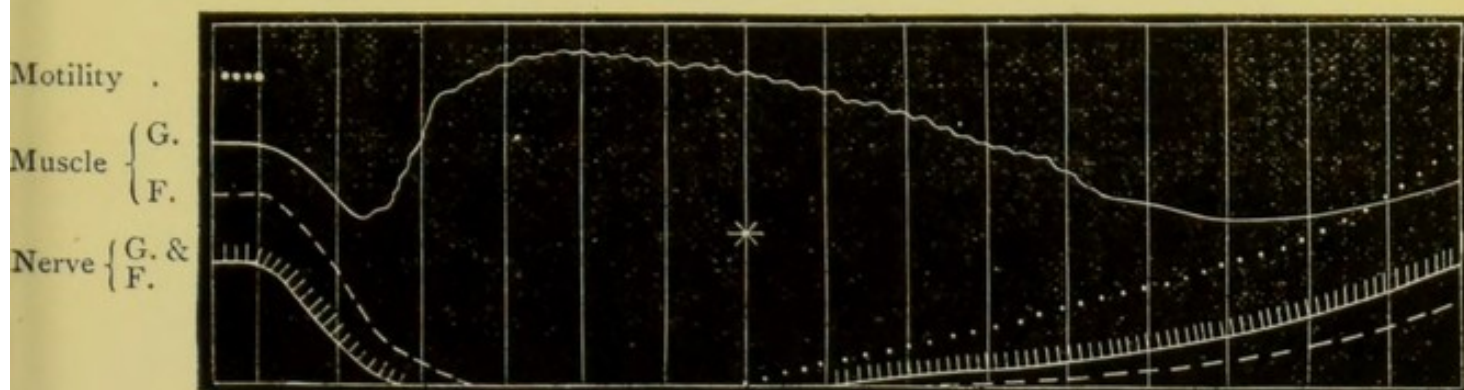


Diagram illustrating reaction of degeneration, with rapid recovery (Erb).

FIG. 14.

0 1 2 4 6 10 15 20 25 30 35 40 45 50 weeks.

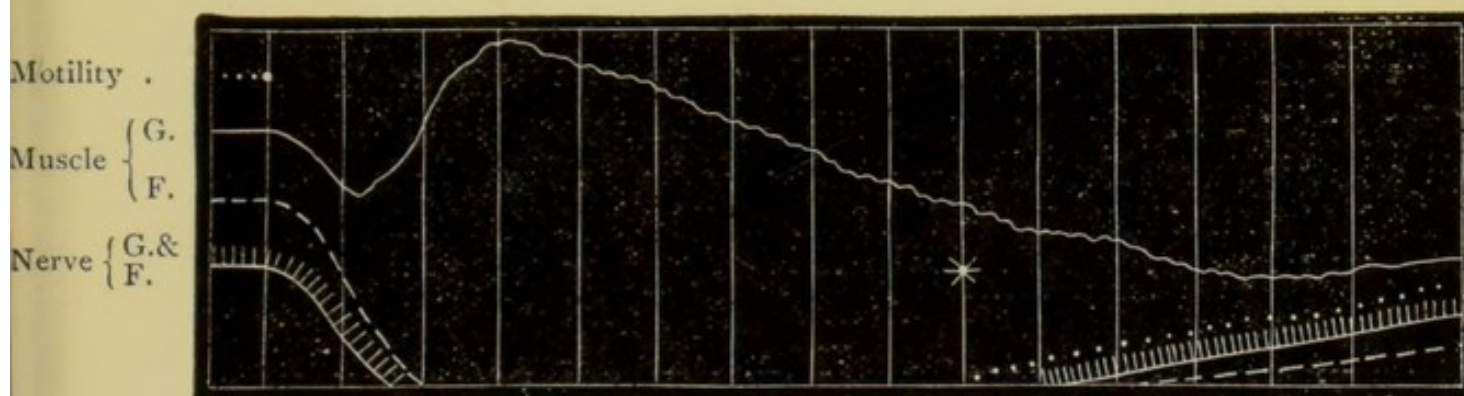


Diagram illustrating reaction of degeneration, with slow recovery (Erb)

FIG. 15.

0 1 3 10 20 30 40 50 60 70 80 90 100 weeks.

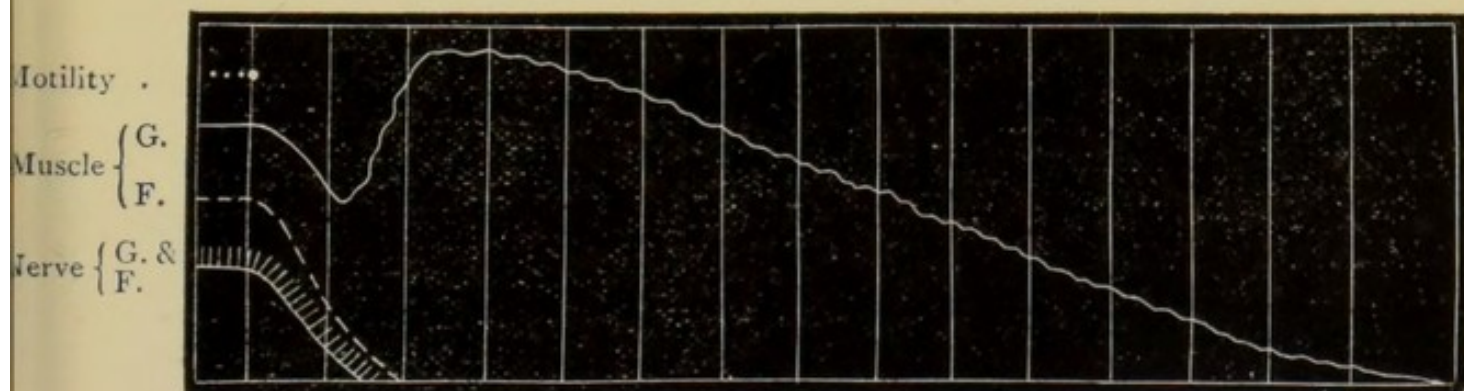


Diagram illustrating reaction of degeneration where recovery does not occur (Erb).

FIG. 16.

4.—The following serial qualitative changes are observed:—The sequence of the polar reactions is altered, the anode taking the place of the cathode, and the order of the contractions with currents of increasing intensity is

$$\left. \begin{array}{l} 1.—A\ C\ C \\ 2.—C\ C\ C \\ 3.—C\ O\ C \\ 4.—A\ O\ C \end{array} \right\} \text{ in place of } \left\{ \begin{array}{l} 1.—C\ C\ C \\ 2.—A\ C\ C \\ 3.—A\ O\ C \\ 4.—C\ O\ C \end{array} \right.$$

5.—The following modal changes are also observed:—The character of the contractions is modified; instead of being short, sharp, and sudden, they are slow, prolonged, and apt to become tetanic; and the contractions are produced only by slow interruptions of the current.

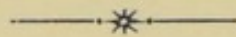
The reaction of degeneration only occurs when the trophic centre of the nerve, or the nerve itself, is injured. Hence it is present—(1) In lesions of the anterior cornua of the cord; (2) In lesions of the peripheral nerves, from injury, plumbism, alcohol, &c.

A variety of R D has been described by Erb where the muscles alone display quantitative and qualitative galvanic modifications, whilst their Faradic excitability (through the intra-muscular nerves) is preserved, and the nerve continues to respond normally or subnormally to both currents. The nerve remains intact, while the muscles rapidly degenerate. Trophic changes may occur either in the nerve or muscle alone. This variety of R D is present in many cases of facial paralysis and in pseudo-hypertrophic

In using Galvanism and Faradism for diagnostic purposes we usually compare the contraction produced on the diseased with that produced on the healthy side, the electrodes, condition of moisture, and points of application being the same. If both limbs are affected we compare the reactions with those obtained on a healthy individual, but it must be remembered that the resistance offered by the skin and tissues may vary on the two sides of the body, and is different in different individuals. The only way in which we can measure this resistance, and so distinguish between a diminished response due to increased resistance and one due to disease, is by the Galvanometer ; by its use we can ascertain the strength of current circulating in any given circuit.

CHAPTER VII.

TROPHIC DISORDERS.



Trophic disorders of the various tissues frequently occur in diseases of the nervous system, there being a nervous mechanism presiding over the nutrition of the tissues.

Trophic disorders of the *skin*, such as patches of erythema, herpetic eruptions, bullæ, and œdema, are frequently present in lesions of the peripheral nerves. Zoster may occur in locomotor ataxia or in meningitis.

Purpura is frequently observed about the legs of tabetic patients, more especially when the pains are severe. The acute bed-sores that occur over the sacrum in acute myelitis and over the buttock in hemiplegia are well known.

Glossy skin, first described by Paget, is observed after severe lesions of the peripheral nerves. When it affects the fingers they are tapering, smooth, shiny, hairless, and often hyperæsthetic.

Pigmentation or bleaching of the skin may also occur in lesions of the peripheral nerves.

Raynaud's disease is symmetrical gangrene and local asphyxia, affecting the fingers and toes, tips of the nose and ears.

The extremities are habitually cold, and the circulation feeble. Acute attacks occur in which the extremities become white, cold, and bloodless; the fingers and toes looking like wax. After a time the parts become congested and painful; bullæ containing blood and ulcers form on the affected parts, and gangrene with loss of the terminal phalanges may occur. This disease is supposed to consist in ischæmia of the smaller arteries, produced by irritation of the vaso-constrictor nerves, but in many cases the peripheral nerves are diseased. Dr. Barlow recommends Galvanism of the affected extremities in the subjects of this neurosis.

Perforating ulcer of the foot occurs in tabes dorsalis and in diseases of the peripheral nerves, in fact in the former disease a peripheral neuritis is probably the cause of this affection. A large corn nearly always precedes the ulceration. The skin around the sinus is very often anæsthetic, but not always so, and the sinus frequently leads down to dead bone, the joints of the great toe being often destroyed and the phalanges necrosed. The favourite seat of perforating ulcer is on the under surface of the metatarso-phalangeal articulations, and both feet may be affected. Similar ulcers have been observed on the hands in cases of tabes, where the cervical region was most affected. Perforating ulcer of the foot may be the first symptom of tabes.

In a case at the Queen's Hospital a young girl had lost the terminal phalanges of the toes of one foot successively. She had a perforating ulcer on the under surface of the metatarso-phalangeal joint of the great toe, and anæsthesia of the peripheral half of the foot. Disease of the sciatic nerve was diagnosed, and this was confirmed, for when the nerve was exposed for the purpose of stretching, it was found to be much atrophied.

The *nails* may fall off in peripheral nerve disease and in tabes; they are, moreover, frequently discoloured, ridged, and brittle.

The *hair* has been known to turn grey suddenly, in a single night, after severe emotion.

Perspiration may be excessive or arrested. Occasionally there is unilateral perspiration. These disturbances depend upon lesion of the sympathetic nerve or of the cerebro-spinal centres connected with the sympathetic.

The trophic nerves to the skin are distributed with the sensory fibres, for it is in such diseases as locomotor ataxia where pains are severe that these lesions occur; moreover, in lesions of the anterior cornua, where there is no sensory disturbance, the skin is unaffected. Just as the nerve cells in the anterior cornua of the cord stand in trophic relation with the muscles and joints, so the posterior cornua of the grey matter exert some influence over the nutrition of the skin.

Trophic disorders of the *viscera* are occasionally observed.

Lesions of the vagus nerve are said to give rise to pneumonia and fatty heart.

Cerebral hæmorrhage is said to be frequently associated with pneumonia of the lung on the side opposite the lesion. I have twice observed pneumonia on the side of the hemiplegia after cerebral hæmorrhage. Acute cystitis and renal congestion commonly occur in acute myelitis.

The *joints* and *bones* have a trophic relation, it is supposed, with the anterior cornua of the cord. Lesions of the peripheral nerves of an irritative kind are apt to cause acute inflammation of the joints supplied by the nerves, and anchylosis may result.

Trophic disorders of the joints and bones frequently occur in *tabes dorsalis*, and are attributed by Dr. Buzzard to disease in the medulla oblongata from their frequent association with gastric crises. Acute synovitis may occur at the onset of an attack of poliomyelitis anterior acuta.

Fractures of the bones, produced by the slightest movements, have been observed by Charcot in *tabes dorsalis*.

In spastic hemiplegia occurring in early life the growth of the bones is often arrested, the limbs being stunted. It is important to remember that the bones of the insane undergo morbid changes, rendering them so brittle that they are readily fractured from trivial causes. We frequently hear of

inquests in these cases, and the attendants might suffer unjustly unless medical men know of this change.

A man suffering from locomotor ataxia or insanity should be handled, therefore, with the greatest care.

The *teeth* are also occasionally affected in tabes. A man in the workhouse infirmary suffering from tabes went insane, and used to pull out his teeth and throw them about the ward. He died in an asylum after having many of his ribs broken.

The various *glandular* organs of the body are greatly influenced by disorders of the nervous system.

Unilateral progressive atrophy of the face is a remarkable trophic disorder, probable depending upon disease of the trophic fibres of the fifth nerve. It is more common in women than in men, and more commonly affects the left side of the face. All the tissues, except the muscles, atrophy on one side of the face; namely, the skin, fat, hair of the head, beard, eyelashes, bones, and occasionally the muscles of mastication. The disease may be mistaken for congenital asymmetry of the two halves of the face; but the atrophy of the skin, hair, and bones, with diminished bulk, and its supervention years after birth, distinguish it.

Case of Progressive Unilateral Facial Atrophy.

E. C., a woman, aged 42, attended at the Queen's Hospital for treatment for dyspepsia a few weeks ago. Observing that there was marked facial

deformity I enquired as to its commencement; she stated that up to her 17th year she was all right, but that after that age the face became gradually affected, the left side being observed to be different from the

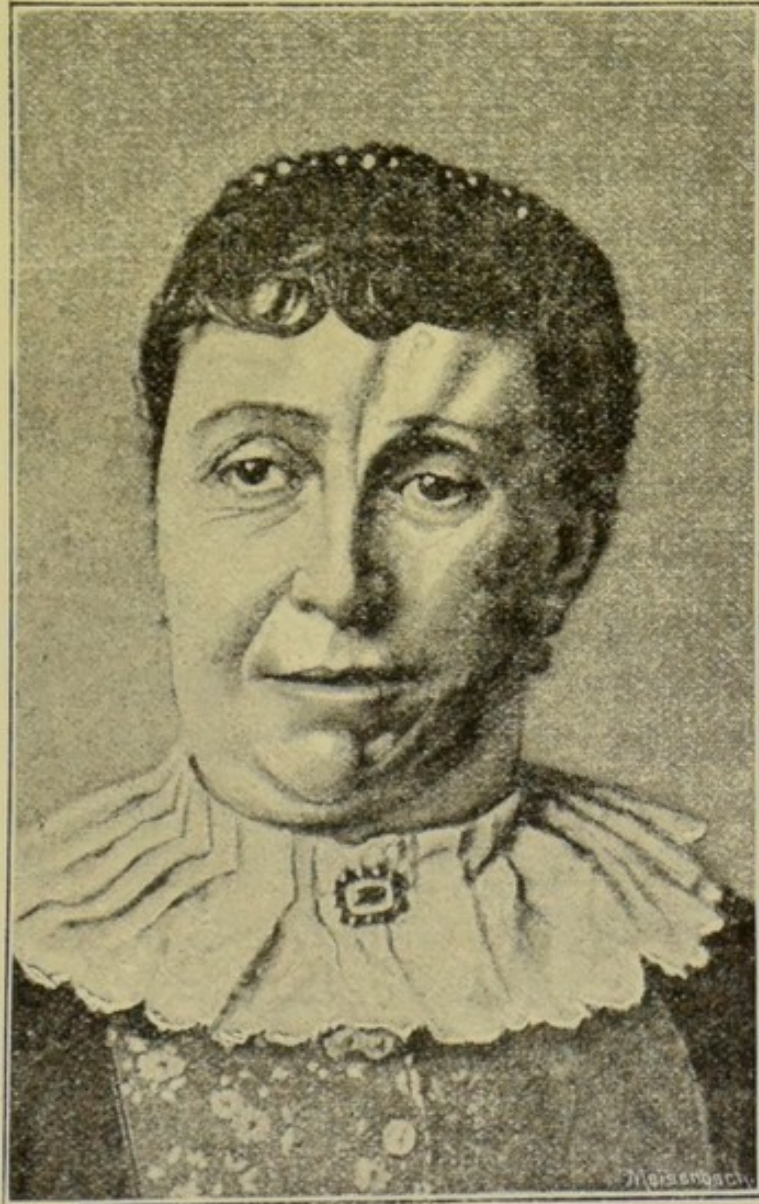


FIG. 17.

HEMIATROPHIA FACIALIS.

other. She has had four children, and she thinks that her face has got worse with the birth of each child. There is no history of syphilis or of any injury.

She has suffered much from tooth-ache, many of the teeth of the upper jaw being carious, but there is no difference in the teeth on the two sides. There is no history of spasm, hyperæsthesia, or neuralgia of the left side of the face, and her general health has always been excellent. There is marked asymmetry of the face, the contrast between the right and left halves being very pronounced. The left half is altogether smaller, there being an abrupt line of demarcation running vertically down the centre of the face, the contrast on the chin being especially apparent, the right half ending in a distinct ridge.

There is no alteration in the colour of the skin or of the hair, but the eyelashes on the left side are scanty. There is alopecia over the left half of the front of the skull, extending to the middle line in the one direction, and to the temporal ridge in the other, and as far backwards as the vertex; the alopecia being confined to the area of distribution of the fifth nerve. The fat seems to have completely disappeared on the left side, the eye being sunken and the palpebral fissure smaller than on the right side. The malar bone is very prominent, and the fossæ above and below very deep. The tongue, soft palate, and vault of the hard palate are unaffected. There is no paralysis, but the left masseter seems much smaller than the right, though the patient has no difficulty in masticating on the left side. The bones are much atrophied, the left malar bone being about half the size of the right. On the left side

the measurement from the angle of the lower jaw to the symphysis is $2\frac{1}{2}$ in., on the right side $3\frac{1}{2}$ in. The depth of the jaw, from the summits of the molar teeth to the lower margin, on the left side is $1\frac{1}{2}$ in., on the right side 2 in.

On the left half of the forehead are two vertical grooves separated by a ridge, these grooves being in the situation of the supra-trochlear and supra-orbital nerves.

There is no alteration in sensibility. The temperature of the left side of the face is 97.2° F., of the right side 96.4° F.

The patient brought her photograph, taken when she was 17 years of age, and in this there was no trace of any deformity. The Faradic response on the left side is considerably greater than on the right, but this is undoubtedly due to the diminished resistance on this side, the skin being thin and without subcutaneous fat. This affection is a rare and interesting one, and is supposed to depend upon a lesion of the trophic fibres of the fifth nerve. It is slowly progressive and incurable, but does not affect the general health.

CHAPTER VIII.

DISEASES OF THE SPINAL CORD.



The Anatomy and Physiology of the Spinal Cord.

The spinal cord, the great conductor of motor impressions downwards from the brain and of sensory impressions upwards to the brain, may be said to consist of a series of superimposed segments ; each segment having a pair of spinal nerves attached to it, and consisting of two exactly symmetrical halves. A transverse section of the cord is seen to be divided into two halves by the anterior and posterior median fissures, each half being again divided by secondary fissures into certain columns. The anterior columns of the cord extend laterally from the anterior median fissure to the anterior roots of the nerves. The inner portions of these columns constitute the direct pyramidal tracts which contain those fibres of the motor tract from the brain which have not decussated in the medulla. Some believe that the fibres of the direct pyramidal tract decussate in the cord, so that there is

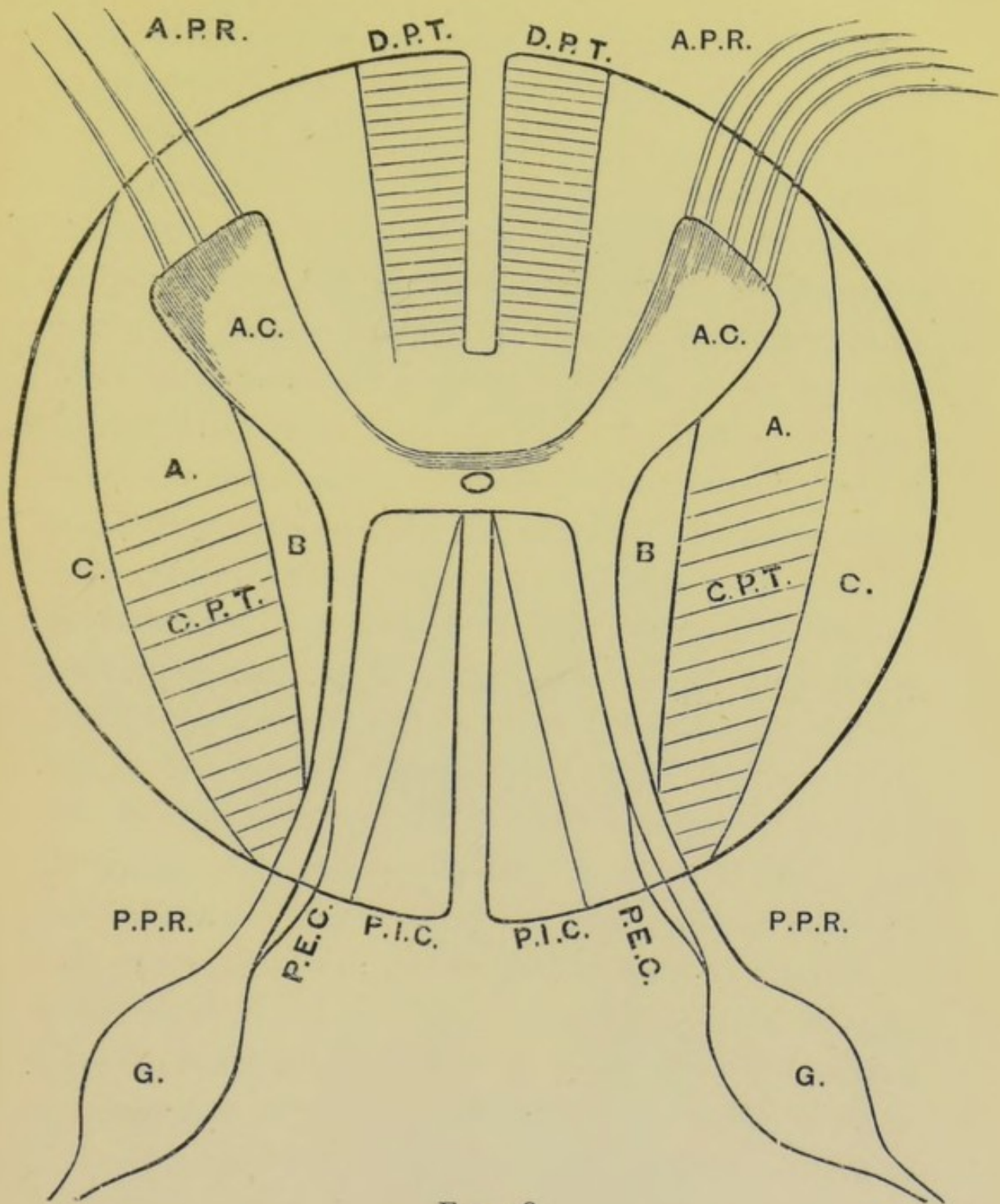
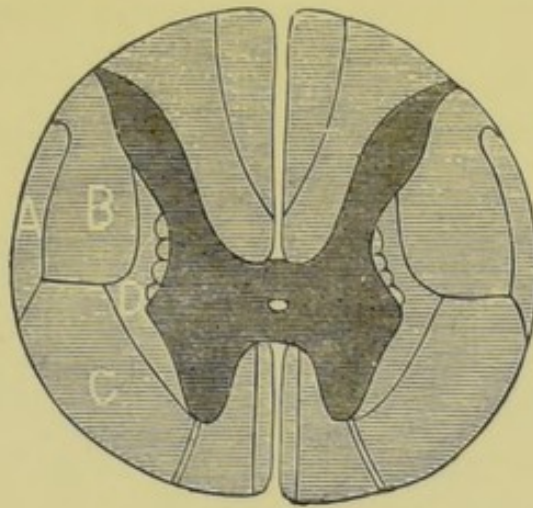


FIG. 18.

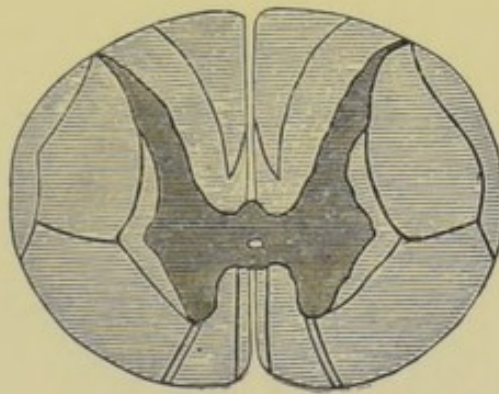
DIAGRAMMATIC REPRESENTATION OF TRANSVERSE SECTION OF THE SPINAL CORD.

A. P. R., anterior primary nerve roots. P. P. R., posterior primary nerve roots. D. P. T., the direct pyramidal tracts. C. P. T., the crossed pyramidal tracts. P. I. C., the postero-internal columns, or the columns of Goll. P. E. C., the postero-external columns, or the root zones, traversed by fibres of the posterior nerve roots. A. C., anterior cornua of the grey matter. G., ganglion on the posterior roots of the nerves. C., the cerebellar tracts. A., region of lateral column containing sensory fibres. B., region of unknown function separating the crossed pyramidal tract from the grey matter.

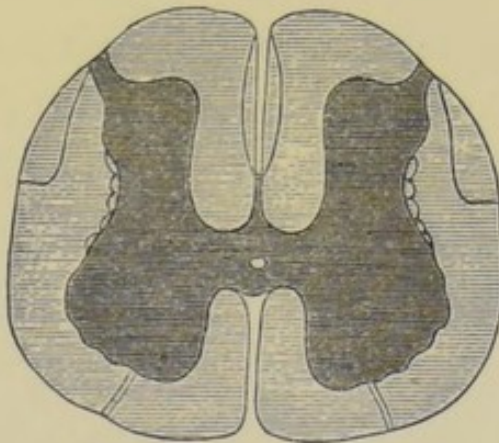
finally a total decussation of the motor fibres from the brain. Dr. Hughlings Jackson, however, believes that the direct pyramidal tracts do not decussate, but that the fibres pass to the cells in the anterior cornua and thence to the motor nerves. The direct pyramidal tracts cease about the middle of the dorsal region. The lateral columns of the cord extend from the anterior roots of the nerves to the posterior roots, and are divided into four distinct areas by Flechsig. A tract on the surface, the cerebellar tract, conducts fibres upwards from the muscles to the cerebellum, for this tract undergoes upward degeneration, after lesions of the posterior nerve roots, the degeneration implicating the cerebellum. The most important tract in the lateral column is the crossed pyramidal tract, which is formed by upwards of 97 p.c. of the fibres of the motor tract of the opposite side of the brain. This tract is separated from the surface of the cord by the direct cerebellar tract. It diminishes in size as it passes downwards, and comes to the surface in the lumbar region, where it is situated external to the apex of the posterior horn of grey matter. There is a layer separating the grey matter from the crossed pyramidal tract, whose function is unknown, and another layer in front of the pyramidal tract, which is probably sensory, for ascending degeneration in this region is said to occur after lesions of the posterior nerve roots. Dr. Gowers thinks that sensations of pain are conducted upwards in this layer.



CERVICAL.



DORSAL.



LUMBAR.

FIG 19.

SPINAL CORD (Flechsig).

The lettered portions are the four regions of the lateral columns.

The posterior columns extend from the posterior roots to the posterior median fissure; each posterior column is divided into an inner portion, the postero-internal column (column of Goll) which conducts tactile impressions upwards and which undergoes ascending degeneration after lesions of the sensory roots; and an outer portion, the postero-external column or root zone of Charcot, which is of great importance, in that it contains fibres of the posterior roots of the nerves passing on to the grey matter, and is the seat of the lesion in locomotor ataxia.

The fibres of the posterior roots of the nerves, after leaving the ganglia, in great part pass directly into the posterior cornua of the grey substance; some, however, pass through the postero-external columns on their way to the grey substance.

The sensory fibres decussate in the spinal cord almost immediately in the cervical and upper dorsal regions, but not immediately in the lower dorsal and lumbar regions, as has been pointed out by Gowers. A transverse lesion of one half of the cord in the lower dorsal or lumbar region may cause anæsthesia in the lower extremity of the same side.

According to Ferrier, the fibres concerned with the function of muscular sense also decussate in the cord, but in a well-marked case of hemilateral myelitis recently under my care, muscular sense was impaired on the side of the lesion, thus upholding Brown Séquard's dictum.

The large multipolar nerve cells in the anterior cornua are—

- 1.—The centres for the various reflex actions of the cord ;
- 2.—The trophic centres for the motor nerves and muscles ; and possibly
- 3.—Preside over the nutrition of the joints.

The rest of the grey matter probably exerts a trophic influence upon the skin and the viscera.

As in the brain, so in the cord, the sensory tract is much less defined than the motor, and probably a very thin strand of grey matter is sufficient to conduct sensory impressions upwards. In a transverse myelitis, motor power is sooner and more completely lost than sensation. Sensation is more rapidly recovered than motion.

Vaso-motor centres, vaso-constrictor, and vasodilator nerves exist in the cord, but their situation is unknown.

The centre for the dilatation of the pupil is situated in the lower cervical region of the cord.

The fibres of the pyramidal tracts have their trophic centre in the motor convolutions of the brain. Lesions severing this connection will be followed by descending sclerosis of the pyramidal tracts, both crossed and direct ; there are, therefore, four tracts in which descending degeneration occurs after a transverse lesion of the cord.

The fibres of the posterior roots of the nerves have their trophic centres in the ganglia ; lesion of

these fibres between the ganglia and the cord, or in their passage through the postero-external columns, as in locomotor ataxia, is followed by ascending degeneration of the postero-internal and cerebellar tracts, and also of the anterior region of the lateral columns.

The two halves of the cord, though to a great extent independent, especially in the cervical region, are yet in intimate relation with one another. In cases where hemiplegia has occurred in early life it is frequently found that the patient cannot open or close the sound hand without the one on the affected side following suit, and in old cases of hemiplegia where the leg does not recover the opposite leg suffers also.

There is no doubt an intimate connection between the multipolar nerve cells on the two sides of the cord.

The Reflex Actions.

The reflex actions may be conveniently arranged in three divisions—

- 1.—The organic reflexes.
- 2.—The superficial or skin reflexes.
- 3.—The deep or tendon reflexes.

The organic reflexes have their centres in the spinal cord and medulla oblongata.

In the medulla are situated the respiratory, the vaso-motor, the cardio-inhibitory, the deglutition, the vomiting, the diabetic, and salivary centres.

In the lumbar region of the cord very important organic centres are situated, namely—the micturition,

the defæcation, the sexual, and parturition centres, which are reflex processes.

The vesical reflex is a highly important one, and it is frequently deranged in diseases of the spinal cord. When the bladder becomes full of urine the sensory nerve endings in the mucous membrane are stimulated, and impressions are conveyed along the sensory nerves to the micturition centre in the lumbar region of the cord. Impressions are also conveyed to the brain, which excite the desire to micturitate.

The brain may inhibit or set in action the micturition centre.

If micturition occurs, that part of the centre which controls the sphincter vesicæ is inhibited, and that which controls the detrusor stimulated; the abdominal muscles are also made to contract.

The proper performance of the act of micturition requires then the integrity of—

- 1.—The sensory nerve endings in the mucous membrane of the bladder.
- 2.—The sensory nerves from the bladder to the cord.
- 3.—The micturition centre in the cord.
- 4.—The efferent nerves to the sphincter and detrusor muscle (fourth sacral).
- 5.—The integrity of these muscles.
- 6.—The connections between the brain and the micturition centre.

In lesions of the brain impairing consciousness, incontinence of urine is observed, but this incontinence is intermittent; the reflex function goes on, micturition being brought about whenever sufficient urine collects in the bladder to sufficiently stimulate the sensory nerves; but there is lack of cerebral control.

The involuntary discharge of urine in coma does not imply any paralysis of the bladder.

A transverse lesion in the cord above the lumbar region does not paralyse the bladder, but by breaking the connection between the micturition centre and the brain causes the act to be purely reflex and involuntary.

Lesions, however, in the lumbar region of the cord destroying the reflex centre cause paralysis of the bladder.

The detrusor and sphincter muscles have separate centres, and one only may be partially or completely paralysed.

The fibres connecting the micturition centre with the cerebral convolutions probably pass along the lateral columns, for in primary lateral sclerosis the patient has little power to initiate or arrest the act of micturition, the process becoming a purely involuntary reflex action. The same remarks apply to the other organic reflexes situated in the spinal cord, although more is known concerning the process of micturition than is known of the other reflex actions.

The superficial and tendon reflexes, like the organic, are of great importance in the diagnosis of diseases of the spinal cord. Their presence indicates the integrity of their reflex paths through the cord, but disease may of course be present in the cord outside the reflex path (which is formed by the posterior nerve root fibres, the grey matter and anterior nerve root fibres) without abolishing the reflexes.

The lateral columns, the postero-internal, and the anterior columns may be destroyed without any injury to the reflex tract.

Absence of a reflex is not a certain indication of disease of the cord for—

(*a.*) Many reflexes, such as the gluteal, epigastric, and interscapular may be absent in health.

(*b.*) Disease anywhere in the reflex path outside the cord, either of the motor nerves and muscles or of the sensory nerves, will abolish the reflex.

The different reflexes pass through the cord at different levels, so that we may test the integrity of the cord throughout its length. If a reflex be absent, the one immediately below and that immediately above being present, then we have strong evidence of disease in the area of this reflex, either in the cord or peripheral nerves.

Loss or diminution of the reflexes may be due to (1) disease in the reflex path; (2) to increase of cerebral controlling influence; (3) to diminution or abolition of muscular irritability.

Increase of the reflexes may be due (1) to increased irritability and diminished resistance in the reflex arc, as produced by strychnia; (2) arrest of cerebral controlling influence conducted down the lateral columns, hence the exaggeration of all the reflexes observed in lateral sclerosis; (3) increase of the irritability of the muscular fibres; hence the exaggeration of the reflexes in wasting diseases.

The *superficial reflexes* are true reflexes. They are produced by stimulation of the skin or mucous membranes.

1.—The *plantar reflex* obtained by irritating the skin of the sole, the muscles of the foot contracting, passes through the lower end of the cord. In cases of neuritis of the great sciatic nerve this reflex is absent on the affected side. I have met with two or three cases of chronic neuritis of this nerve for which I could discover no cause.

2.—The *gluteal reflex* consists in contraction of the gluteal muscles, brought about by irritating the skin over the buttock. It passes through the cord at the level of the fourth and fifth lumbar nerves. This reflex is rarely to be obtained in the healthy subject.

3.—The *cremasteric reflex* by which the testicle is drawn up when the skin on the inner side of the thigh is irritated, passes through the cord at the level of the first and second lumbar nerves. This reflex is pretty constantly present in health. It is, however, more lively in boys than in men.

4.—The *abdominal reflex* consists in contraction of the abdominal muscles upon irritating the skin on the side of the abdomen in the nipple line. This reflex passes through the cord from the eighth to the twelfth dorsal nerves. It is frequently absent in health.

5.—The *epigastric reflex* consists in the dimpling of the epigastrium caused by contraction of the upper fibres of the rectus produced by stimulation of the side of the chest in the fifth and sixth spaces. This reflex passes through the cord between the fourth and sixth dorsal nerves. It may be absent in health.

6.—The *erector spinæ reflex* consists in a contraction of these muscles caused by stimulation of the skin from the angle of the scapula to the iliac crest. This reflex is rarely present in health, but is frequently present in wasting disease, and its production depends upon the integrity of the cord in the dorsal region.

7.—The *interscapular reflex* is produced by stimulation of the skin between the scapulæ, and consists in contraction of some of the scapular muscles. This reflex is frequently absent in health. It passes through the cord at the level of the upper two or three dorsal and lower two or three cervical nerves.

8.—The *palmar reflex*, consisting of a contraction of the flexors of the fingers induced by stimulating the skin of the palm, and is only obtainable in young infants. It is not obtainable during the waking hours

after this period of life, the hand being much more under cerebral control than the foot. This reflex passes through the cervical enlargement of the cord.

9.—*Cranial reflexes.* The chief reflexes of the cranial nerves are :—

(a.) The conjunctival reflex caused by irritating the conjunctiva. This is lost in peripheral lesions of the facial nerve and of the ophthalmic division of the fifth nerve.

(b.) The reflex contraction of the iris to light.

(c.) The palatal reflex, the soft palate being elevated when the mucous membrane covering it is stimulated. This reflex is lost in paralysis of the soft palate on one or both sides, according as the paralysis is uni- or bi-lateral. It is also frequently lost after diphtheria without actual paralysis.

The act of sneezing is a reflex caused by irritation of the mucous membrane of the nose.

These cranial reflexes unlike some of the skin reflexes are constantly present in health and their absence implies disease.

The *deep reflexes* consist of contractions of muscles produced by striking their tendons or by suddenly stretching the muscles in other ways.

The knee-jerk and ankle clonus are the best known of the tendon percussion contractions.

The *knee-jerk*, or *patellar-tendon-reflex*, or the *knee-phenomenon* (so named by Westphal, who first perceived its great importance), is probably not caused by reflex but by direct action, and is due to a sudden

stretching of the muscle, but the integrity of the reflex loop through the lumbar region of the cord is essential for its production.

Dr. Gowers has proposed the term "myotatic contraction," as being more appropriate than the term "deep reflex." For all practical purposes we may regard the knee-jerk as a reflex, for a lesion anywhere in the reflex path through the lumbar region of the cord will abolish the phenomenon.

In obtaining the knee-jerk it is important to uncover the knee and to percuss the bare skin. I have once or twice found the knee-jerk to be present where medical men had supposed it to be absent because they had percussed the tendon through the clothes.

The knee-jerk when diminished, but not entirely abolished, can be more easily obtained by telling the patient to clench his fists tightly at the moment the tendon is struck, or to link the fingers together and pull energetically.

In 1883, Dr. Ernst Jendrassik, of Buda Pesth, showed that muscular exertion, as lifting weights, clenching the fists, &c., increases the response to a blow on the patellar tendon. It has since been found that every muscular exertion increases the tendon reactions. This increase is explained by supposing that the will force is not confined to one channel, but that there is an overflow affecting all the spinal ganglia and momentarily increasing the

muscle tone, a single muscular contraction bracing up every other muscle in the body through the overflow it sets in movement.

Loss of the knee-jerk is pathological, and indicates disease somewhere in the reflex path. The knee-jerk is rarely if ever absent in health, and is never absent in hysteria.

In a case of hysterical anæsthesia of both legs, recently under my care, the knee-jerk was lost. The patient, a woman, was in the infirmary for two years, and all my efforts to remove the anæsthesia were unavailing. However, after leaving the infirmary she applied leeches to her legs, and returned with the anæsthesia gone, but still having loss of knee-jerk and other symptoms of locomotor ataxia.

The knee-jerk is absent—

1.—In locomotor ataxia, the lesion here interrupting the reflex in the root zone. Loss of the knee-jerk may be the first symptom of this disease, and may precede all other symptoms for years. Simple loss of the knee-jerk should always excite grave anxiety as to the future.

2.—In lesions affecting the lumbar region of the cord, in meningitis, myelitis, tumour, or other lesion implicating the anterior or posterior nerve roots or the grey matter; and in lesions of the anterior cornua, as in infantile paralysis.

3.—In disease of the afferent and efferent nerves of the reflex; hence in peripheral neuritis, when the

branches of the lumbar nerves are involved. The knee-jerk is lost, *e.g.*, in—

- (*a.*) Alcoholic paralysis.
- (*b.*) Diphtheria, even without actual paralysis.
- (*c.*) Diabetes.
- (*d.*) Peripheral neuritis from other causes, such as injury, cold, gout, lead poisoning.

4.—In pseudo-hypertrophic paralysis when the disease is advanced.

5.—In cases of tumour of the cerebellum, it is occasionally lost on one or both sides.

6.—In those rare cases of progressive muscular atrophy in which the wasting commences, and is mostly marked in the legs, the lesion here probably being in the peripheral nerves. The knee-jerk is lost somewhat early in this form of progressive muscular atrophy long before the atrophy is extreme.

The knee-jerk is occasionally, but very rarely, present in cases of locomotor ataxia, and in cases of combined sclerosis of the postero-external and lateral spinal tracts, it is exaggerated.

I have a case of locomotor ataxia at present under observation with myosis and Argyll-Robertson pupils, gastric, urethral and rectal crises, and slight ataxy without loss of knee-jerk. The knee-jerk once lost in locomotor ataxia never returns. In peripheral neuritis it may, and its return in diphtheria is hastened by the administration of strychnia.

The knee-jerk is exaggerated (1) in disease of the lateral columns, either primary or in the form of

descending degeneration ; (2) in all other lesions by which the cerebral controlling influence is shut off ; (3) in all cases in which the muscular irritability is exaggerated, or in which the irritability of the grey substance of the cord is increased.

In cases of myelitis (above the lumbar region) or tumour pressing upon the spinal cord and leading to contracture of the lower extremities, the knee being flexed to a marked degree and the rigidity being extreme, the knee-jerk may appear to be absent, but careful observation will show that the rigidity is increased when the tendon is percussed.

Ankle clonus consists of a rhythmical clonic spasm of the foot due to rhythmical contraction of the calf muscles. To obtain it the foot must be suddenly flexed at the ankle, the Achilles tendon being thus forcibly stretched.

Ankle clonus is not present in health or in disease of the extreme end of the cord implicating the roots of the nerves or the grey matter. Its presence denotes disease, or, at any rate, diminution or loss of cerebral control.

The great cause of ankle clonus is (1) sclerosis of the lateral columns, but it may be present in (2) hysterical paralysis and (3) even in pressure on the cord without actual disease, as is observed in cases of spinal caries, where ankle clonus may disappear after rest or the application of a Sayre's jacket.

Wrist clonus.—Other forms of clonus may be obtained, for instance, in cases of hemiplegia, where

descending degeneration has taken place to a marked degree, a wrist clonus may be obtained by sudden hyper-extension of the wrist.

A patella clonus, produced by sudden depression of the patella, may be observed under the same conditions as ankle clonus.

Muscular contractions may also be induced by tapping the bones.

The *masseteric reflex* or *jaw-jerk* has been investigated by Dr. Watteville. It is only slightly marked in health. To obtain it the lower jaw should not be fixed by any voluntary muscular contraction; a flat object, such as a paper knife, is placed over the molar teeth, and a blow, with a percussion hammer or with the finger, struck near the teeth.

In some cases a regular clonus has been obtained, as in the case of amyotrophic lateral sclerosis, published by Dr. Beevor, in "Brain," Vol. VIII.

The latent period of this reflex is very short, too short for the phenomenon to be a true reflex.

I have seen the jaw-jerk well marked in a case of trigeminal neuralgia.

Periosteal and fascial reflexes.—The *front tap contraction*, first described by Dr. Gowers, and consisting of contractions of the quadriceps femoris, produced by tapping the inner surface of the tibia, is a delicate test of morbid irritability, and is not present in health. It depends for its presence upon the same conditions as does the production of ankle clonus.

Bicipital reflex.—In the late rigidity of hemiplegia, tapping the lower end of the radius is followed by contraction of the biceps, the bicipital reflex; tapping of the ulna, by contraction of the triceps.

Tapping the clavicle causes contraction of the deltoid and pectoralis major.

Tapping the spine of the scapula causes contraction of its muscles.

Tapping the lumbar fascia may cause contraction of the erector spinæ muscles.

The early occurrence of these deep reflexes in hemiplegia is of grave omen, indicating extensive damage to the motor tract.

In contrast with the deep reflexes, where we find muscles contracting when put on the stretch, a peculiar condition, first observed by Westphal, occurs occasionally in locomotor ataxia, and has been observed in paralysis agitans; it consists in the contraction of a muscle induced by suddenly approximating its points of origin and insertion. It is best seen in the tibialis anticus.

If dorsal flexion of the foot be either suddenly or gradually produced, this muscle, though greatly relaxed, gets into a state of contraction, its tendon becoming prominent. This contraction may persist for several minutes. Contraction produced by relaxing a muscle was named by Westphal the "paradoxical contraction." It may affect other muscles on the front of the leg in addition to the tibialis anticus.

THE REFLEXES.

I. THE ORGANIC REFLEXES	Respiratory Vaso-motor Cardio-inhibitory Deglutition Vomiting Having their centres in the Medulla Oblongata. Vesical Defecation Parturition Sexual Having their centres in the Lumbar Region of the Cord.
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II. THE SUPERFICIAL OR SKIN REFLEXES:—	<i>Description of.</i>	<i>Level of Cord upon which it depends.</i>
1. Plantar —	Obtained by irritating the skin of the sole, the muscles of the foot contracting. Usually present in health.	The Conus Medullaris (extreme end of cord).
2. Gluteal —	Contraction of gluteal muscles, brought about by irritating the skin over the buttock. Rarely present in health.	4th and 5th Lumbar nerves.
3. Cremasteric —	Testicle drawn up when skin on inner side of thigh is irritated. Usually present in health.	1st and 2nd Lumbar nerves.
4. Abdominal —	Contraction of abdominal muscles upon irritating the skin on the side of the abdomen in the nipple line. May be absent in health.	8th and 12th Dorsal nerves.
5. Epigastric —	Dimpling of Epigastrium caused by contraction of rectus, produced by irritation of side of chest in the 5th and 6th spaces. May be absent in health.	4th, 5th, and 6th Dorsal nerves.
6. Erector Spinæ —	Contraction of spinal muscles caused by irritation of skin from the angle of the scapula to the iliac crest. Rarely present in health.	The Dorsal Region.
7. Interseapular —	Contraction of scapular muscles produced by irritation of skin between the scapulae. Rarely present in health.	Upper two or three Dorsal and lower two or three Cervical nerves.

THE CRANIAL REFLEXES:—

1. The Conjunctival.
2. The Reflex contraction of the iris to light.
The optic being the afferent nerve, the third the efferent nerve, and the centre being placed in the floor of the aqueduct of Sylvius.
3. The Palatal Reflex.
4. The Act of Sneezing.
The cranial reflexes are constantly present in health.

III. THE DEEP OR TENDON REFLEXES.—(The exact nature of these reactions is still a matter of debate):—

<i>Reflex.</i>	<i>Description of.</i>	<i>Level of Cord upon which it depends.</i>
1. The Knee Jerk —	Always present in health.	Depends upon the cord at level of 2nd and 3rd Lumbar nerves.
2. Ankle Clonus —	A series of clonic contractions at the ankle joint, produced by suddenly producing dorsal flexion of the foot. Never present in health.	Depends upon extreme end of cord.
3. Patellar Clonus —	Clonic spasm of Quadriceps femoris, produced by sudden depression of patella. Not present in health.	2nd and 3rd Lumbar nerves.
4. Wrist Clonus —	Not in health.	

PERIOSTEAL REFLEXES:—

1. **Front-tap Contraction**—
Contraction of Quadriceps femoris, produced by tapping the tibia. Not in health.
2. **Bicipital Reflex**—
Tapping the lower end of the radius is followed by contraction of the biceps. Not in health.



CHAPTER IX.

DISEASES OF THE SPINAL CORD

(CONTINUED).



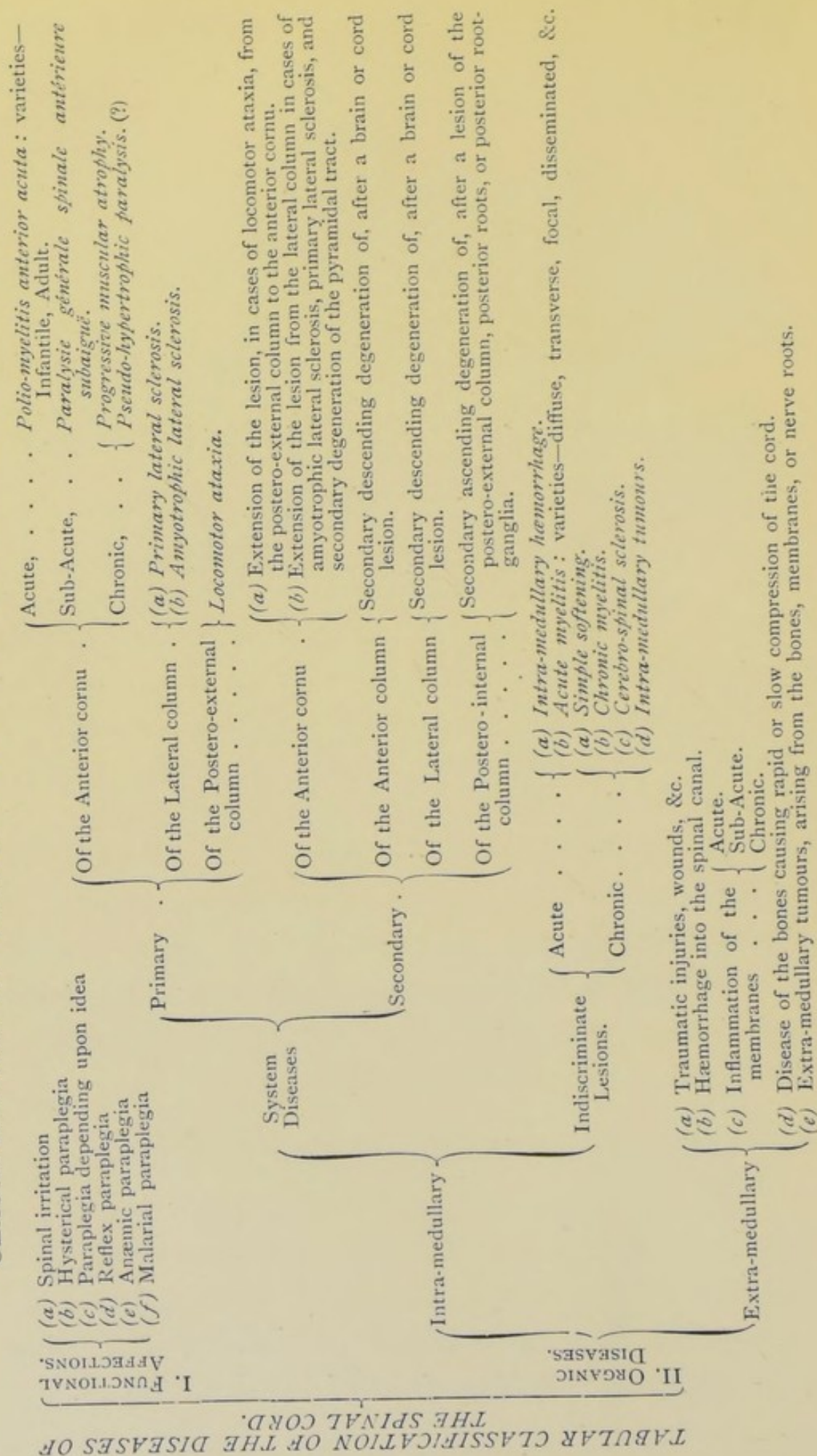
By extra-medullary disease is meant disease, outside the cord itself, of the bone, membranes, or nerve roots, the spinal cord suffering secondarily; and by intra-medullary disease is meant primary disease of the cord.

By system disease is meant the limitation of disease to definite physiological spinal tracts, the lesion being bilateral and symmetrical, and extending a considerable distance along the cord. An indiscriminate lesion affects any part of the cord, and is not limited to any definite tract.

Functional Affections of the Spinal Cord.

Spinal irritation is a functional affection of the spinal cord occurring in women, rarely in men. The subjects of this complaint present various hysterical symptoms, and there is little doubt that the spinal symptoms are manifestations of hysteria also.

CLASSIFICATION OF DISEASES OF THE SPINAL CORD (BRAMWELL).



The characteristic symptom in spinal irritation is pain in the back, and extreme tenderness on even lightly tapping the spinous processes of the vertebræ; with various motor and sensory disturbances in the area of distribution of the nerves given off from the cord in the region corresponding to the tender points. The tenderness may be so great that the patient cannot bear the pressure of her clothes. The patient often complains of neuralgic pains in the limbs. Menstrual disturbance is almost invariably present.

It is important to remember that pain in the back is not a prominent symptom in organic disease of the spinal cord; it is present in disease of the bones and membranes, but it is never so marked as in spinal irritation.

The exquisite hyperæsthesia over the spinous processes, and the pain elicited by the lightest touch, are pathognomonic of spinal irritation, a purely functional disorder.

Hysterical paraplegia is by far the commonest form of hysterical paralysis. It is of the highest importance to remember that paralysis from organic cause may be present in an hysterical subject, and that in all organic affections of the nervous system in women hysterical symptoms are apt to supervene.

It must be remembered that the brain really is the seat of whatever lesion there may be, the power of the will being in abeyance; as Paget has well said, "she cannot will."

In the diagnosis of hysterical paraplegia the following facts must be borne in mind :—

1.—Paralysis of hysterical origin may be gradual or sudden in its onset, often far too sudden to be due to any organic disease of the cord, except intramedullary hæmorrhage, which is exceedingly rare; and it frequently follows a trivial injury or emotional shock.

2.—The sphincters are unaffected.

3.—No trophic lesions, such as local muscular atrophy, bed sores, cystitis, &c., occur. Œdema, however, is occasionally seen in the paralysed or contracted limbs of hysterical subjects, but this œdema, unlike that observed in paralysis from organic cause, varies from time to time, is apt to disappear and recur about the menstrual periods or on emotional disturbances.

4.—Qualitative electrical changes are never met with, but there may be electrical anæsthesia and delayed muscular response.

5.—The symptoms will be found to be incongruous. For instance, there may be complete anæsthesia as well as paralysis, and yet no wasting, no cystitis or paralysis of the sphincters, which would most certainly be present if the anæsthesia were due to organic disease of the cord. The patient may be quite unable to move her legs in bed and yet be able to stand when out of bed.

6.—In hysterical paraplegia there is nearly always rigidity and exaggeration of all the reflexes, both superficial and deep, ankle clonus being well marked.

Primary lateral sclerosis, the symptoms of which are frequently exactly simulated in hysteria, is rare, and very rare indeed in women ; moreover it commences insidiously and lasts for years before complete paralysis ensues, while in hysterical paraplegia the onset is frequently sudden and the paralysis complete. It is important to remember that in time hysterical rigidity may end in sclerosis. Charcot has reported cases of this kind.

Case of Locomotor Ataxia with Hysterical Anæsthesia.

M. C., a woman aged 32, a penmaker, unmarried, was admitted on May 5th, 1883, complaining of difficulty in walking. No family history of nervous disease could be obtained, except that her father had hemiplegia. Three years before she came to the infirmary she had been an in-patient of a large hospital, suffering from severe pains in the legs, which used to occur paroxysmally ; these pains still occasionally occurred. No distinct history of syphilis could be obtained. The patient had great difficulty in walking, the gait being very ataxic ; her body swayed from side to side, and she had to cling to surrounding objects for support ; the ataxy affected the body far more than the legs. The ataxy was rendered worse by closing the eyes, swaying being then well marked

(Romberg's symptom). In the dark she was quite unable to get about. She complained of a sensation of "pins and needles" on placing the feet to the ground. The knee-jerk was abolished on both sides; the plantar reflex was also absent. There was complete loss of sensation in both lower extremities up to the middle of each thigh, analgesia and anæsthesia being absolute; sensation elsewhere was normal. Co-ordinating power in the upper and lower extremities was normal, and she could accurately tell the position of her limbs. She suffered from shooting pains in the legs, and pain in the back. There was no paresis or rigidity of the legs; no spinal or ovarian tenderness. The pupils were unequal; they responded normally to accommodation, but not to light (Argyll-Robertson pupil). Faradisation of the skin of the legs caused no pain, but Faradisation of the muscles caused normal contractions and slight pain. She had recently suffered from attacks of vomiting. She was very emotional, giving considerable trouble during the investigation of her case, and being careless in her replies. She had a typical "facies hysterica" and nictitation. There was no ischæmia in the anæsthetic area, and no vaso-motor or trophic change. There was no history of clavus, globus, or fits. Micturition, defœcation, and menstruation were normal. This case was diagnosed as being one of locomotor ataxia, and the anæsthesia was supposed to be hysterical. For two years the patient was under observation in the infirmary; and isolation,

Faradisation, blisters, and Burq's treatment were tried without any influence upon the anæsthesia. Vertigo was occasionally complained of. On May 11th, 1885, she took her own discharge, and returned to the infirmary on August 26th, 1885. On re-admission it was found that the anæsthesia of the legs had quite disappeared. The knee-jerk was still absent, and the Argyll-Robertson pupil and ataxy with vertigo and occasional pains were still present. She said that while out of the infirmary it struck her that "leeches would make the blood circulate." She applied a leech to each knee and ankle, with the result that sensation was completely restored. The scars of the leech bites could be seen. Localisation, discriminative tactile sensibility, and perception of pain and heat were normal. Faradisation of the skin occasioned pain, but there was some loss of muscular sense. The patient is still under observation with the above mentioned symptoms of locomotor ataxia.

The above case illustrates the important truth that in all organic affections of the nervous system in women hysterical symptoms are apt to appear, and that organic disease of the nervous system may be present in an hysterical subject. Such a group of symptoms as loss of the knee-jerk, the pupil phenomena, pains, ataxy, and vertigo could not be ascribed to hysteria. Loss of the knee-jerk I have never seen or read of in hysteria. On the other hand, the anæsthesia in this case was far more extensive and complete than that usually met with in tabes. Its

hysterical nature was proved by its disappearance immediately after the application of leeches.

Paraplegia depending upon idea.—This affection was first described by Dr. Russell Reynolds. It is a purely functional affection of the brain, in which the patient gets the idea that she is paralysed, and remains so until this idea is negatived.

In both cases an injury was the exciting cause.

The subjects of this form of paralysis belong to the neurotic class of individuals, and not to the hysterical class, the distinctions between which are well marked, and have been graphically described by Dr. Clifford Allbutt.

Ideal paralysis is cured immediately the diagnosis is made, in this respect widely differing from hysterical paralysis, in which the cure is always difficult.

The diagnosis is made by the paralysis supervening in a highly nervous individual after a trivial injury, all signs of organic disease being absent.

Cases of Paralysis Depending upon Idea.

CASE 1.—John Riley, aged 16, was sent to me on March 25 last, by Mr. Jordan Lloyd, complaining of complete paralysis of the left leg, from which he had suffered for two years.

The patient is a bright, intelligent boy, and, according to his mother, is very emotional, good tempered, unselfish, and very well-behaved. The

mother tells me that all the family are "nervous," but I can obtain no history of organic disease. The boy was most particular in relating his case to me, and evidently took notice of the slightest ache or twitch. He attributed the paralysis to an accident two and a half years ago, when he was hit on the inner side of the left ankle by a cricket ball. The joint did not swell, and he was able to walk about after the injury. Two or three weeks after this he knocked his left knee against a chair very slightly; he had pains in the left calf immediately after this injury, and tucking of the left leg, which he says was straightened by a medical man under ether, but which returned with the return of consciousness. He was able to walk about after the second injury until the tucking of the leg came on, when he took to his bed, and one night found that he had lost all use in the limb, the leg, which previously was stiff and tucked, becoming completely flaccid. He never lost control over the urine or fæces. There was no loss of feeling in the limb, but some pain. The paralysis continued unchanged up to the time of my seeing him. He used to see objects green, and at times double. He had never had any previous illness, but was always a nervous and apprehensive boy. Had never had fits or suffered from headache. On examination, the left lower extremity was found to be completely paralysed and flaccid, he could not even move the toes, and there was absolutely no movement at any joint in the limb. The left foot was a

little bluish, but only very slightly colder than the right, the rest of the leg was just as warm as the right. The limb was evidently wasted, but there was no local atrophy.

The circumference of the right leg two inches below the tubercle of the tibia was 13 inches; of the left, $11\frac{1}{2}$ in. The circumference of centre of calf on right side, 12 in.; left, $11\frac{1}{4}$ in. Circumference of thigh 6 in., above patella on right side $15\frac{1}{2}$ in.; left, $13\frac{1}{2}$ in. The plantar reflexes were absent on both sides; there was no ankle clonus; the knee-jerk was rather excessive on both sides; there was no front tap contraction; the cremasteric reflexes were well marked; there was a general slight increase in the reflexes, except that the plantar were absent. Sensation in the paralysed limb was perfect; there were no traces of bed sore or any trophic mischief; no spinal tenderness or deformity; no cerebral symptoms; and no twitchings of the muscles of the face. He had acne on the face, and was evidently anæmic, there being pallor of the mucous membranes, and soft systolic bruits at the base and apex of the heart. He never had suffered from rheumatism. Bowels were constipated.

On testing the nerves, motor points, and muscles of the left limb with a powerful Faradic current, the muscles responded well, only very slightly less than those of the sound side; great pain being caused by the current. With weaker currents contractions were

obtained, only slightly less in intensity than those on the sound side.

After informing him that I should continue giving him the battery till he could move his toes and raise his heel from the ground sufficiently to enable me to pass my fingers under it, he quickly moved them and wriggled his foot till he got his heel on my fingers. Another fact observed greatly aided me in the diagnosis. I made him support himself entirely on his crutches, letting his legs hang freely ; I then pushed the right leg backwards away from the vertical position, making a considerable deflection backwards ; the left leg went back with the right, and did not remain hanging perpendicularly as it would have done had it been paralysed from organic disease.

It will be seen that there were absolutely no signs of organic disease except the wasting of the muscles ; but this was slight and general, and easily explained by the long duration of the paralysis (over two years). I was forced then to conclude that the paralysis was functional. But the boy was not of the "hysterical type." He had no hysterical symptoms, and no variation in the paralysis had occurred ; but was vivacious, good tempered, unselfish, anxious, and intelligent, always nervous and apprehensive. He was evidently of the "neurotic" class of individuals.

Dr. Russell Reynolds has described a form of paralysis depending upon imagination, where there is

no malingering, but the patients are thoroughly convinced that they are suffering from paralysis. This, I believe, is the explanation of the above case. The absence of signs of organic disease, the temperament and mental state of the boy, show it to be a case of ideal paralysis.

Slight paralysis and atrophy of extensor muscles occur in chronic joint diseases, due to changes set up in the cord by the local irritation. In these cases the extensor muscles chiefly suffer, and complete paralysis is rare. Charcot reports a case of a young man who had received an injury to his knee, which was followed by marked paralysis of the extensors of the leg on the thigh. These cases form one variety of reflex paralyses. Was this monoplegia, in the case under notice, of the nature of a reflex paralysis, or was it ideal? The slightness of the injury, which was never followed by any evident joint mischief, the absence of any local atrophy, and the completeness of the paralysis show that it was not due to joint mischief.

After making the diagnosis of ideal paralysis, I informed the boy's mother that there was no organic disease, and that the paralysis would get better very soon. The boy was also told that the leg would get all right, and that he was to use it as much as possible. After one application of a powerful Faradic current, he was able to flex the toes and move the ankle. He attended daily to be Faradised, and in a week was ordered to leave off the use of

crutches and to use a stick. In a few days the stick was also dispensed with. On April 10th (about a fortnight after I first saw him) he walked to the Queen's Hospital without any support, a distance of about two miles. For a few days he limped slightly with the left leg, this was due partly to the wasting, but chiefly to the disuse for the last two years, the various movements of the extremity having to be re-acquired. The patient resumed his employment, and has been perfectly well since.

CASE 2.—Sarah F., a widow, aged 46, was sent to the Queen's Hospital, to be admitted under my care, by Dr. Middleton, of Harborne, on the 15th of March last, and was discharged well on March 28th. She has led a very industrious life, having supported her five children by needlework since her husband's death ten years ago. On October 9th, 1885, she tumbled downstairs, falling down twenty-one steps on to her back. She was much hurt and "dazed," and very much frightened, thinking every bone in her body was broken. She walked about after the accident for a day or two, but suffered from pains in the back, down the legs, and round the body; these pains were intermittent, very severe, and lasted altogether three weeks. Two days after the accident she took to her bed, and a day or two later found that she was paralysed in both her legs, and had lost all feeling in them. Since the accident she had suffered from constant headache and backache. Upon examining her I found that she had a little

power over the legs, but very little. Slight ankle clonus could be elicited on the left side. The plantar and abdominal reflexes were lost, but the knee-jerk was well marked. There was complete analgesia and thermal anæsthesia in both legs up to the knees. She was unable to localise tactile sensations below the knees, the anæsthesia not being complete. When touched with a sharp-pointed instrument she said she felt as if she were being pushed. Muscular sense was unaffected. There was superficial tenderness over the lower dorsal spines, but no pain on firm pressure or on percussion, and no irregularity of the spine. The bladder and rectum were unaffected, and I was informed by Dr. Middleton that she had never lost control over the bladder, nor had any sign of bed sore during her illness. There was no œdema of the legs, no alteration of temperature, and no change in the colour or aspect of the skin or nails. In fact, there were absolutely no trophic changes whatever. The response of the muscles to Faradisation of their nerves was normal, but there was considerable electrical anæsthesia. In summing up the symptoms present, I of course observed their incongruity, if we supposed the case to be one of myelitis, or, in fact, as due to any organic lesion. If the anæsthesia were due to actual lesion of the grey matter, how could the bladder and rectum escape? How was it that there was no sign of bed sore, no œdema, or other trophic change, such as muscular wasting? I diagnosed the case immediately as being purely

functional, and, the woman being of the neurotic type, as one of paraplegia depending upon idea. I at once acted upon this diagnosis, and told the patient that I should give her the battery, which I said would at once remove the loss of sensation and enable her to move her legs as well as she ever did. She was delighted to hear this, and after a minute's Faradisation sensation was completely restored, and she had full command over her legs. I then told her that on my next visit I should expect to see her walking about the ward, for I was sure that she would be able to do so. I instructed Mr. Whittindale, my house physician, to see that she got up after I left, and to apply the Faradic current whenever she displayed any hesitancy. On my next visit, two days later, I found her up and able to walk and run about the ward perfectly well. She was quite delighted at her recovery, and had been most industrious with her needle since getting up. She expressed deep gratitude, and hoped to be able to repay our kindness. I may add that I saw the patient six months later, and she was quite well.

How was the cure effected in this case? Undoubtedly, I think, it was by the mental state of faith or expectation, or both, on the patient's part. She fully believed what I told her, and thus I was able to remove the idea she had that she was paralysed. The essential thing, then, in these cases is to make a correct diagnosis, and then the patients can safely and with success be told that they will quickly

recover. Now, had this been an hysterical woman, I should in all probability have failed to cure her, or at any rate, the cure would have been more tedious. I have previously recorded the case of a woman under my care suffering from locomotor ataxy with hysterical anæsthesia. For two years I did all I could to cure the anæsthesia, and failed; but when she left the infirmary she applied leeches to her legs, and returned with the anæsthesia gone. Isolation, so useful in hysterical cases, is not needed in these.

In the same ward with my case of ideal paralysis there was a young woman suffering from hysterical neuralgia, and the contrast between the two patients was very marked. The hysterical woman was listless and apathetic, did not manifest any desire to be cured, and would scarcely allow that she was any better than when admitted. When told that she was well enough to go back to her duties as a teacher she seemed disappointed, and said that she did not intend to begin work again for some months. The neurotic woman, on the contrary, wanted to leave the hospital immediately she could walk. The hysterical woman was observed to be always sitting opposite the neurotic woman, contemplating her with her hands idly folded in her lap, watching the other busily working.

Such cases as these illustrate the enormous influence of the mind over the body. It is with these cases that the so-called faith healers work pretended miracles. What an opportunity for a faith

healer my two cases would have afforded! Just as the mind has an enormous influence in causing disorders of sensation, of motion, and also of the organic functions, so also its influence can be used as a practical remedy in disease. As Dr. Wilks has well observed, "the practice of medicine is not only one of physic, but of psychology also."

Reflex paraplegia is paraplegia due to irritation of peripheral nerves acting reflexly.

Paralysis, like spasm, may be brought about by irritative diseases of various organs, but especially of the urethra, bladder, or rectum.

The paralysis which is apt to be associated with disease of the bladder or rectum is not always functional or purely reflex.

1.—In some cases myelitis is present, caused by ascending neuritis of the nerves of the diseased organ.

2.—In some cases there is neuritis of the branches of the lumbar and sacral plexuses spreading from the nerves of the diseased organ.

3.—In a third class of cases there is no myelitis or other change in the cord, and no affection of the nerves. These are the cases of true reflex paralysis. We can only diagnose reflex paralysis when there are no signs of organic disease of the cord or nerves and when removal of the peripheral irritation at once cures the paralysis.

The term reflex paraplegia was first used by Brown-Séquard, who supposed that the paralytic

phenomena were due to reflex spasm of the vessels of the spinal cord.

Malarial paraplegia is said to be intermittent, and to occur at intervals, often taking the place of a paroxysm of ague. It is said to be a functional affection of the cord and to be cured by quinine.

Anæmic paraplegia.—The arrangement of the blood vessels in the lower end of the cord fully explains the occurrence of paraplegia in severe cases of anæmia.

The branches from the ilio-lumbar and lateral sacral arteries to the spinal cord are very long and small, and pass along the branches of the cauda equina to the conus medullaris. Hence it is that the extreme end of the cord suffers in anæmia, the blood supply being weakest here. The late Dr. Moxon first pointed this out a few years ago in his lectures on cerebral anæmia and hyperæmia.

A woman who had been confined several weeks previously was admitted into the Queen's Hospital under my care. She had suffered from severe hæmorrhage after her confinement, and the day after getting up, about the tenth day after confinement, she felt numbness and tingling in the feet, and gradually lost the use of the legs.

The paralysis was complete in the feet, much less in the legs and thighs, with flaccidity. Sensation was only slightly affected, and the reflexes were

normal with the exception of the plantar which were abolished.

The sphincters were unaffected and no trophic lesions were present, the preponderance of the paralysis in the feet being fully explained by the feeble vascular supply to the conus medullaris.

This patient only stayed in the hospital a few days, but she recovered some months after.

CHAPTER X.

DISEASES OF THE SPINAL CORD

(CONTINUED).

Organic Affections of the Spinal Cord.



Polio-myelitis Anterior Acuta.

This is a system disease affecting the anterior cornua of grey matter in the cord. There are two varieties—

- 1.—That occurring in infants, and
- 2.—That occurring in adults.

This disease is well known under the name of infantile paralysis or essential paralysis of children.

The following are its characteristic features :—

- 1.—The onset of paralysis is sudden and attended with fever.
- 2.—The maximum of paralysis is at once reached, and subsequent changes are in the direction of improvement.
- 3.—The paralysed limbs are flaccid.
- 4.—There are no sensory disturbances except in rare cases, and no bed sores.

5.—Qualitative electrical changes are present, varying in degree according to the extent of the damage to the motor cells in the anterior cornua.

6.—Rapid wasting of the paralysed muscles occurs.

7.—The bladder and rectum are unaffected.

8.—The reflexes are abolished.

9.—In the infantile variety the bones are arrested in their growth; deformities and contractures are common.

This disease occasionally occurs in adults, and in them deformities, contractures, and arrest of development of the long bones seldom occur.

Nearly all cases of paralysis in children are apt to be put down to this disease.

The following diseases must be carefully distinguished from polio-myelitis anterior acuta :—

1.—Peripheral neuritis.—After specific fevers or from other causes neuritis of certain nerves may occur, which may lead to wasting. I have met with several such cases.

If sensory disturbances, in the case of a mixed nerve, are absent the only way in which a differential diagnosis can be made is by accurately observing the distribution of the paralysis.

If the distribution of the paralysis is an *anatomical* one; that is, if the muscles supplied by certain nerves only suffer, the lesion is in the peripheral nerves.

If, on the other hand, those muscles suffer which are *physiologically associated* or *functionally related*, then the lesion is in the anterior cornua.

Take for instance the muscles that flex the elbow:—Disease of the spinal cord causing paralysis of one of these muscles causes paralysis also of the others, although they are supplied by different nerves; the biceps and brachial anticus being supplied by the musculo-cutaneous nerve, the supinator longus by the musculo-spiral.

2.—Another affection, first described by Duchenne, under the name of “*paralysie obstétricale infantile du membre supérieure*,” or obstetrical paralysis, is occasionally met with in infants.

It is a paralysis of the upper extremity occurring in children whose birth has been effected by instruments or by turning, and it is due to injury to the brachial plexus. There is not complete paralysis of the limb but it hangs immovable in an extended position, and is forcibly rotated inwards. The child can move the hands and fingers but is unable to flex the forearm or to raise the arm.

Usually the paralysis is similar to that form first described by Erb, in which there is simultaneous paralysis of the deltoid, biceps, brachialis anticus, and the long and short supinator muscles, the lesion being one of the fifth cervical nerve.

Two years ago I showed one of these cases before the Midland Medical Society as a case of peripheral monoplegia due to injury of the brachial

plexus at birth. Such cases are to be distinguished by their existence from birth and by the distribution of the paralysis.

3. — Rickets.— Parents frequently bring their children to the hospitals thinking that they are paralysed in the legs. It is at once seen that the child can move the limbs, and that the inability to stand is due to the muscular weakness depending upon the ricketty condition.

Subacute Inflammation of the Anterior Cornua.

General Spinal Paralysis.—This disease was first described by Duchenne ; it is characterised by weakness, first affecting the lower limbs, the feet before the legs, the legs before the thighs ; then affecting the upper limbs, the extensor muscles of the fore-arms suffering first, then the arms.

The muscles of the trunk may or may not be affected. The lesion being one of the anterior cornua, the symptoms can be readily understood, *e.g.*

- 1.—“The reaction of degeneration” is met with.
- 2.—Rapid wasting of the muscles takes place, being preceded by paralysis.
- 3.—There are no sensory disturbances.
- 4.—There is no paralysis of the rectum or bladder.

In rare cases the upper limbs are affected before the lower. This disease can be distinguished from—

- 1.—Polio-myelitis anterior acuta, by its gradual onset and by the progressive character of the paralysis, which never reaches its maximum at once.

2.—Progressive muscular atrophy, by its more rapid progress, by the qualitative electrical changes, and especially by the fact that paralysis precedes the atrophy, not following and being proportional to it.

3.—Acute multiple neuritis, which greatly resembles it by the absence of sensory disturbances, and of tenderness along the course of the nerves, and also by the anatomical distribution of the paralysis in multiple neuritis.

Progressive Muscular Atrophy.

This is a chronic disease of the anterior cornua of the cord, characterised by progressive atrophy of the muscles, first usually of those of the thenar eminences.

The following are its characteristic features :—

1.—There is no paralysis until all the muscular fibres are destroyed.

2.—The weakness depends upon and is proportional to the atrophy.

3.—The reflexes and the Faradic response are not abolished until all the muscular fibres are destroyed.

4.—The bladder and rectum are unaffected.

5.—Sensation is also unaffected.

This disease leads to a peculiar distortion of the hand called the “clawed hand” or “main en griffe,” which is due to paralysis of the interossei muscles. The “clawed hand” may be caused by lesions of the

ulnar nerve, which is the motor nerve to the interossei muscles, but in this case there would be sensory and trophic disturbances also.

In addition to the common and classical form of progressive muscular atrophy, to which in fact the name was first applied, there are several types of the disease marked by sufficiently constant and peculiar features to warrant their separate consideration. The *infantile type* was described by Duchenne in 1855. It occurs in childhood and affects first the muscles of expression of the upper part of the face, which are rendered immobile, thus imparting a fixed expression to the face. The forehead is smooth and free from wrinkles, and in the act of laughing the mouth is moved laterally only, the rest of the face being immobile ("*rire en travers*"). To this condition of the face the term "*facies myopathica*" has been applied.

The muscles of mastication and deglutition are unaffected ; the atrophy, however, affects the shoulder and arm muscles, and late in the course of the disease attacks the hand muscles. There is no reaction of degeneration, and no fibrillary tremor, the disease being probably myopathic and not neural. Heredity is a marked feature.

The *juvenile type* was described by Erb in 1884. This form of progressive muscular atrophy is characterised by its occurrence about the period of puberty, first appearing in the shoulder muscles, the hands being rarely affected except in an advanced stage.

The legs frequently suffer, fibrillary tremors do not occur, nor is the reaction of degeneration present.

The leg type of progressive muscular atrophy has recently attracted considerable attention.

Charcot and Marie, in the "Revue de Médecine," for February, 1886, contribute an article upon this form of progressive muscular atrophy, and fully describe five cases which they have met with.

Dr. H. A. Tooth, in a thesis, has recently fully described and collected all the previously recorded cases of this form of atrophy. Dr. Tooth names this variety the peroneal type, because the atrophy most frequently commences in the peronei muscles, which being weakened, cause the foot to be turned inwards (*talipes varus*). But the atrophy may begin in the calf muscles or in those on the front of the leg, and, moreover, the foot is not always turned inwards, so that perhaps it is well to use the more general term and call it the leg type.

The following instances of this form of progressive muscular atrophy have recently come under my notice :—

CASE I.—E. B., a woman, aged 40, was admitted into the Queen's Hospital on November 15th, complaining of weakness of her hands and legs.

The patient remembers that her father was unable to straighten his fingers, and that his hands were wasted like her own. She has heard her father talk of an uncle of his who was affected in the legs and hands, and she says that it was looked upon as a

family complaint. She has two sisters who are affected in the same way as herself.

About three years ago she noticed that she could not easily lift her feet. They dragged and often caused her to trip, until to avoid this constant falling, she got into the habit of raising her feet well off the ground when walking. Soon she noticed weakness in the calf muscles, with wasting, the weakness and wasting gradually increasing up to the present time. About twelve months ago she observed that her hands were wasting and becoming weak.

There is effacement of the thenar and hypothenar eminences; the "clawed hand" is present on both sides, and the forearms are slightly wasted. The legs are much wasted, the calf muscles being almost destroyed, and there is a depression along the outer side of the tibia, marking the atrophy of the extensor muscles.

The feet are dropped, flaccid, and turned inwards, there being no power of dorsal flexion, but she can flex the toes and move the feet from side to side. The soles of the feet are deeply hollowed. There is marked wasting of the vastus internus on each side, causing the inner condyle of the femur to appear abnormally prominent.

The shoulder, trunk, buttock, and facial muscles are unaffected.

There are no fibrillar contractions, nor can any be evoked by filliping the muscles. Sensation is

unaffected but she has suffered from cramps in the calf muscles.

The plantar reflex is lost on both sides, the knee jerk is present on the right side, absent on the left.

The feet and hands are always bluish and cold.

With the *Faradic* current, in the right leg the peronei contract very feebly. The tibialis anticus and extensor proprius pollicis also respond very slightly. The extensor longus digitorum does not respond.

The calf muscles, interossei and peroneus tertius all respond, but the response is decidedly below normal.

In the left leg the tibialis anticus and peronei respond as do the calf muscles, but the response of the extensor communis digitorum is much diminished.

With *Galvanism*, the reaction of degeneration is present on both sides, the contractions being quantitatively diminished and sluggish, A C C predominating over C C C.

There was in this case no enlargement of any muscles, and no history of lead or alcoholic poisoning.

The patient's two sisters came to see me at the hospital, and I found that they were both suffering from the same type of progressive muscular atrophy.

CASE 2.—C. M., aged 41, states that her illness commenced four years ago, and that she noticed it first after a confinement, observing that she could not pick her toes up; and she often used to pitch forwards. She has had nine children and no miscarriages.

The muscles of the feet and legs are extremely atrophied, and she has no power over the feet, which are perfectly flaccid, dropped, and turned inwards.

The circum. of R. leg 6 in. below patella = 9 in.; L. $8\frac{1}{2}$ in.

„ „ „ above „ = 13 in.; L. 13 in.

With Faradism the extensor communis digitorum and the peronei on both sides give no response, the gastrocnemii respond freely. With Galvanism A C C predominates over C C C in the leg muscles.

The knee jerk is absent on the left side, feebly present on the right side.

The gait is very characteristic, the patient having to bend the knees to a considerable extent to enable her feet to clear the ground, "equine gait," there being no waddling.

Sensation is unaffected but she has suffered from pains in the legs and from girdle pain.

The thenar and hypothenar muscles are wasted, and the grasp is very feeble. She is unable to extend the fingers, which are flexed at the phalangeal, extended at the metacarpo-phalangeal joints (*main en griffe*).

There is no fibrillar contraction, no muscular hypertrophy, no pupil alterations, and no wasting of the face, tongue, shoulder, hip, or trunk muscles. The thighs are wasted in the lower third only. The disease commenced insidiously, and the weakness and atrophy are slowly progressing *pari passu*.

CASE 3.—The other sister, A. B., 49, the eldest of the three sisters, is affected in the same way as her

sisters, but the disease in her case is more advanced. She states that it has been coming on twenty-nine years. The knee-jerk is lost on both sides. The legs and lower third of each thigh are much wasted, the feet being in the position of talipes varus. There is no power of dorsal flexion of the foot. The hands are clawed and wasted, and there is marked "high action" gait. There are no fibrillar contractions, no sensory changes, no wasting of the face, shoulder, hip or trunk muscles. The muscles on the front of the legs gave no response to the Faradic current; some pain having been given her she refused to let us try the Galvanic current.

CASE 4.—J. B., a man, aged 50, at the present time an inmate of the Birmingham Workhouse Infirmary, is in an advanced stage of progressive muscular atrophy, which has completely destroyed the muscles of the legs. The disease commenced nine years ago, when he noticed that he was clumsy in picking his feet up, and often used to trip. He had syphilis twenty-five years ago, and has been an ironworker, having had to pull up heavy melting pots out of a furnace, there being thus a great strain on the legs. The legs and lower third of the thighs are extremely atrophied, the feet are dropped, and he has completely lost all power over them, being unable to produce the slightest movement even of the toes. He can bend the knee, but has difficulty in extending it again. The circumference of the right leg three inches below the patella

is 11in., of the left at the same level $11\frac{1}{2}$ in. The circumference of the thigh five inches above the patella on the right side measures 13in., on the left $13\frac{1}{4}$ in. The gait is very characteristic, and similar

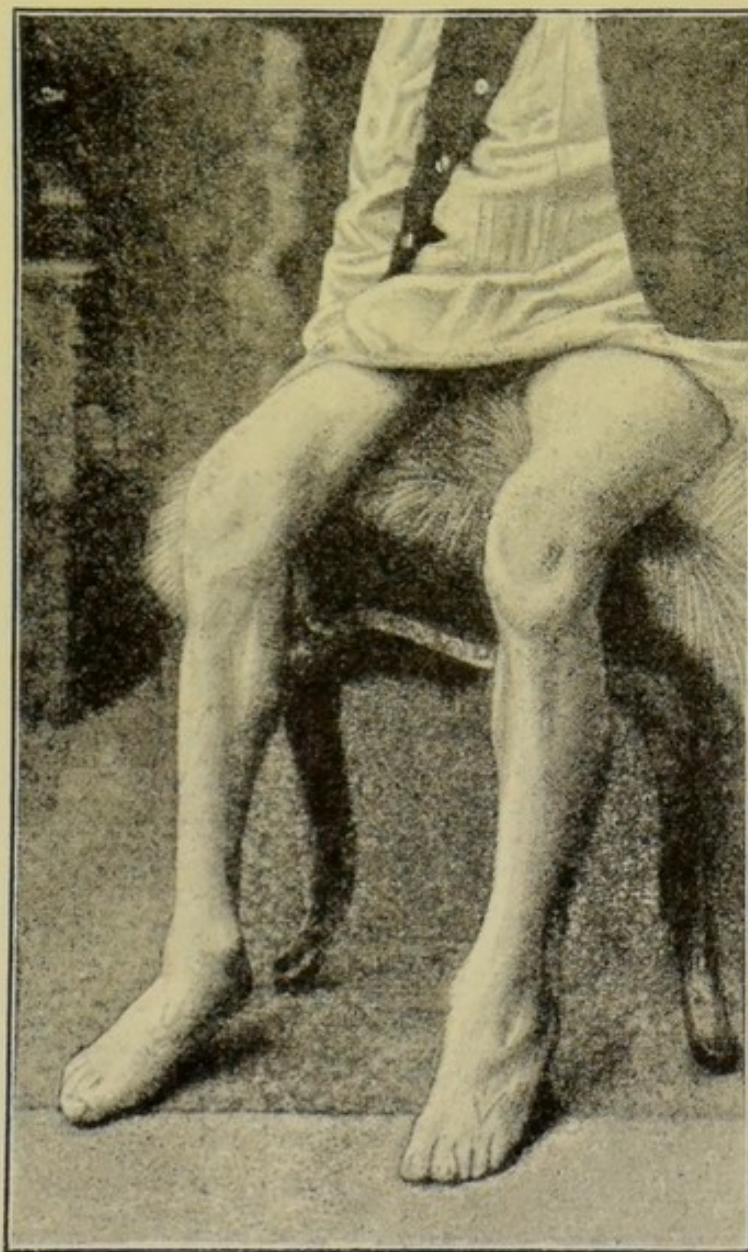


FIG. 20.

THE LEG TYPE OF PROGRESSIVE MUSCULAR ATROPHY.

to that in the above cases, not at all waddling, but of the high action character. The thigh muscles are much atrophied in the lower third, especially in the

situation of the vastus internus, the internal condyles projecting to a marked extent. The muscles of the sole of the foot are much wasted, and in standing there is complete talipes valgus. With the Faradic current there is no response in any of the muscles on the front of either leg, and a feeble response only of the peronei and calf muscles. With Galvanism there is no response of the muscles on the front of the legs. In the peronei and calf muscles A C C predominates over C C C. There is a little flattening of the thenar and hypothenar eminences in each hand, but no clawed condition. Sensation is normal, but he complains of coldness of the hands and feet, which in fact are cold and bluish. There is no wasting of the facial, shoulder, or trunk muscles, and no fibrillar contractions. He can give me no family history of importance.

Progressive muscular atrophy must be distinguished from

- 1.—Lesions of the peripheral nerves.
- 2.—Lead paralysis.
- 3.—The atrophy that is frequently present in cases of writer's cramp and other professional hyperkineses.

Paralysis and muscular atrophy due to lead poisoning, in the absence of the blue line on the gums, may be distinguished by the following facts:—

- 1.—The onset of the paralysis is usually rapid when due to lead.
- 2.—The paresis is in excess of the atrophy.

3.—Qualitative electrical changes are usually present.

4.—There is improvement on administering iodide of potassium internally.

In the muscular atrophy which sometimes accompanies writer's cramp and other professional hyperkineses the atrophy is preceded by paresis or cramp, and partial or complete recovery ensues when absolute cessation of the act of writing is insisted upon and galvanism is employed. The atrophy does not become general and endanger life.

Pseudo-hypertrophic Paralysis.

The pathology of this disease is by no means definitely known, some authorities believing it to be a disease of the motor cells in the anterior cornua, others a disease of the muscles.

This disease may be confounded with progressive muscular atrophy, for there is always atrophy of some muscles and towards the end a condition of general paralysis with great emaciation. But even then the calf-muscles and the infra-spinati will be found to be enlarged. The knee-jerk will be lost and talipes equinus with inability to perform dorsal flexion of the foot present.

Of course the presence of the waddling gait, the characteristic mode of rising from the recumbent and sitting posture, and lordosis at once distinguish the disease.

It is important to remember that this disease occurs in adults. I have met with three such cases, two of which I have recorded. All three patients had been informed that they were suffering from "general debility," and that they would get well.

The characteristic symptoms of the disease are :—

1.—Enlargement of the muscles of the calves, buttocks, and forearms, with marked weakness.

2.—Atrophy of certain muscles, especially of the biceps and costo-sternal portions of the pectoralis major.

3.—Marked lordosis in the erect position, a plumb-line let fall from the most prominent part of the spine falling three or four inches behind the sacrum.

4.—A waddling gait and the characteristic mode of rising from the sitting and recumbent position.

5.—In a late stage loss of the knee-jerk.

When progressive muscular atrophy commences in the lower extremities and back it is difficult to distinguish it from pseudo-hypertrophic paralysis, but a waddling gait is peculiar to the latter disease, while in the former the gait is of the high action or "equine" type.

Pseudo-Hypertrophic Paralysis in Adults.

CASE 1.—J. McD., aged 33, a commercial traveller, applied on account of weakness in the back and legs, by which he had been affected for

nearly three years. There was no history of any similar disease, to the patient's knowledge, in his family. He was perfectly strong and well up to within three years ago, and had never noticed any enlargement of his calves or weakness in the legs when a boy. He could not assign any cause for his illness; he had never done any laborious work, and had never worked in lead, nor been exposed to wet or cold. He had been rather a free drinker, but there was no history of syphilis or of rheumatism.

Three years ago, in the winter, when in South America, he noticed himself becoming weak in the back, and found that he could not run; and if at any time his foot slipped, he had difficulty in recovering himself. At that time, his arms and legs were unaffected, but his back kept getting worse, and in about six or seven months he noticed his thighs to be getting smaller and weaker, and, at the same time, his arms wasted and became weaker. When first seen, his height was 5ft. $6\frac{1}{4}$ in., formerly it had been 5ft. $7\frac{1}{2}$ in.

The patient, apart from the atrophied parts, looked a strong, healthy man. His gait was markedly waddling; he walked on his toes, the heels not touching the ground; if he separated his legs, he could make his heels touch the ground, but not otherwise.

He walked with his shoulders thrown back, and there was marked lordosis and considerable projection of the belly. The lordosis disappeared when

he sat down, being replaced by a convexity backwards. A plumb-line, dropped from the shoulders, was about $3\frac{1}{2}$ in. from the sacrum. When asked to sit down, he first jerked his body forwards, and then went down suddenly. When going upstairs, he took hold of the balusters, and having put one foot on the step above, at a considerable distance from the balusters, so as to avoid the necessity of bending his knee, he dragged himself up by the balusters. When lying on his back, he could not sit up unaided, except by turning over on to his side, and pushing himself up. If, however, his feet were fixed, and thus his legs prevented from moving, he could bring himself straight upwards and forwards into the sitting posture. If he wished to stand up, he had to turn over on his side, and then push himself upwards to the length of the arms, and place his hands on his knees, thus climbing up his own body.

There was marked atrophy of the muscles of the arm, buttock, and thigh on each side. The biceps had almost disappeared on both sides. The scapular portion of the deltoid was much atrophied, the clavicular portion little altered. The inner head of the triceps on the right side was unduly large. The forearm and hand on each side seemed to be splendidly developed, the thenar and hypothenar eminences being very large; yet, when tested with the dynamometer, the grasp was only forty pounds. The scapular muscles were much atrophied. The scapular portion of the trapezius was atrophied,

while the clavicular portion was unaffected. The dorsal muscles did not seem to be reduced in volume. The pectoral muscles on both sides were markedly atrophied, and the patient could not bring his arms to the middle line in front—that is, he could not clap his hands unless he flexed his elbows. There was a marked hollow between the scapulæ, the posterior borders of these bones projecting a great deal (winged scapulæ). The serrati muscles were evidently paralysed, for the patient could not raise his arm above the horizontal line on the right side, and only a little above on the left side. The buttock and thigh muscles were much wasted, but the gluteus medius could be felt to contract when he walked. The calves were firm and large, and the swelling was high up. Dorsal extension of the foot could not be produced to the normal extent. Sensation was perfectly normal, and the bladder and rectum unaffected. The temperature of the legs was 95° F. There was no mottling of the skin, nor could any fibrillary contractions be evoked.

The patient was very sensitive to cold, and was always better in warm weather. His intellect was unaffected, being much above the average. The patellar tendon reflex was abolished on both sides, the other reflexes were normal. The muscles responded to Faradism, except the latissimus dorsi, the serrati, the pectoral muscles, and the erector spinæ, which did not respond at all. The response of the biceps muscle on each side was much

diminished; Galvanism of the nerve trunks caused contraction of the forearm and hand muscles; but the contraction was below the normal, while Galvanism applied directly to the muscles showed a much greater diminution of response. No response could be obtained in the case of the pectoral muscles, the serrati, and rhomboidei, showing that they were totally destroyed.

CASE 2.—F. B., aged 35, a married woman, attended the Queen's Hospital on April 21st, 1885. She complained of weakness in the back and legs, and instability when standing, saying that she easily fell or was pushed over, and of occasional pains in the joints. Her father died from consumption at the age of 35; one of her brothers had a "stroke." Besides this brother no one else in the family ever suffered from any weakness or paralysis. She has had two miscarriages and six children born alive, one of whom died from whooping-cough; of the other five children, one little boy is weakly, all the others perfectly well and strong. The patient has occasionally had pains in her joints, but otherwise her health has been good; there is no history of syphilis; she has not worked in lead or been much exposed to cold or wet. Eleven years ago she noticed weakness and pain in the bottom of her back, the weakness being the chief trouble. This got gradually worse, and has extended during the last four or five years to the arms and legs, and she has noticed that she has been getting stouter in her limbs and body. The

patient looks a strong, healthy woman, is well developed, and rather stout. She stands with her feet apart and her back much arched, there being very considerable lordosis. She can, however, stand with her feet close together, and the heels touch the ground. When standing, a plumb-line dropped from the most prominent part of the spine falls three inches behind the sacrum; when in the sitting posture the lordosis disappears. Her gait is waddling, the body swaying from side to side as she walks. She says that she falls even if a child runs against her. In raising herself from the ground when lying on her back, she first turns over to the left, puts both hands on the ground, gets upon her feet, then places the hands upon the knees, and climbs up her thighs, and finally jerks herself into the upright position. When rising from the sitting posture she puts her hands upon the knees and raises herself by jerking movements. The prominence of the calf muscles is very large on both sides and firm. On the right side there is a well-marked abrupt ridge formed by the calf muscles; the circumference of the largest part of the right calf = $15\frac{1}{4}$ in.; of the left, $14\frac{3}{4}$ in. The patellar reflex is present on both sides, but is much diminished on the right, and the right leg is weaker than the left. The buttocks are also large, the glutei muscles being felt to contract on walking. The sterno-costal portion of the pectoralis major of the right side is much wasted. She has great difficulty in raising her arms vertically upwards, and she cannot hold her

right arm horizontally forwards for more than a few seconds, and when she does so the posterior border of the scapula projects markedly backwards, showing paresis of the serratus magnus. She can place the right hand upon the head, but with difficulty. The right infra-spinatus muscle is very large, the biceps on the right side is very feeble and atrophied, and she cannot flex the elbow when the forearm is supinated. The forearm is apparently finely developed on both sides, and the prominence of the extensors of the wrist is excessive, while the power of extension is very feeble. The thenar eminence is very well marked on both sides, and there is no evidence of atrophy in the small muscles of either hand. Although the upper limbs look splendidly developed, yet she complains of great weakness in them, and speedily wearies even by holding them out for a few seconds. The grasp of the right hand by the dynamometer registers 70lbs.; of the left, 75lbs. The erector spinæ muscles are atrophied and weak. Sensation is everywhere normal, and there are no vasomotor disturbances, nor any affection of the bladder or rectum, and no fibrillary contractions can be obtained by flipping the muscles. The intellect is also normal. Faradism to the nerves, motor points, and muscles brings about feeble contractions; Galvanism to the nerve trunks causes a stronger contraction than when the current is applied directly to the muscles. The patient brought her little boy, whom she said was weak in the legs, to see me. The calves in his case

seemed large, but not markedly so. The gait was waddling, and he complained that he was easily upset, but there were no other symptoms of the disease.

Pseudo-hypertrophic paralysis is very rare in adults, except in those cases where the disease begins in infancy, and the patient lives to adult age. Two cases, similar to those reported above, are recorded in the Clinical Society's "Transactions," Vol. XVI. The waddling gait, the absence of patellar reflexes, the talipes equinus, the hardness and increased size of the forearms and legs, distinguish the disease from progressive muscular atrophy. Talipes equinus is only met with late in the disease, and the patellar reflexes are not abolished as long as the quadriceps extensor is not completely atrophied.

Primary Lateral Sclerosis.

This disease is a primary system disease affecting the crossed pyramidal tracts of the lateral columns, with the following symptoms:—

1.—It is a disease of adult life very rarely affecting women.

2.—Weakness and rigidity, with spasmodic twitchings of the legs, are the characteristic symptoms. The onset is very insidious, the rigidity being present from the first; the paresis and rigidity commencing simultaneously and advancing *pari passu*.

3.—There are no sensory disturbances.

4.—The deep and superficial reflexes are exaggerated, ankle clonus being present.

5.—The gait is very characteristic, the patient having great difficulty in elevating the toes, which scrape the ground.

We must distinguish between this disease and secondary degeneration of the pyramidal tracts following a chronic myelitis or following compression of the cord, as in the paraplegia which often follows spinal caries, or in that produced by an extra medullary tumour; because in chronic myelitis all sensory disturbances may disappear, leaving a condition exactly similar to that observed in primary lateral sclerosis. We must then depend upon the history of the onset of the illness.

In chronic myelitis:—

1.—The weakness precedes the rigidity.

2.—Sensory disturbances are usually present.

3.—The bladder and rectum may be paralysed, and these organs are always more affected than in primary lateral sclerosis.

In compression of the cord the onset and progress are more rapid than in primary lateral sclerosis; the paresis also precedes and preponderates over the rigidity.

In disseminated sclerosis the sclerosis might affect only the pyramidal tracts and cause symptoms exactly similar to those observed in primary lateral

sclerosis, but sooner or later characteristic symptoms of the former disease would appear, such as nystagmus, vertigo, syllabic utterance, &c.

Amyotrophic Lateral Sclerosis.

This is a combined system disease of the crossed pyramidal tracts, and of the anterior cornua, and was first described by Charcot.

1.—The upper extremities are usually first affected, the symptoms consisting in weakness, with rigidity and contracture, followed by

2.—General atrophy of the muscles of the upper extremity, followed by

3.—Weakness and rigidity of the lower extremities, and after a time diffuse atrophy of the muscles of the lower extremities.

The reflexes are exaggerated, there is no disturbance of sensation and no affection of the bladder or rectum. The disease is a very fatal one, lasting two or three years, death being produced by extension of the disease to the medulla and the production of bulbar symptoms.

I published in the "Birmingham Medical Review," March, 1884, an account of a man still in the Workhouse Infirmary, who is suffering from this disease. The disease has not yet extended to the medulla, but the patient is quite bedridden.

The diagnosis of this disease is very easy. From progressive muscular atrophy we can distinguish it:—

1.—By the atrophy being general and following the paresis, for this reason Charcot called this disease “*atrophie en masse*.” In progressive muscular atrophy it is local, a group of muscles, or even a single muscle, being affected

2.—By the presence of rigidity and the increase of reflexes.

3.—By the rapidity of its progress.

4.—By the signs of spastic paraplegia, which supervene in the lower extremities.

Pachymeningitis cervicalis hypertrophica somewhat resembles amyotrophic lateral sclerosis; but the former disease is attended—

1.—With marked sensory disturbances, severe pains, and hyperæsthesia in the upper extremities.

2.—There is no atrophy of the muscles of the lower extremities.

3.—The disease frequently ends in complete recovery, and does not implicate the medulla.

Primary lateral sclerosis is easily distinguished from amyotrophic lateral sclerosis—

1.—By the absence of general atrophy of the muscles.

2.—By its far more chronic nature.

3.—By there being no tendency to bulbar paralysis.

Locomotor Ataxia, or Tabes Dorsalis.

This disease is so well known to you that it is needless for me to enumerate its symptoms. It is a primary system disease of the postero-external columns of the cord or root zones. Loss of the knee-jerk is an early symptom of this disease ; it may, in fact, precede all other symptoms for years, although usually it is associated with the Argyll-Robertson pupil (loss of the pupil reflex to light only).

The various diseases in which the knee-jerk is lost must be carefully kept in mind before the diagnosis of tabes is made. There is little doubt that many supposed cases of this disease which have been cured were cases of peripheral neuritis from alcohol or other cause.

Primary optic atrophy may precede all other symptoms, even loss of the knee-jerk, for years. In one of my cases perforating ulcer of the foot was the first symptom observed.

The combination of anæsthesia with shooting pains and cramps, termed "anæsthesia dolorosa," is often observed in this disease. This condition is of considerable value as a diagnostic sign, for it shows that the lesion is situated outside the grey matter of the cord. It is produced by pressure on the posterior nerve roots, or on the sensory fibres as they pass through the postero-external columns of the cord ; therefore it is often present in locomotor ataxia, and is a marked symptom also in cases of extra-medullary growths which compress the posterior nerve roots.

The diagnosis of *tabes dorsalis* can be made long before the gait becomes ataxic:—

Thus (1) loss of the knee-jerk, (2) with the characteristic pains, and (3) the pupil phenomena are in themselves sufficient; or grey atrophy of the discs, with loss of the knee-jerk, would suffice for the diagnosis of the disease, even in the absence of ataxy. The disease may commence in the upper extremities. Mr. Chavasse recently sent me a man, who complained of numbness in the fingers and inability to use them in picking up things. He was a hatter by trade, and he could not hold hats in his left hand as well as usual. There had been no pains, and there was no paralysis or anæsthesia, but there was marked ataxy and loss of muscular sense in the upper extremities. He could not distinguish a half-pound from an ounce weight suspended in bags from his fingers. He said that he could only just perceive a pound weight similarly suspended. There were no electrical changes or other indications of a peripheral neuritis. The knee-jerk was normal, and the legs were not ataxic, nor were there any ocular symptoms. I think there can be no doubt that this was a case of *tabes dorsalis* beginning in the lower cervical region of the cord.

Loss of muscular sense may occur—

- 1.—In lesions of the peripheral nerves.
- 2.—In lesions of the cord, especially in locomotor ataxia.

3.—In lesions of the internal capsule, as previously mentioned.

I have several times seen maniacal symptoms supervene in cases of locomotor ataxia and symptoms of general paralytic dementia.

The joint disease occurring in locomotor ataxia, and described by Charcot as a trophic lesion, is said to be distinguished from chronic rheumatic arthritis by the following features:—

1.—Pain is absent in this disease, present in rheumatic arthritis.

2.—Effusion is generally present here and absent in rheumatic arthritis.

3.—Dislocations are common in tabes, but rare in rheumatic arthritis, moreover the bones are apt to fracture very readily, and without pain in the former.

4.—In the joint lesion of tabes there is preternatural mobility.

5.—In tabes the knee and shoulder are most often affected, in rheumatic arthritis the hip and smaller joints.

6.—There is more atrophy of bone in tabes, more out-growth or hypertrophy in rheumatic arthritis.

Locomotor ataxia must be distinguished from

1.—The ataxy of cerebellar disease.

2.—Hysterical ataxy.

3.—Multiple sclerosis, with implication of the postero-external columns.

4.—Syphilis, which may cause symptoms very like those of true tabes, with paralysis of the cranial nerves and pains in the legs.

5.—Ataxia due to peripheral neuritis, from alcoholism, diphtheria, diabetes, &c.

In cerebellar disease there is a staggering gait, not the peculiar jerking gait of the ataxic.

The knee jerk is sometimes lost in this disease, so that a mistake may easily occur.

But the presence of vomiting, headache, double optic neuritis, or convulsive seizures would clear up the case.

In hysterical ataxy the patient is able to use the limbs with perfect order while lying down, ataxy being marked when she sits up or stands. Pains are usually absent and the knee jerk is not lost.

In locomotor ataxia temporary paralysis of the ocular muscles is common, and it is characteristic in that the paralysis gets well of itself, contrasting with the optic nerve atrophy, which never recovers.

Mercury and iodide of potassium cure syphilitic affections, but I have never observed any appreciable improvement from the administration of iodide of potassium, even in large doses, in locomotor ataxy.

Combined disease of the postero-external and lateral spinal tracts.

Sclerosis of the postero-external columns of the cord may be combined with sclerosis of the lateral columns.

In these cases there is weakness of the lower extremities combined with ataxy.

The reflexes are exaggerated, the knee jerk being excessive, and ankle clonus is present.

Pains are usually absent or slightly marked.

The pupil symptom and optic atrophy are rarely present.

Visceral crises are also very rare.

In this disease the lesion in the lateral columns occurs simultaneously with that in the postero-external tracts.

In ordinary tabes weakness may supervene from extension of the disease to the lateral columns, and wasting from extension to the anterior cornua, but in these cases the knee jerk, which is lost early, does not reappear.

Dr. Gowers has recently fully described this condition, which he calls "ataxic paraplegia" in a clinical lecture published in the "Lancet," July, 1886, and in "Brain," Vol. VIII., is contained a critical digest of the subject by Dr. Ormerod.

Friedreich's Disease.

An hereditary ataxia, first described by Friedreich, and hence named after him, must be distinguished from ordinary tabes.

Hereditary ataxia attacks many members of the same generation, and runs in families.

It occurs in early life (4-18), and in both sexes, while ordinary tabes is a disease of adult life.

Ataxy of the lower extremities, with loss of the knee jerk, is always present, the ataxy soon affecting the muscles of the trunk and upper extremities.

There are no pains, no loss of pupil reflex to light, no transient paralysis of the ocular muscles, no optic atrophy, no visceral crises, and no joint or bone lesions.

But speech is usually affected, being slurred, or there may be a mere drawl or stammering.

In some cases speech becomes unintelligible.

Nystagmus occurs late in the disease, and curvature of the spine may occur early or late from defective muscular support.

The rapid spreading of the ataxy upwards is characteristic.

The indiscriminate lesions of the spinal cord are

- 1.—Myelitis.
- 2.—Disseminated sclerosis.
- 3.—Hæmorrhage.
- 4.—Tumours.

Myelitis may be acute, subacute, or chronic, and may attack the whole or any part of the transverse section of the cord, and a variable length of the cord.

Acute transverse myelitis or other lesion affecting the whole transverse section of the cord will cause the following symptoms :—

- 1.—Paralysis of the muscles supplied by the nerve cells of the anterior cornua in the inflamed

segment, and of all the muscles supplied by nerves coming off from the cord below the lesion.

2.—Anæsthesia below the lesion and in the area corresponding to the inflamed segment.

3.—A narrow band of hyperæsthesia is usually present at the upper level of the lesion, due to irritation of the sensory nerve fibres at this level.

If the myelitis be in the dorsal region this hyperæsthesia is in the form of a band round the body and occasions the girdle sensation.

If the myelitis be in the lumbar or cervical region then the hyperæsthesia is distributed over the limbs according to the distribution of the sensory nerves at the upper level of the lesion.

4.—The reflexes passing through the cord above the lesion are unaffected, those passing through the seat of the lesion are abolished, and those passing below the lesion are exaggerated, cerebral control being shut off.

5.—In the muscles supplied from the inflamed area of the cord rapid wasting and degeneration both of motor nerves and muscles takes place, but there are no qualitative electrical changes or rapid wasting in the paralysed parts supplied from the cord below the lesion.

Trophic changes in the skin in the form of acute bed sores also occur in the sensory area corresponding to the inflamed portion of the cord.

6.—The bladder and rectum are always affected but in a manner depending upon the situation of the lesion.

If the lesion is in the lumbar region then the sphincters are paralysed and cystitis with ammoniacal urine rapidly supervenes.

If the lesion is above the lumbar region then the reflex acts of defæcation take place without the knowledge of the patient, and cannot be controlled by him.

7.—Elevation of temperature in the paralysed limbs is common, and œdema is also often present, being due to vaso-motor paralysis.

8.—Later on, descending degeneration of the pyramidal tracts takes place, causing rigidity and contracture.

In the other varieties of myelitis the symptoms will of course vary with the seat of the lesion in the transverse section.

Case of Acute Myelitis.

G. C., a man aged 20, a shoemaker, admitted into the Queen's Hospital, March 19th, died May 5th, 1884.

The patient never had any previous illness to his knowledge, but there were scars of strumous abscesses in his neck; there was no distinct history of syphilis, and anti-syphilitic treatment when tried had no good effect, but was injurious. There was no history of alcoholism or exposure, but his occupation compelled him to lead a sedentary life. The family history as far as could be ascertained was good. The patient had been ill for eight weeks previous to admission, the illness commencing as pain passing

round the body about midway between the umbilicus and ensiform cartilage (girdle pain). He had also numbness in the feet spreading quickly upwards, with sensation "of pins and needles." He had twitchings of the legs. He took to his bed and rapidly lost all power over the lower part of the body, and all sensation. There was also some difficulty at this time in micturition. On admission the patient was found to be completely paralysed in the lower extremities.

Sensation (as to touch, pain, heat, and cold) was completely abolished up to the level of the umbilicus, where there was a well-marked hyperæsthetic zone. A pin drawn over this area occasioned a cutting sensation. In this region also there is well-marked *tache cérébrale*. The special senses were perfectly normal.

Reflexes. Superficial.—The plantar reflexes were exaggerated; the cremasteric were also well marked; the abdominal and the epigastric reflexes were absent. The scapular reflex was well marked on both sides.

Deep. No ankle clonus or front tap contraction could be obtained. The patellar-tendon reflexes were exaggerated. The lower limbs were wasted, but no local atrophy could be made out.

Bedsore were found over the right trochanter and anterior part of the iliac crest. [The patient had been lying chiefly on this side.] The skin over the trochanter on the left side was discoloured and evidently about to break down. There was no bed-

sore over the sacrum. The patient complained of pain in the back, and pain was elicited when percussion was employed over the 6th dorsal spine. A hot sponge passed down the spine did not discover the hyperæsthetic zone. If one foot was pricked with a pin, or if the sole was tickled, a reflex action was brought about in the opposite foot. There was œdema of both feet. The ribs were observed to move very slightly during inspiration, and the lower intercostal muscles (below the 6th space) were paralysed. The temperature was 103° F. The urine slightly alkaline with phosphates; there was no cystitis and no paralysis of the bladder. The patient's intellect was unaffected. In a few days after admission bedsores formed on the left side where the skin had been red; but they were much worse on the right side and discharged pus freely. The superficial reflexes were much more increased on the right side than on the left. The urine soon became markedly ammoniacal, and a small amount of pus was present with triple phosphate, cystitis being set up. The urinary disturbance in the first place was merely due to loss of cerebral control, the reflex taking place unconsciously; there was never any complete paralysis of the bladder, although the catheter had to be passed occasionally.

On March 25th the patient had a rigor and was much troubled by involuntary twitchings of the legs. The pulse was quick (132) and dicrotous; diarrhœa came on but was easily checked.

April 17th.—The bedsores have spread considerably, the bone is exposed and carious.

April 30th.—Persistent vomiting set in, which still further distressed the patient, who died from exhaustion on the 5th of May.

The temperature throughout was of a remittent type, averaging 101° F. in the evening, and coming down to normal in the morning.

Autopsy.—The body was much emaciated, rigor mortis was very slight. There were old cicatrices of strumous glands in the neck. Extensive sores over both trochanters (major) and over anterior superior iliac spine. There was no sore over the sacrum. The brain was normal.

The cord, after the removal of the spines and laminae, appeared normal, excepting the portion between the 5th and 9th dorsal vertebrae. Here the dura appeared coated with yellowish caseous material; this substance extended also through the intervertebral foramina, and for some distance along the intercostal nerves. The cord having been removed and the membranes longitudinally incised, the cord itself was found to be normal in appearance, except in the length corresponding to the length of dura coated with caseous material. In this situation the cord was acutely softened, white and creamy, the distinction between the grey and white matter being lost; both sides of the cord were equally affected. The heart and lungs were normal; there was no lardaceous

change. A tubercular ulcer was found near the ileo-cæcal valve.

Kidneys.—The left weighed $9\frac{1}{2}$ ounces, and was fatty. The right 12 ounces, with yellow cheesy deposits beneath the capsule and in the cortex. In one place was an abscess from softening of a caseous mass in communication with the pelvis.

Remarks.—The case was a typical one of acute diffuse myelitis; the characteristic features of which are the rapid loss of motion and sensation in the parts supplied by nerves given off from the cord below the seat of lesion, attended with fever. The stage of irritation was first present, there being pain in the back, and twitchings, and perverted sensations, but this was quickly followed by complete loss of motion and sensation. The lesion was easily localised by means of the reflexes. 1. The cremasteric reflex passes through the cord at the level of the 1st and 2nd lumbar nerves. 2. The abdominal through the cord between the 8th and 12th dorsal nerves. 3. The epigastric through the cord about the 5th, 6th, and 7th dorsal nerves. 4. The interscapular through the cord about the two lower cervical and three upper dorsal nerves. Now the cremasteric and interscapular were present; the abdominal and epigastric absent; therefore we supposed the lesion in the cord to be situated between the 4th and 12th dorsal nerves, and it was found between the 5th and 9th. We also judged that the lesion was of considerable vertical extent. The lumbar enlargement was found to be

intact, thus accounting for the absence of paralysis of the bladder and sacral bed sore. The presence of the sores over the front of the crest of the ilium and trochanters is explained by the fact that the skin here is chiefly supplied by the lateral cutaneous branch of the last dorsal nerve.

The prognosis was rendered even worse than it usually is by the evident strumous condition of the patient. The prognosis in acute myelitis depends upon—(1) The situation of the inflammation, *i.e.*, the region of the cord affected; (2) The extent of the transverse section of cord affected; (3) The vertical extent. It is always more fatal in the cervical and lumbar regions than in the dorsal; more serious when the grey matter is affected, because bedsores break out; and more serious the greater the length of cord affected.

In the above case the cord was found after death in a condition of white softening; it was like cream. This was due to the extensive fatty degeneration of the medullary sheaths of the nerves, and also of the nerve cells, and to the formation of masses of fat granules. This stage is preceded by red softening, when the affected part of the cord is swollen, red, and injected, and its consistency diminished. It was, of course, impossible that recovery could take place after such extensive softening, but had the patient not been previously debilitated he would not have died so soon.

As to the cause of the myelitis in this case. It was found that the softening of the cord exactly

corresponded to the situation of caseous material on the outer surface of the theca. Probably this material, by constricting the vessels to this part of the cord (for it extended along the nerves), was the predisposing cause.

Hemilateral myelitis or other lesion affecting one half of the transverse section of the cord will cause the following symptoms :—

1.—Paralysis on the same side of the body, there being paralysis of all muscles supplied by the nerves coming off at the seat of the lesion, and from the same side of the cord below the lesion.

2.—Since the sensory nerves decussate almost immediately on entering the cord, inflammation of one half segment of the cord causes anæsthesia on both sides of the body in the area of the inflamed segment. If the lesion is in the dorsal region, then there is a band of anæsthesia round the body ; above this band of anæsthesia there is generally a band of hyperæsthesia, due to irritation of the sensory fibres, which enter the cord just above the lesion.

3.—Below the lesion there is anæsthesia on the opposite side ; hyperæsthesia on the same side.

The sensory fibres not decussating immediately in the lumbar and lower dorsal regions of the cord, a unilateral lesion in these regions will cause anæsthesia, as well as paralysis, of the leg on the same side.

4.—The reflexes passing through the inflamed segment are lost, those passing through the same side of the cord below the lesion are increased.

5.—There is vaso-motor paralysis with elevation of temperature and loss of muscular sense on the same side as the lesion, that is, in the paralysed extremity.

6.—Those muscles supplied by the inflamed segment of the cord will present qualitative electrical changes, and will undergo rapid atrophy.

A unilateral lesion of the cord then causes motor paralysis, vaso-motor paralysis, and loss of muscular sense, with hyperæsthesia on the side of the lesion, and anæsthesia on the opposite side.

Case of Acute Hemilateral Myelitis due to Syphilis.

P. C., a man aged 61, was treated for primary syphilis in the Birmingham Workhouse Infirmary, in August, 1884. In November of the same year he was under my care for secondary ulceration of the throat and tongue. In December he was again admitted, and treated for syphilitic iritis. The patient as soon as he felt relieved persisting in leaving the infirmary. On the 28th of May, 1885, he was again admitted under my care, complaining of complete paralysis of the left leg. The loss of power, he said, had come on somewhat gradually, and was complete in about a week. He had had no pain in the leg or around the body. There were several pigmented scars on the leg, and the pupils were irregular and refused to respond to light or accommodation.

It was found that the patient was suffering from complete motor paralysis of the left leg, with flaccidity. There was marked hyperæsthesia on the paralysed side, ordinary impressions being painful. Sensations of touch, temperature, and pain were all exaggerated. Contacts made by the ends of a pair of compasses coalesced and were perceived as a single sensation when the distance between the points was less than three inches on the right leg, but on the left the distance between the points had to be diminished to three-quarters of an inch before fusion of the two impressions was produced. The discriminative sensibility of the skin was, in fact, much increased in the left leg, much diminished in the right. There was, moreover, vaso-motor paralysis and impairment of muscular sense in the paralysed limb, with elevation of temperature. The temperature of the right lower extremity was 98.4° F., that of the left 97° F. The patient could appreciate the difference between 1 oz. and 2 oz. weights suspended in bags from the toes on the right side, but not on the left. This experiment was performed after the patient had recovered motor power in the leg and could thus bring into play the muscular sense to aid in the estimation. In the right lower extremity there was complete analgesia and thermal anæsthesia. As to the sense of touch, the power of localisation was perfect, but the discriminative sensibility was diminished.

The analgesia on the right side and the hyperæsthesia on the left extended upwards over the

abdomen to a line drawn round the body two inches above the umbilicus, being sharply defined at the upper limit and ending at exactly corresponding levels, sensation abruptly becoming normal at this line. There were no zones of anæsthesia or hyperæsthesia encircling the body at the upper level of the lesion in the cord as are described in text books to be present in unilateral cord lesions. The plantar reflex on the left side was excessive. There was no ankle clonus on either side. The patellar reflex was diminished on the right side, absent on the left. The cremasteric reflex was well marked on the right side, absent on the left. The abdominal reflex was also present on the right side, but absent on the left. The epigastric reflexes were present on both sides. He suffered from retention of urine for a day or two after admission, and then recovered control, but passed his fæces in bed for some days. The pulse was 130 and irregular, the temperature 104° F. The urine was acid and contained no albumen. He was at this time delirious towards night and very stupid during the day.

The patient was put upon a water bed and given 20-grain doses of iodide of potassium and half-drachm doses of the liq. hydr. perch. twice daily, the urine being drawn off. On June 2nd albumen in small quantity appeared in the urine, which remained acid; no pus was present. On June 3rd the urine was faintly ammoniacal; the iodide was increased to 30 grains and the liq. hydr. perch. to one drachm thrice daily.

June 6th. Rusty coloured sputum was expectorated, and crepitation was heard over the upper lobe of the right lung; the lung mischief, however, quickly subsided, the treatment remaining the same.

June 9th. Benzoate of soda in 20-grain doses was given with each dose of his medicine; this quickly restored the acid reaction of the urine, it having been ammoniacal since the 3rd instant.

June 11th. The patient could move the left leg, bending the knee and drawing up the limb without assistance. The patellar reflex had reappeared on the left side, but the epigastric and abdominal still remained absent. The temperature had now fallen to normal. Constipation was marked, and necessitated the use of enemata.

June 24th. The patient could walk without difficulty. The sensory disturbances in each leg were still present but diminished. No ataxy was observed on the return of motor power. A small bedsore formed over the great trochanter on the left side, but soon healed.

July 2nd. In addition to the administration of iodide of potassium and mercury internally a drachm of mercurial ointment was ordered to be rubbed into the skin of the arm-pits daily.

July 15th. The patient is much improved, in fact, he is well as regards motor power, but the sensory disturbances are still present though to a less degree.

Brown-Séquard first thoroughly investigated the group of symptoms observed in unilateral spinal

lesions. The condition of the reflexes in unilateral lesions has not been fully investigated, but in this case the abolition of the patellar, cremasteric, and abdominal reflexes, and the increase of the plantar on the paralysed side, indicate the seat of the lesion to have extended in the cord on the left side from the 8th dorsal to the 4th lumbar nerves, and negatives the supposition that the centripetal fibres concerned in reflex actions have a crossed course within the cord.

Brown-Séquard observed that there existed in cases of hemi-myelitis a vertical space of about one inch, near the median line, anteriorly and posteriorly, in which hyperæsthesia is absent on the side of the lesion, and a similar space on the front and back in which there is an absence of any great degree of anæsthesia on the side opposite the lesion. This he explains by the fact that the sensory nerves of the two sides overlap for a short distance near the median line; these spaces were not to be found in this case.

Dr. Broadbent has pointed out that it is in those cases of syphilis where the secondary affections are slight that the nervous system is especially liable to suffer, and the tertiary stage arrives early. In this case the secondary phenomena were well marked.

The absence of any phenomena of irritation preceding the paralysis is characteristic of syphilitic myelitis. The absence of the zones of hyperæsthesia and anæsthesia at the upper level of the cord lesion is noteworthy; but in this case localisation of tactile

impressions on the opposite side to the lesion was perfect, and may possibly be explained by supposing that the lesion chiefly affected the lateral columns, grey matter, and anterior cornua, and only slightly the posterior nerve roots. The early occurrence in this case of nervous disease (nine months) after the contraction of syphilis is interesting. In the "British Medical Journal" for June 27th, 1885, a case is reported by Dr. Churton where ataxy supervened within four months of the contraction of the chancre.

In many cases of spinal cord disease I have been able to prevent the supervention of cystitis by giving benzoate of soda, or by washing out the bladder. The first indication of approaching cystitis is an ammoniacal reaction of the urine. I give benzoate of soda in preference to washing out the bladder unless the cystitis be established, for there is considerable danger in the use of catheters.

Early anti-syphilitic treatment is, of course, of the highest importance, and iodide of potassium alone cannot control the disease; mercury must be given either by the mouth or by inunction. In several instances I have found iodide of potassium to fail unless combined with mercury. The prognosis in these cases is favourable but should be guarded. The recovery of motor power, the sensory disturbances remaining, though to a less extent, is contrary to what is usually seen in myelitis. Sensation is, as a rule, first recovered.

Myelitis must be distinguished from

1.—Hæmorrhage outside the cord or into its substance.

2.—Acute meningitis.

3.—Compression of the cord by tumours or by thickened dura mater as in spinal caries.

Hæmorrhage into the cord is very rare but may complicate a myelitis, or may be due to injury. The onset of paralysis is immediate, far more rapid than in myelitis, and no fever and no irritative phenomena occur, as in myelitis.

In acute meningitis and in tumours, the lesion being extra medullary, *irritative phenomena* are far more prominent and lasting than in myelitis, the motor and sensory nerves being first compressed, symptoms due first to their irritation and later on to their destruction, are observed before the cord is implicated.

Meningitis and myelitis are the only two acute spinal diseases beginning with fever, excluding poliomyelitis anterior acuta.

In meningitis—

1.—Pain in the back is much more prominent than in myelitis and is aggravated by movement.

2.—Contractures and cramps are present.

3.—Shooting pains in the limbs are prominent symptoms.

4.—There is rarely absolute paralysis as in myelitis, but paresis only.

5.—The sphincters are rarely paralysed, nor is cystitis present.

It is in the chronic form of myelitis that difficulty may occur in the diagnosis as to whether the lesion is primary in the cord or results from external pressure, and in these cases a careful examination of the spine should always be made, for spinal caries is frequently overlooked in cases of paraplegia.

The diagnosis of primary lateral sclerosis from descending sclerosis of the lateral columns following a chronic myelitis has already been mentioned, as has also the distinction between functional and organic paraplegias.

Acute Ascending Paralysis.—Landry's Paralysis.

"*Progressive paralysis*" (Graves) is a form of progressive paralysis commencing in the feet and passing upwards to the trunk and upper extremities, and finally causing death by implicating the medulla. The duration of the disease is usually only a few days, and it is characterised by the following negative features, there being :—

- 1.—No atrophy.
- 2.—No reaction of degeneration.
- 3.—No increase of reflex excitability.
- 4.—No contractures.
- 5.—No paralysis of the bladder or rectum.

This disease must be distinguished from sub-acute general spinal paralysis and from multiple neuritis.

The negative features of Landry's paralysis above enumerated distinguish it from these diseases.

Multiple Sclerosis.

In disseminated or multiple sclerosis the cord may or may not be affected, and if affected the symptoms vary according to the distribution of the patches of sclerosis.

The lesion may be confined to the postero-external columns of the cord and the case thus simulate tabes, or to the lateral columns (simulating lateral sclerosis), and causing a spastic paraplegia, the latter being the commonest form of cord lesion in this disease.

The presence of the cerebral symptoms characteristic of the disease would at once enable the diagnosis to be made.

Case of Disseminated Sclerosis.

A. S., a man aged 30, came to the Queen's Hospital in December last complaining of giddiness and difficulty in walking. He was a draper by trade and had lived a fast life. Eleven years ago he had a chancre of doubtful character and gonorrhœa.

There was no history of nervous disease in the family, and no history of injury or of lead or mercurial poisoning. The present illness came on gradually two years ago with stiffness of the arms and legs, and his gait gradually became affected.

Three weeks before coming to the hospital he suffered from diplopia, particularly when looking to the right or left.

He had suffered from frontal headache but had not vomited and his memory was good.

His gait was ataxic, he could not walk without a stick, and in walking widely separated his feet and frequently lurched to one side, the neck being held stiffly, and coarse tremor, especially of the head, being marked. The gait was not like that of tabes; he did not jerk out his legs, bringing the heels down violently.

He had never suffered from pains in the legs.

The pupils were unequal but they responded normally to light and accommodation, and clonic spasm of the iris was observed. Nystagmus was not present during ordinary movements but when the patient was directed to look to one side so as to strain the ocular muscles it was marked. These movements were taken as evidence of paresis of the 3rd nerve. There was no optic neuritis or atrophy. The patient suffered at times from vertigo, probably due to the ocular disturbance. The only sensory disturbance present was slight numbness of the fingers. The speech was unaffected and the only psychical disorder present was irritability and occasional loss of self-control and inability to bear the slightest noise, especially of children. Occasionally he had difficulty in passing his urine. The knee jerk was increased on both sides and there was a tendency to ankle clonus, but no rigidity or paresis of the extremities. There was marked tremor of the head, neck, and body when walking, and of the head when sitting

and standing, disappearing when lying down; also slightly of the left arm, observed when he raised a glass of water to his mouth.

The peculiar gait; the nystagmus with clonic spasm of the iris; the coarse tremor of the head and body ceasing during rest; together with the increase of the deep reflexes; leave little doubt as to the diagnosis. Cerebellar tumour, locomotor ataxy, and paralysis agitans are easily excluded.

Extra-Medullary Lesions.

Extra-medullary lesions are conveniently classified by Bramwell as follows :—

- | | | | | | | |
|----------------------------------|---|---|---|---------------|---|---|
| (i.) Meningitis | { | Leptomeningitis | { | Acute | { | |
| | | | | Chronic | | |
| | | (Inflammation of the pia and arachnoid) | { | Externa | | { |
| | | Pachymeningitis | | Interna | | |
| (Inflammation of the dura mater) | | Hæmorrhagica | | | | |
| | | | | Hypertrophica | | |

(ii.) Extra-Medullary Hæmorrhage.

(iii.) Extra-Medullary Tumours.

In the diagnosis of the nature of a paraplegia, as in the diagnosis of hemiplegia, the first question to decide is :—Is the paralysis functional or organic? If organic, is the lesion spinal or peripheral? If spinal, is the lesion *intra*-medullary or *extra*-medullary? Extra-medullary lesions irritate and compress the anterior and posterior nerve roots; therefore pain in the back, shooting pains, hyperæsthesia and anæsthesia, together with spasm and paralysis, occur.

Paraplegia occurs late in extra-medullary lesions, and is preceded by symptoms caused by irritation of the motor and sensory nerve roots.

Acute Spinal Meningitis.

Acute spinal meningitis is always diffuse and frequently associated with cerebral meningitis as in the epidemic form.

The characteristic symptoms are:—

1.—Pain in the back not increased by percussion but aggravated by movement.

2.—Symptoms denoting irritation of the motor and sensory nerve roots, such as twitchings, spasms, and rigidity, with shooting pains and hyperæsthesia.

3.—There is generally rigidity of the spine, with retraction of the head caused by spasm of the posterior cervical muscles and flexion of the thighs on the abdomen, and legs on the thighs as is observed in typical cases of epidemic cerebro-spinal meningitis.

4.—Later on compression of the motor and sensory nerves, by the inflammatory effusion, causes paralysis and anæsthesia, with abolition of reflexes.

5.—The cord may be affected by pressure, and partial but rarely complete paraplegia may ensue.

6.—The bladder and rectum may be paralysed if the nerves to these organs are implicated.

Should the inflammation extend upwards the intercostal and phrenic nerves may be involved and death result from asphyxia.

Acute spinal meningitis must be distinguished from acute myelitis (see myelitis) and from tetanus.

In tetanus:—

1.—There are no sensory disturbances—there is severe pain during the exacerbations but no marked hyperæsthesia or anæsthesia.

2.—The spasms are much more general and constant than in meningitis and trismus is present.

3.—The reflexes are much more exaggerated than in meningitis.

4.—There is less fever than in meningitis.

Chronic meningitis resembles acute meningitis in its symptoms except that the lesion is more localised, the fever less, and pressure on the cord leading to paraplegia is common.

It may be distinguished from chronic myelitis by the same features which distinguish acute meningitis from acute myelitis.

Chronic meningitis occurring in the lumbar region may cause loss of the knee jerk and pains similar to those observed in locomotor ataxy, but the eye symptoms are absent.

Pachymeningitis.

Pachymeningitis *externa* chiefly occurs in disease of the vertebræ. The inflammation is localised, the thickened patch of dura compressing the nerves and the spinal cord.

The surface of the cord being compressed there is frequently paraplegia with exaggeration of the deep reflexes without impairment of sensation or paralysis

of the bladder. In such cases always look for Pott's disease of the vertebræ.

Sensation may, however, be destroyed and the bladder paralysed in bad cases.

Pachymeningitis *interna hæmorrhagica*, like the cerebral form, is characterised by its liability to sudden hæmorrhage with production of paralytic symptoms. Apart from hæmorrhage the symptoms would be those of a chronic meningitis.

Pachymeningitis *interna hypertrophica*. This condition where the dura mater is greatly thickened, forming a ring around and compressing the cord, and the nerves coming off from it, usually affects the dura in the cervical region, and was first described by Charcot.

As in all extra-medullary lesions, symptoms due to irritation of the nerve roots are at first prominent, there being shooting pains in the neck and arms and hyperæsthesia with spasm, later on anæsthesia and paralysis with trophic disturbances in the area of distribution of the affected nerves.

The pressure of the thickened dura leads to a transverse myelitis with secondary degeneration, a spastic paraplegia being thus produced.

There is, in fact, a general paralysis, both arms and legs being affected, but wasting and contractures are confined to the upper extremities.

Pressure upon the cervical region of the cord causes paralysis of the upper extremities, "a cervical paraplegia" before the legs are affected, the nerves for the upper extremities being nearer the surface.

Amyotrophic lateral sclerosis can be distinguished from this disease by the marked sensory disturbances and progress to recovery, more or less complete in the latter.

Pott's disease, which might lead to symptoms exactly resembling this disease, is excluded by the absence of spinal tenderness, deformity, cachexia, fever, or signs of scrofula.

Tumour of the bones, membranes, or nerve roots might cause some difficulty, but the symptoms here progress steadily from bad to worse, and do not improve.

Case of Pachymeningitis Cervicalis Hypertrophica.

W. B., a boy, aged 18, was admitted into the Workhouse Infirmary, May 25th, 1883, with paralysis and tucking of both legs.

When 5 years of age, he fell down some steps on to the back of his head. Ever since he has had an internal strabismus of the left eye. Another fall two years previously to his admission, he says, caused the paralysis; in his opinion the paralysis commenced shortly after.

The patient had been in the General Hospital for twelve months before he was brought to the infirmary, and while there, both arms and legs were paretic and rigid. The paralysis began in the right arm; the onset was gradual, and attended with numbness, tingling, and loss of sensation.

There was great pain in the neck and scalp, for which a seton was introduced, and the neck was rigid, movement causing great pain. The pain was along the great occipital and third cervical nerves, upward towards the head, also down the shoulders.

Two or three months after the paralysis of the right arm, the left arm, both legs and back, began to get weaker, until the patient was completely paralysed.

The arms were rigid, with extreme flexion of the fingers and wrists. There was no paralysis of the bladder or rectum.

Five months before admission (in 1883) he began to recover the use of his arms, and they have been getting better ever since.

There was never any facial paralysis, or any cerebral affection or disease of the cranial nerves, no vertigo, delirium, or vomiting.

The legs were weak when the arms were affected. At first they were flaccid, but they gradually began to get stiff, and contracted and trembled. All four extremities were paralysed together, but the arms got better while the legs got stiff.

When admitted, the patient was much emaciated, had spasmodic internal strabismus of the left eye and torticollis, the head being twisted to the right. He could rotate his head more to the right than to the left.

No tenderness could be elicited by percussion over the cervical spines, and there was no fever or any sign of scrofula.

Both legs were paralysed and flexed at the hips and knees, with adduction of the thighs.

The arms had recovered power, but were still weak. There were no sensory disturbances.

Ankle clonus and front tap contraction were markedly present in both legs. On account of the contracture, the patellar reflexes could not be obtained.

The plantar reflexes were present; the cremasteric, abdominal and epigastric absent.

There was no sensory disturbance in the lower extremities, and no marked wasting.

The optic disc and fundus on each side was normal. The patient was treated with iodide of potassium, and counter-irritated over the cervical spine, and when examined in July, 1884, was much better, could rotate the head more, could use the right leg well, and there was little rigidity on this side, but still ankle-clonus.

The left leg was still tucked, and wasted with increase of reflexes.

He was well fed, and spent as much time in the open air as possible, going about on crutches. A shot bag was suspended to the ankle of the left foot, to diminish the contracture. On the 14th March, 1885, the patient, wishing to leave, was again examined. The chin was still slightly turned to the right, the reflexes (bicipital, &c.) in the upper extremities well marked, but there was no rigidity, and the grasp on both sides was very powerful.

Ankle-clonus had disappeared, and the contracture of the left leg was much diminished. He could not, however, put the left heel to the ground, there being talipes equinus. The left leg was considerably wasted. The circumference of the left calf was $10\frac{1}{2}$ in., of the right, 13 in.

This wasting could only be accounted for by supposing that an extension of the morbid process in the crossed pyramidal path to the anterior cornua had taken place.

The electrical reactions of the nerves and muscles of the left leg were normal.

As to the diagnosis of the case:—

Amyotrophic lateral sclerosis is excluded by the marked sensory disorders and progress of the case to recovery.

A transverse myelitis is excluded by the great predominance of the irritative phenomena, and absence of anæsthesia and complete paralysis of the leg, preceding the rigidity.

Progressive muscular atrophy, by the presence of rigidity and sensory disturbances.

Atrophy is said not to occur in the leg in this disease, but in this case it has occurred in the left leg. There is no doubt that the lesion was one of the spinal membranes, and to be certain of the diagnosis of pachymeningitis cervicalis hypertrophica, we must eliminate Pott's disease and tumour of the membranes.

Pott's disease is excluded by the absence of spinal tenderness, deformity, cachexia, and fever; also by the absence of scrofula.

Tumour is more difficult to eliminate, but the gradual recovery excludes it, so that there can be little doubt about the case. A traumatic form of the disease has been described, and this case was probably due to injury.

The position of the hands shows the lesion to have been in the upper half of the cervical enlargement of the cord.

Spinal Hæmorrhage.

Hæmorrhage may occur either in the substance of the cord (a very rare occurrence) or outside the cord into the membranes. Both varieties may result from traumatic violence, and are characterised by their sudden onset, with absence of febrile disturbance.

The one variety may be distinguished from the other in the same way that we distinguish extra-medullary and intra-medullary diseases generally.

Spinal Tumours.

Tumours also may be extra- or intra-medullary. A tumour may exist in the spinal canal without symptoms for a considerable time until it compresses the nerve roots and the cord, when the symptoms gradually increase in severity, paraplegia with contracture and severe cramps rapidly supervening.

A tumour in the cord, according to Sharkey, disturbs its function even from the first, but it may

continue to grow for a long time before it produces marked symptoms, which it does either by pushing the cord against the bone walls of the spinal canal or by exhausting the elasticity of the spinal membranes.

A tumour outside the cord will of course be likely to cause symptoms due to pressure on the spinal nerves.

The symptoms of an intra-medullary tumour will vary with its situation and with the extent of the transverse area of the cord involved.

CHAPTER XI.

DISEASES OF THE PERIPHERAL NERVES.



Peripheral Neuritis.

It is only during the last few years that lesions of the peripheral nerves have attracted the attention they deserve, and in fact demand. Peripheral neuritis was shown to be the lesion in alcoholic paraplegia in 1881, by Lancereaux, and since then Dreschfeld, Wilks, Broadbent, Buzzard, and others have added considerably to our knowledge of this subject.

There are many causes of peripheral neuritis in addition to alcohol, *e.g.*, lead poisoning, exposure to cold, traumatism, and general diseases, such as diphtheria, gout, diabetes, tuberculosis, syphilis, rheumatism, locomotor ataxy, and leprosy.

Peripheral neuritis occasionally occurs after typhoid and other specific fevers, and it may also be idiopathic.

The paralysis caused by the fumes of bisulphide of carbon probably depends upon a multiple neuritis.

Neuritis may be acute or chronic, of one nerve or of almost all the peripheral nerves, when a condition of general paralysis is present and the disease is termed multiple neuritis.

In the early stage of neuritis symptoms due to irritation of the nerve fibres, such as pain, hyperæsthesia, and other sensory disturbances, with cramps and muscular twitchings, occur; and later on symptoms due to destruction of the nerve fibres and complete cessation of their conductivity causing anæsthesia and paralysis.

Other symptoms are also usually present, since the muscles are shut off from their trophic nuclei in the cord there is muscular atrophy varying with the degree of damage to the nerve.

Qualitative electrical changes are always present in severe cases, the Faradic response being lost, and the reaction of degeneration present.

Trophic changes are often observed, the nails becoming brittle, ridged, and discoloured; the skin of the fingers and toes hairless and glossy.

Vaso-motor disorders are of frequent occurrence, consisting of patches of redness on the hands and feet, and œdema of these parts. Profuse perspiration either of the general surface of the body or limited to the feet also occurs.

The deep reflexes are abolished at an early period of the disease, the knee jerk being soon lost when the nerves of the lower extremities are affected, but the superficial reflexes may be retained.

The paralysis and anæsthesia resulting from peripheral neuritis is always most marked at the extremities of the limbs.

The extensor muscles of the fore-arm and leg being especially affected, there is dropped wrist and dropped feet. The latter is a very characteristic feature, the patient being quite unable to perform dorsal flexion of the foot, while he may be able to flex and extend the knee.

There is frequently tenderness along the nerve trunks and occasionally they may be felt to be enlarged.

In multiple neuritis both sides of the body are affected symmetrically, the numbness and paresis being always first noticed in the feet and hands, gradually spreading upwards.

The muscles of the trunk and those supplied by the cranial nerves may be affected and death may be produced by paralysis of the diaphragm and intercostal muscles, or from paralysis of the pneumogastric nerve.

The paralysed extremities are always flaccid at first, but contracture of the unopposed healthy muscles may occur in severe cases.

Paralysis never supervenes suddenly in peripheral neuritis, but is gradual in its onset and requires some time to become general, being usually preceded by more or less severe pains in the extremities.

Before the paralysis is complete incoordination may be marked and may exactly resemble that of locomotor ataxy.

Although the extensors of the legs and of the forearms suffer most, all the other muscles are weakened.

The lower are always earlier and more affected than the upper extremities.

The bladder and rectum are not affected in the majority of cases, but only in those rare instances in which the nerves to these viscera are diseased.

Bedsore are not usually present.

Sensory disturbances are present in very varying degrees, from total anæsthesia to the mere sensory disorder of "pins and needles."

The mental faculties are unaffected except in cases due to alcoholism, when there is nearly always loss of memory or other mental symptoms such as insomnia, delirium, &c.

Hyperæsthesia of the skin and muscular structure is generally marked, and on pinching the calf muscles considerable pain is elicited.

We must now consider the diagnosis of peripheral neuritis from lesions of the spinal cord and brain.

Monoplegia, paraplegia, hemiplegia, or paralysis of all four extremities may be cerebral, spinal, or peripheral.

In a cerebral monoplegia as in all cerebral paralyse—

- 1.—There are no qualitative electrical changes.
- 2.—Sensation is usually unaffected.

3.—The paralysis is followed by rigidity, with exaggeration of the reflexes.

4.—There is no rapid muscular wasting.

5.—The lesion is most commonly in the motor area of the cortex, when Jacksonian epilepsy may be present, but it may be in the internal capsule.

6.—We should probably have optic neuritis, headache, vertigo, or other cerebral symptoms present.

In a spinal monoplegia the lesion is usually one of the anterior cornua. The onset is sudden, and the reaction of degeneration present, but there are no marked sensory disturbances—the reflexes are abolished.

In a monoplegia from disease of the peripheral nerves, as we have already seen, sensory disturbances are prominent, the reflexes are abolished, and the reaction of degeneration is usually present.

Peripheral neuritis may also be distinguished from lesions of the anterior cornua of the cord by the fact that in the former case the distribution of the paralysis is anatomical, the muscles paralysed being in groups supplied by particular nerves, and the paralysis is gradual in its onset and progressive, while in the latter, muscles functionally associated are paralysed and the onset is sudden, the maximum of paralysis being quickly reached.

There is no difficulty in the *diagnosis* of well-marked cases of multiple neuritis, but in some cases there may be absolute motor paralysis without any

sensory disturbance, and all the muscles of one limb may be paralysed. It is a general rule that in inflammation and injuries of mixed nerves the motor nerve fibres suffer far more than the sensory, and therefore paralysis is usually far in excess of anæsthesia or other sensory disturbance. The nerves, moreover, may be affected beyond the points at which their sensory branches are given off, and thus motor symptoms alone are present, though the nerve is a mixed one. Moreover, in peripheral neuritis there may be complete motor paralysis without any electrical changes. In all cases tenderness along the nerve trunks should be searched for.

After diphtheria absolute paraplegia may occur without any sensory disturbance, and without electrical change, and it is difficult to decide whether the lesion is in the cord or in the peripheral nerves, but recovery is so rapid and complete that there can be little doubt that in these cases the lesion is in the peripheral nerves.

Paraplegia may be cerebral, spinal, or peripheral, as may hemiplegia, and the diagnosis can be made on the same grounds as in the case of a monoplegia or paralysis of one extremity only.

Peripheral neuritis must be distinguished from—

- 1.—Locomotor ataxia.
- 2.—Polio-myelitis anterior acuta.
- 3.—Myelitis.
- 4.—Meningitis.
- 5.—Subacute general spinal paralysis.

6.—Acute ascending paralysis or Landry's disease.

It is highly probable that many cases of peripheral neuritis, due to alcohol or other cause, have been diagnosed as cases of locomotor ataxia, and their cure as that of this hopeless disease. Loss of the knee-jerk, pains, and ataxy are present in both diseases, but the pupil symptoms of locomotor ataxia are not present in peripheral neuritis.

Qualitative electrical changes if present would at once distinguish them. Before a diagnosis of locomotor ataxia is made, peripheral neuritis from alcohol, diphtheria, &c., must be carefully excluded.

In locomotor ataxia, peripheral neuritis occasionally occurs, and is the cause of such symptoms as perforating ulcer of the foot, optic atrophy, &c.

In paraplegia due to myelitis, bed sores, bladder and intestinal troubles are almost invariably present, while in peripheral neuritis these symptoms are very rarely present.

Spinal meningitis or pachymeningitis closely resembles peripheral neuritis, in fact, the nerves themselves are affected, but the marked pain in the back, aggravated by movement, together with retraction of the head and muscular twitchings, distinguish the former disease.

Subacute general spinal paralysis is not attended with any sensory disturbances, and the distribution of the paralysis is that peculiar to disease of the anterior cornua; muscles functionally associated being

paralysed. In this disease, moreover, the order in which the muscles are attacked is different from that observed in neuritis. When the spinal disease commences in the lower extremity of the cord and spreads upwards, the paralysis is first observed in the foot muscles, and then attacks in succession the muscles of the legs, thighs, buttocks, and trunk. Later on the intercostal muscles are paralysed, and the intrinsic muscles of the hand. The latter, which derive their nervous supply from the first dorsal nerve, are affected before the extensor muscles of the wrist and fingers (which are supplied by the seventh cervical nerve). Now, in multiple neuritis the paralysis, which first attacks the feet and then the legs and thighs, skips the trunk muscles and affects the extensors of the wrists before the small muscles of the hand.

Landry's disease is unattended with electrical alterations or sensory disturbances.

Alcoholic Paralysis.

Dr. Dreschfeld ("Brain," Vol. VII.) divides alcoholic paralysis into two types—*alcoholic ataxia* and *alcoholic paralysis proper*.

In alcoholic ataxia, which occurs chiefly in men, the symptoms greatly resemble those of locomotor ataxia, there being marked incoordination in the lower extremities, lancinating pains, and loss of the knee-jerk, without paralysis or atrophy. But the history of alcoholic excess, of morning sickness

or hæmatemesis, and the presence of mental symptoms, such as loss of memory, together with the absence of myosis, of the Argyll-Robertson pupil, of arthropathies, and of visceral crises, suffice to enable us to distinguish between ataxia due to chronic alcoholism and that due to tabes dorsalis. Moreover, in the former the ataxia disappears and the knee-jerk returns when alcohol is entirely withheld. The symptoms in the ataxic form of alcoholic paralysis are due to a multiple neuritis, especially of the sensory nerves, and these cases differ from those of the second type in the absence of hyperæsthesia, hyperalgesia, paralysis, and atrophy.

The second type of alcoholic paralysis is the more common, occurring chiefly in females who have previously suffered from symptoms of chronic alcoholism. Sensory disturbances are well marked, there being extreme hyperæsthesia of the feet and lower extremities, which may later on be followed by anæsthesia and analgesia. Lancinating pains are complained of, especially in the legs, but often in the upper extremities also. The calf muscles are exquisitely tender when grasped, and tenderness is found in the course of the nerve trunks. A remarkable perversion of the sense of temperature has been observed in the subjects of alcoholic paralysis, all objects in contact with the skin appearing cold. Paralysis chiefly attacking the lower extremities, and always commencing and being most marked in them, is present. The extensor muscles of the legs suffer

most, so that the feet are dropped, the patient being quite unable to perform dorsal flexion of the feet. The arms are affected later than the legs, and to a much less degree, the extensor muscles here also suffering most, dropped wrist being frequently present. The paralysis may be general, and in those cases where the extensor muscles only are paralysed evidence of weakness in the other muscles is present. Marked muscular atrophy is usually present, together with qualitative electrical changes constituting the R. D.

Vasomotor changes as shown by redness and oedema of the hands and feet also occur.

Purpura has been observed.

The skin may present the glossy appearance first described by Paget, but it is often harsh and dry. The nails are frequently affected, being curved, ridged, and brittle.

Emotional alterations, impairment of memory, insomnia, restlessness, delirium, and hebetude, together with digestive disturbances such as morning sickness or hæmatemesis, are characteristic features of chronic alcoholism; and when occurring with ataxy or paralysis render the diagnosis of the nature of these two symptoms certain.

Dr. Dreschfeld describes a peculiar form of delirium present in the subjects of alcoholic paralysis; the patient although unable to move saying that he gets up every day or that he has been out to a certain place.

I can confirm this from my own experience, for in one case of cirrhosis of the liver, with general emaciation and debility, the patient would assure me that he had walked several miles that morning, and was apparently rational in all else he said.

In another case of cirrhosis of the liver the patient used to tell me that she had just been to the "Rose and Crown," for a "noggin." She used to get out of bed and shake the other patients, telling them it was "time to open."

Dr. Dreschfeld examined the spinal cord and peripheral nerves in two fatal cases. He found that the cord was healthy but the peripheral nerves which were examined, namely, the two sciatics, the musculo-spiral and the anterior crural were found to be diseased, the myeline and axis cylinders being broken up.

Diphtheritic Paralysis.

In many of its features diphtheritic paralysis very closely resembles alcoholic paralysis, but it is rarely the case that the symptoms are so unequivocally the result of peripheral neuritis in the former as in the latter complaint. There are several degrees in the intensity of the nervous disturbance after diphtheria, which may be grouped as follows:—

1.—The knee jerk is very frequently lost and there may be no other symptom present.

This loss of knee jerk occurring as it does so commonly after diphtheria and often unaccompanied by any other symptoms seems to be a

point in favour of a peripheral lesion, especially as in these cases the jerk reappears again in a few weeks.

2.—In other cases, in addition to the loss of the knee jerk, there is ataxy with more or less general paresis without sensory alterations.

3.—In some cases there is absolute paraplegia with loss of knee jerk without any disturbance of sensation and without electrical alterations.

The symptoms in these cases being those of an acute lesion of the multipolar nerve cells of the anterior cornua of the cord. Since, however, recovery is usually rapid and complete, it is difficult to suppose that the lesion can be in the cord, for nerve cells do not recover from injury so easily as nerve fibres, and the former when once destroyed are not regenerated, while the latter are readily regenerated.

4.—In other cases of diphtheritic paralysis, sensory disturbances, hyperæsthesia, anæsthesia, tenderness along the nerve trunks and shooting pains are present. These cases cannot be due simply to lesion of the anterior cornua of the cord.

In rare cases we have a condition exactly like that observed in severe cases of alcoholic paralysis, there being dropped feet and hands, sensory disturbances, tenderness along the nerve trunks and of the muscles, with wasting and qualitative electrical changes, there being no doubt that in these cases the lesion is a neuritis of the peripheral nerves.

It is difficult to suppose that the lesion in the cases that recover is one of the anterior cornua of the spinal cord, while in those rare and severe cases which often end in incomplete recovery, the lesion is one of the peripheral nerves only.

It is more reasonable to suppose that the lesion in all cases of diphtheritic paralysis is a peripheral neuritis, but that in the former type of case the lesion is a slight one, and in the latter type a severe one. In injury to a mixed nerve it is well known that there may be paralysis without anæsthesia or qualitative electrical change, provided the injury is a slight one. In more severe cases, anæsthesia, wasting, and the reaction of degeneration are met with.

After diphtheria, paralysis of the ocular muscles and of the soft palate are of common occurrence, and these paralysees are certainly in favour of the peripheral nature of the lesion in diphtheritic paralysis. Buzzard, Mendel, and Charcot are inclined to the view that the lesion in diphtheritic paralysis is a neuritis of the peripheral nerves. The cases on record where neuritis of the peripheral nerves has been found after death are of course very few, as diphtheritic paralysis is rarely fatal.

A case of acute multiple neuritis following diphtheria was sent to me by Mr. Latham, of West Bromwich, in July last. The patient (Samuel Westwood), aged 23, was admitted into the Queen's Hospital, July 19th, 1886. His father died of diabetes, but there was no other family history of importance.

Previous to this illness he had never had anything the matter with him except bad colds. Thirteen weeks before his admission, the patient's sister and niece had diphtheria, and he himself had a sore throat with membranous exudation.

Mr. Latham tells me that he attended the sister and niece, the latter of whom died from diphtheria, and that he did not see the patient, who lived in the same house, for ten days after the onset of his sore throat, but that the sister saw ash-coloured spots on the tonsils and soft palate. Three weeks after the onset of the sore throat the patient noticed that his legs were getting weak. He gradually became unable to walk, and in a month completely lost all power over his feet. As his legs began to get weak he suffered from very severe pains shooting down them, which occurred paroxysmally; his legs were also very tender and he could not bear them rubbed. Shortly before admission his arms began to get weak also, but he had no pains in them. On admission the patient was found to have lost all power over his feet, which were dropped; he could bend and straighten the knees, but the feet hung helplessly, being perfectly flaccid.

There was also drop-wrist on both sides, and the grasp was very feeble, that of the right hand tested by the dynamometer being 8 lbs., that of the left 10 lbs. The muscles of the legs and forearms were much wasted, and the thenar and hypothenar eminences almost effaced. The fingers were flexed

slightly at all three joints and could not be straightened voluntarily. The plantar reflex and the knee jerk were absent on both sides. The cremasteric

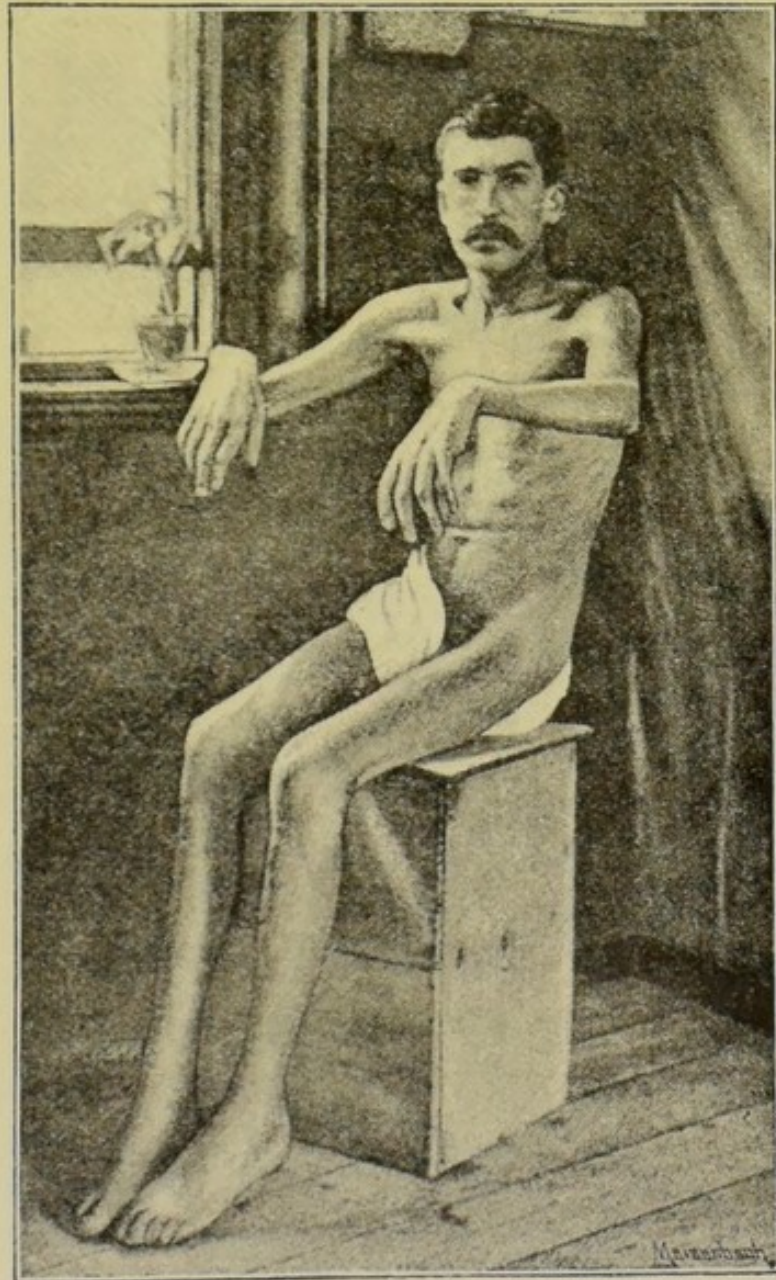


FIG. 21.

ACUTE MULTIPLE NEURITIS DUE TO DIPHTHERIA.

abdominal and epigastric reflexes were present on both sides. The following sensory disorders were present. The power of localisation of tactile impressions

was not much impaired, though the patient had to consider a short time before he could answer. Tactile and painful impressions were evidently delayed in their transmission to the brain. One prick with a pin produced about two seconds later a sense of contact, followed after a short interval by a sense of pain. There was decided analgesia and diminution of thermal and muscular sense. The patient complained of pains in his legs, with numbness and tingling of the hands and feet. The calf muscles were very tender, and when pinched caused him great pain; he could scarcely bear the contact of the bed-clothes. There was well-marked tenderness along the course of the great sciatic and internal popliteal nerves. The idio-muscular contractility was increased, fibrillary contractions being produced by filipping the wasted muscles. The feet were cold and the skin red and œdematous. The skin of the fingers was tense and shining, and the nails presented several white transverse lines. There was no paralysis of the internal or external muscles of the eyes or of the soft palate, but anæsthesia of the throat and larynx was well marked, it being possible to put the finger into the larynx without discomfort. There was no albumen in the urine, no bed sore, and no trouble with micturition or defæcation.

Electrical Reactions. — The Faradic current applied to the nerves, motor points and muscles of the legs produced a very slight contraction of the gastrocnemii, but none of the other muscles

either at the back or front of the legs, or of the foot, responded in the slightest.

Galvanisation of the great sciatic nerve produced no contraction of the muscles supplied by it. Galvanisation of the gastrocnemii with twenty cells caused sluggish contractions, the anodic closure contraction exceeding the cathodic closure contraction. With twenty-four cells the anodic closure contraction also predominated in the extensor muscles of the legs. The response of the muscles in front of the thighs was diminished on both sides, the anodic closure contraction exceeding the cathodic closure contraction. The hamstring muscles responded well, CCC exceeding ACC. Faradism applied to the extensor muscles of the forearms caused no contraction on the right side and only a feeble one on the left; the supinator longus and the flexor muscles acted well on both sides. The thenar and hypothenar muscles and the interossei did not respond at all on either side. With Galvanism the extensor muscles of the forearm responded to the current of eighteen cells, ACC exceeding CCC. The short muscles of the thumb also presented the reaction of degeneration.

The patient was ordered strychnia, the dose being gradually increased, and the galvanic current was applied daily to the affected muscles. He could not bear massage on account of the extreme tenderness of the muscles, and he had restless nights from the pain in the feet.

On the 26th of July it was found that the extensor muscles on the back of the left forearm responded well to Faradism, but there was still no response on the right side.

On the 29th the extensor muscles on both sides responded well to Faradism, but there still was no Faradic response in the muscles of the legs or in the thenar and hypothenar muscles.

On the 1st of August the drop-wrist had disappeared, the patient had much greater power with his hands and also slight voluntary movement in his feet.

On the 5th a trace of albumen was found in the urine. The patient was now taking twenty-two minims of Liq. Strych. (B. P.) In the short thumb muscles CCC now exceeded ACC.

On the 8th it was found that the hyperæsthesia of the calf muscles was not so marked as previously, but there was still much tenderness.

On the 10th he complained of twitching and jumping of the legs. The tenderness formerly present along the great sciatic and internal popliteal nerves was not nearly so marked.

On the 20th the muscular tenderness had disappeared, as had also the tenderness in the course of the nerves. The improvement followed the application of blistering fluid along the course of the nerve trunks. He was now taking Liq. Strych., $\text{m}30$, thrice daily.

On the 28th there was marked hyperidrosis along the inner side and under surface of both feet, and this had been present for some days. The reflexes remained in the same condition, and there was no Faradic response in the extensor muscles of the legs or in those of the thenar eminences. He was now taking Liq. Strych., $\text{m}36$, thrice daily, and it was not thought advisable to increase the dose further.

September 4th. Sweating of the feet still continued and was accompanied with extensive desquamation, small ulcers forming on one or two toes, but these quickly healed.

September 16th. The hands had desquamated like the feet. The fingers were a little flexed still and could not be quite straightened, but the tendency to contracture was much less than it had been. The œdema of the feet had almost disappeared and the sweating was much less.

On the 17th the following were the electrical reactions with Faradism:—In the right forearm the extensores ossis, primi and secundi responded well, the extensor indicis, ext. com. dig., ext. carpi ulnaris very feebly, the ext. min. dig. did not respond at all. The muscles on the front of the right forearm responded normally. The thenar muscles gave no response, but the hypothenar muscles and the interosse responded well. On the left side the thenar muscles did not respond, with the exception of the adductor pollicis. All the muscles on the back of the forearm responded. In the right leg

there was no response of the muscles on the front and outer side, and an almost imperceptible response only in the calf muscles. Similarly in the left leg.

With Galvanism.—The muscles on the posterior surface of the right forearm responded, A C C exceeding C C C. The contractions of these and of the thenar muscles were sluggish and prolonged. The muscles of the right leg responded well, in the extensors C C C exceeded A C C, in the peronei A C C predominated.

October 2nd. The thenar muscles on both sides responded feebly to Faradism. The clawed condition of the hands was not nearly so marked as it had been.

At the present time (December 2nd) the clawed condition of the hand is quite gone, and he can extend the wrists. The grasp of the right hand by the dynamometer equals 50lbs., that of the left hand 60lbs. The feet are still dropped, and there is no Faradic response with a strong current in the extensor muscles of either leg. The peronei on the left side and the gastrocnemii on both sides respond feebly. Sensation is still a little delayed but has much improved. The knee-jerk and plantar reflexes are still absent. The reaction of degeneration is well marked in the thenar muscles and extensor muscles of the forearms. In the extensor communis digitorum and extensor proprius pollicis of both legs the reaction of degeneration is also well marked. The peronei respond better than the extensors, C C C predominating.

As to the diagnosis in this case. Is the lesion spinal or peripheral? A lesion of the anterior cornua of the cord would account for the paralysis, rapid wasting, and electrical alterations present, but would not explain the sensory disturbances so marked in this case, nor the tenderness of the nerves or muscles. Moreover, in this case it is the peripheral portions of the extremities only which are affected, the hands and forearms, the feet and legs; while the arms and shoulders, thighs and hips are unaffected. The disease also commenced in the feet and legs, missed the trunk, and appeared in the hands. This is not what we should have expected in a spinal lesion, but it is just what occurs in multiple neuritis.

In an acute lesion of the anterior cornua, moreover, the paralysis at once reaches its maximum; in this case the disease was progressive, and after the legs were affected it was some weeks before the hands suffered; so that polio-myelitis anterior acuta must be excluded. Subacute general spinal paralysis (a disease of the anterior cornua) is also excluded by the sensory disturbances present.

In acute ascending paralysis (Landry's paralysis) there are no sensory disturbances and no electrical alterations. The bladder and rectum being unaffected and there being no bedsores, no fever, no girdle pain or hyperæsthetic zone, we can exclude myelitis. Spinal meningitis or pachymeningitis might give rise to symptoms exactly resembling those of multiple neuritis, but there was in this case no pain in the

back, no pain on movement of the spine, no rigidity and no retraction of the head. I think there can be little doubt that the case is one of multiple neuritis.

Neuralgia.

Neuralgia consists in severe periodical paroxysms of pain limited to the course of a sensory nerve, not being due to any evident organic lesion of the nerve.

Tender spots, first described by Valleix, may be found at various points in the course of the affected nerves, where the nerves pierce muscles or fasciæ, or emerge from a bony canal, or where they can easily be compressed, as when they lie in contact with bone. Various symptoms are associated with neuralgia, especially those due to vaso-motor changes.

Neuralgia may affect any of the sensory nerves of the body, either of the trunk and limbs, or of the viscera.

We must distinguish between neuralgia of peripheral and of central origin.

In peripheral neuralgia:—(1) painful points are present; (2) remedies applied peripherally relieve; (3) the pain is limited to definite branches of the nerves.

In central neuralgia tender spots are absent. There are usually mental symptoms and the pain is not so strictly limited to definite branches of one nerve but affects several of the cranial nerves.

Neuralgia is usually unilateral, when bilateral organic mischief must be suspected. The presence

of intercostal neuralgia, especially when bilateral, should lead you to explore the spine carefully for Pott's disease, &c.

Sciatica is usually unilateral; when bilateral, suspect the presence of some organic disease of the spinal cord, such as locomotor ataxia or ataxic paraplegia; a malignant or other growth compressing the cauda equina might occasion bilateral sciatica.

Bilateral sciatica is occasionally present in diabetes.

In all cases of obstinate sciatica examine the patient per rectum to detect if present any swelling in the pelvis.

In an old man who suffered from intense pain along the left sciatic nerve a swelling was detected on the left side of the sacrum during life, there were no tender spots in the course of the sciatic nerve, and the limb gradually emaciated without actual paralysis or anæsthesia.

At the autopsy an osteo-sarcoma of the sacrum was found, the left sacral plexus being compressed and the left sciatic nerve extremely atrophied.

Case of Plantar Neuralgia.

H. E., a single woman, aged 38, employed as a domestic servant, was admitted into the Workhouse Infirmary, April 9th, 1885, complaining of pain in the soles of the feet, and inability to walk or stand on account of the pain.

Both her parents had suffered from rheumatism, and two of her brothers have had rheumatic fever.

The patient has had jaundice twice, otherwise has enjoyed good health, but is not robust.

About four years ago she had pains in both her feet similar to the present illness, but soon recovered. Three weeks before admission she got her feet damp, and the next day they were very painful. There were shooting pains confined to the soles of the feet near the heel. The pain was much aggravated by walking or standing, and much relieved by lying down.

When examined on admission there was considerable hyperæsthesia of the soles of both feet and tenderness. The hyperæsthetic area was close to the heel, and measured an inch and a-half in length and one inch in width on each foot. When the feet were allowed to hang down the painful areas became red, and the veins stood out, and free perspiration followed. The same phenomena occurred after walking a few yards. There was a tender spot behind the internal malleolus and another over the heel. There was no special tenderness, no pain in the back, or any sign of disease of the spinal cord or its membranes. There was no pain in the joints, nor any heart murmur. The urine was normal. The patient was kept in bed, and a blister applied behind the internal malleolus of each foot. Simple rest in bed relieved the pains, but she always suffered from them at night. Repeated blistering finally caused complete cessation of the pains.

At the present time (May 9th) hanging the feet out of bed does not cause them to become red or to perspire, and walking does not cause pain.

For the time at any rate the patient is well.

This disease was first described by Weir Mitchell, and is said to be rare in women.

On the 20th of May the patient left the Infirmary perfectly well.

On the 25th the patient, having tramped about and having been much exposed, was re-admitted, the pains having returned, but in a less degree than when first admitted.

Neuritis of a nerve must be distinguished from neuralgia :—

In neuritis.	In neuralgia.
1.—The pain is continuous.	The pain is intermittent.
2.—Tenderness extends along a considerable length of the nerve.	Tenderness is present at certain spots only.
3.—The nerve may be felt to be thickened.	
4.—Anæsthesia and paralysis occur only in neuritis, though paresis is sometimes present in sciatica.	

Injuries of Peripheral Nerves.

As has been elsewhere stated, the electrical reactions in diseases of the peripheral nerves vary according to the extent of the lesion.

When a motor nerve is divided there is total loss of response to both currents when applied to the nerve, and with Galvanism applied to the muscle the "reaction of degeneration" is obtained.

The same changes are found after severe injury to a motor or mixed nerve.

When the degree of injury is less there may be complete paralysis with loss of Faradic irritability but with normal Galvanic reactions.

In still a slighter degree of injury there is paralysis but with normal electrical reactions.

The prognosis in cases of injury to nerve trunks can thus be accurately estimated by the electrical reactions.

Case of Division of the Median Nerve.

The patient, a medical student, was reaching a bottle of soda-water out of its case, when the bottle burst in his hand, severely cutting his right wrist in front and in the middle line.

This occurred on the 25th of September, 1885.

At the time of the accident he perceived, as it were, a great rush of blood into the hand, which felt as though it was being greatly distended, and became cold and numb.

During the dressing of the wound, and every time the pad was used to swab up the blood so as to look for a divided nerve or artery, the pain was most acute, and was probably due to the pad coming in contact with the divided nerve.

The nerve was not seen during the treatment of the wound. The hand was put in splints for three or four days; and on their removal it was found that the muscles of the hand supplied by the median nerve

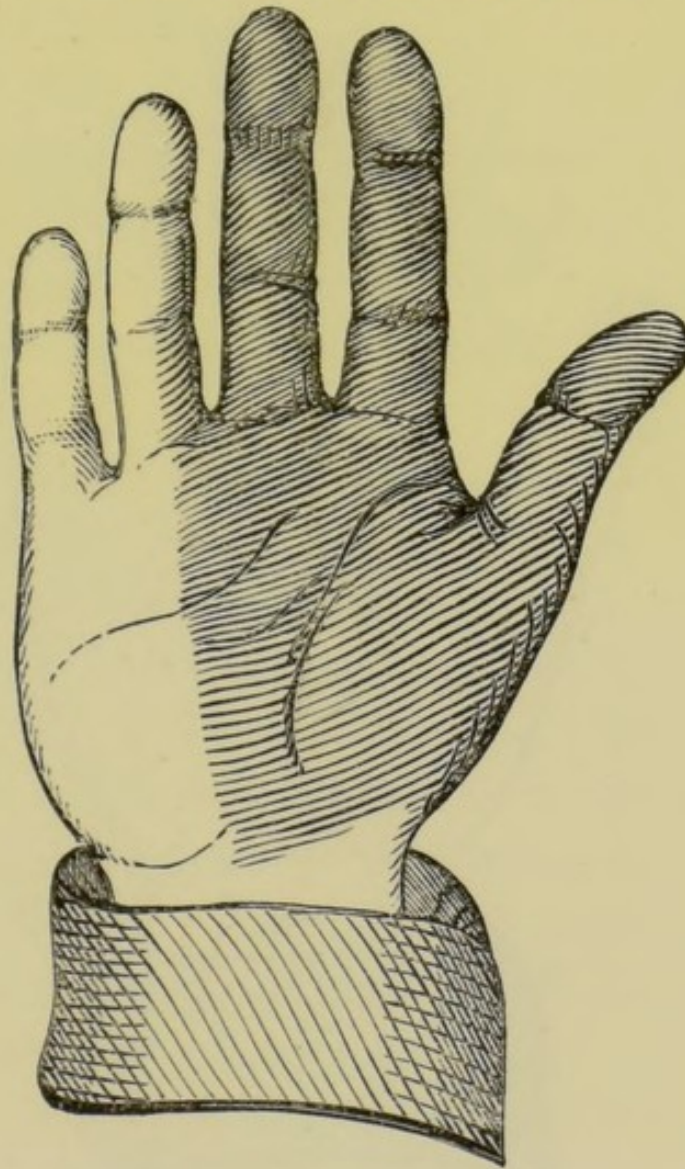


FIG. 22.

PALMAR SURFACE THREE DAYS AFTER ACCIDENT.

The Shaded portion represents complete Anæsthesia.

were paralysed, and that there was total anæsthesia in the area of distribution of its sensory branches.

Three days after the accident there was total anæsthesia of the radial half of the palm and thenar

eminence, the anæsthetic area being sharply defined and not gradually shading off; it extended in the palm to a line contiguous with the axis of the ring-finger.

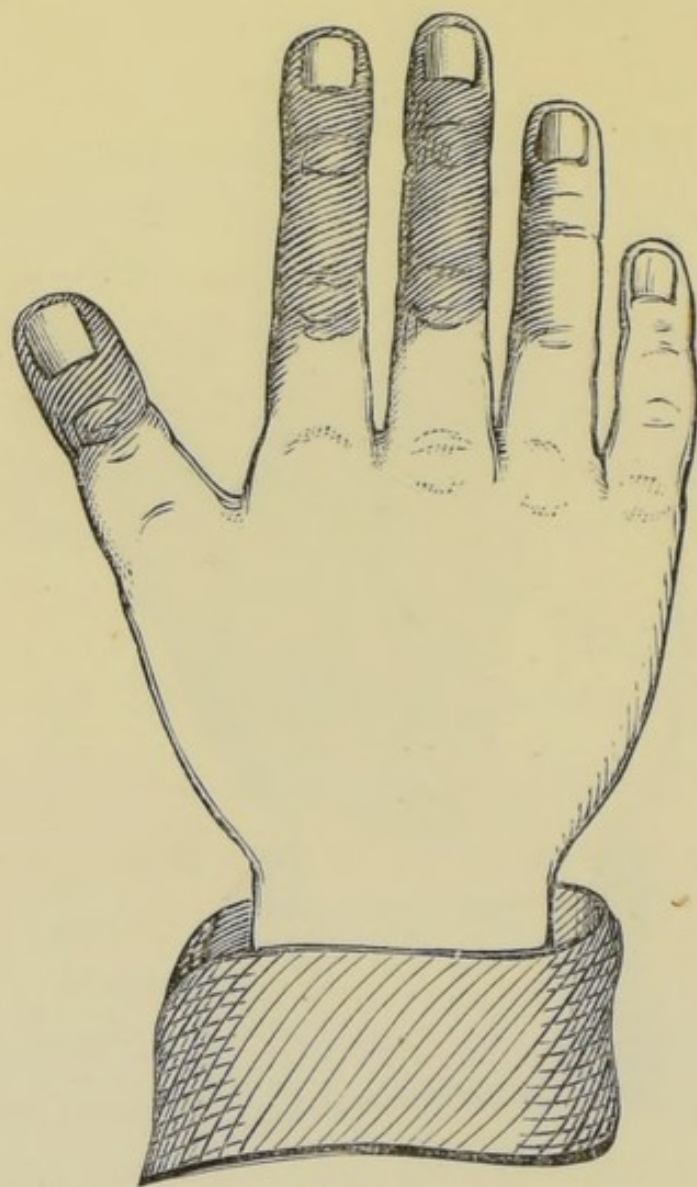


FIG. 23.

DORSAL SURFACE THREE DAYS AFTER ACCIDENT.

The Shaded portion represents complete Anæsthesia.

There was also complete anæsthesia of the palmar surfaces of the thumb, index, middle, and radial half of ring-finger.

On the dorsum of the hand there was total anæsthesia of the ungual phalanx of the thumb and of the second and third phalanges of the forefinger and middle finger, and of the radial halves of the same phalanges of the ring-finger.

A few days after the accident he began to feel sharp shooting-pains in the hand and fingers, which would come on suddenly and as suddenly disappear. These pains frequently prevented sleep, but after lasting about a month they gradually ceased.

When he attempted to move the fingers he was always troubled with a sharp pain of a tearing character, which would at times extend into the forearm; this was especially acute on moving the index-finger. This pain also gradually ceased.

Since the accident there has been a great diminution of temperature in the three and a half fingers, and during the cold weather he had great difficulty in keeping them warm. After exercise he frequently noticed the little and half the ring-finger and the inner part of the hand to be covered with perspiration, while the rest of the palmar surface of the hand was quite dry.

At the present time (February), nearly five months after the accident, there is a scar in front of the right wrist passing obliquely across the tendon of the flexor carpi radialis and median nerve. The scar is very sensitive when touched. A small bulla, containing blood, is seen on the inner side of the terminal phalanx of the index-finger.

No changes in the nails are to be observed. He has recovered power to a considerable extent, being able to write with the right hand; but the grasp is much weaker than on the left side.

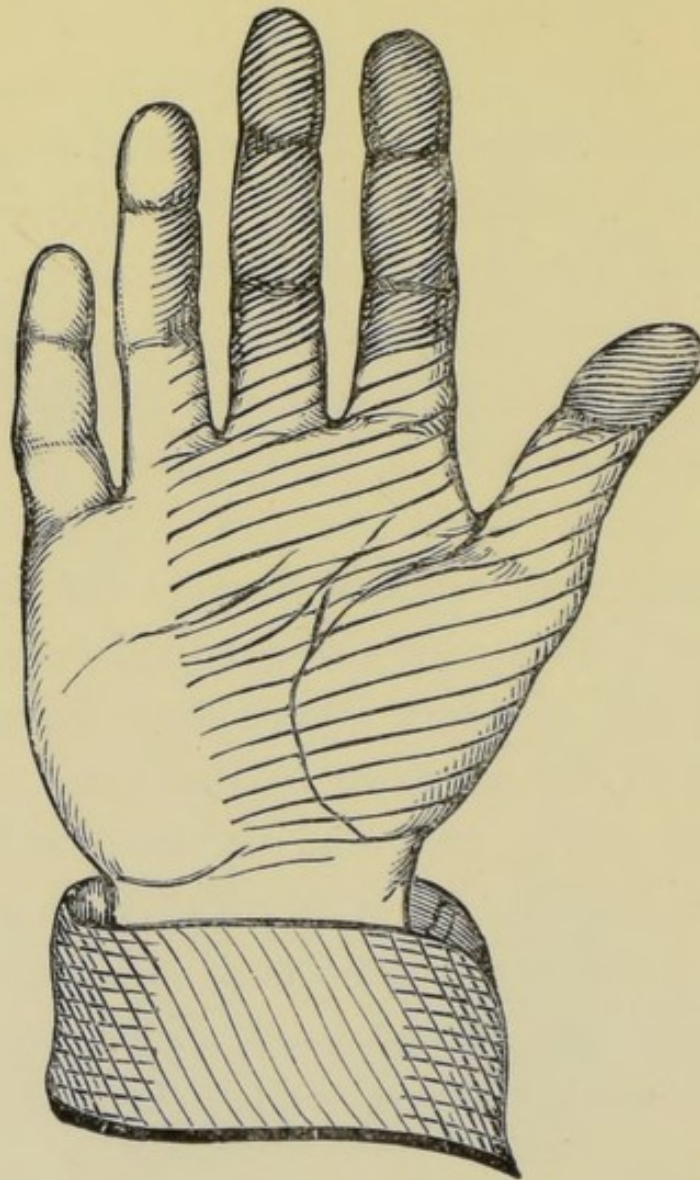


FIG. 24.

PALMAR SURFACE FIVE MONTHS AFTER ACCIDENT.

The portion thickly Shaded represents the Anæsthesia; the thinly Shaded portion Hyperæsthesia.

There is wasting of the thenar eminence on the right side. The circumference of the base of the right thumb being five inches, that of the left, five

and a half. The response of the muscles of the hand supplied by the median nerve is much diminished to Faradism. To Galvanism the response is increased, the contractions being slow and prolonged, the anodic closure contraction predominating.

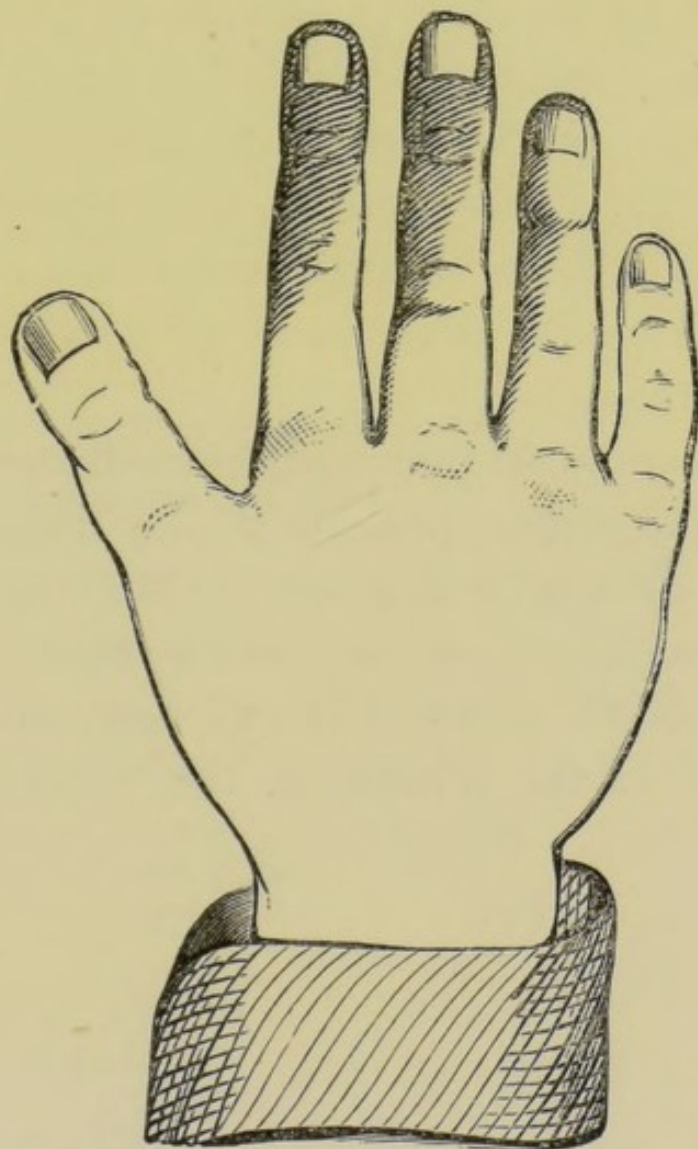


FIG. 25.

DORSAL SURFACE FIVE MONTHS AFTER ACCIDENT.

The portion thickly Shaded represents the Anæsthesia; the thinly Shaded portion Hyperæsthesia.

The distribution of the anæsthesia at the present time, nearly five months after the accident, is as

follows. The area of the right palm previously anæsthetic is now everywhere hyperæsthetic, a slight touch causing pain, the pain radiating into the fingers.

The hyperæsthesia extends over the palmar surface of the first phalanx of the thumb and encroaches on the first phalanges of the forefinger, middle finger, and ring-finger.

The skin of the rest of the phalanges is still anæsthetic on slight pressure, but hyperæsthetic on deep pressure, the pain radiating into the fingers.

On the dorsum of the hand the following changes have occurred:—

The anæsthesia previously present on the dorsal aspect of the ungual phalanx of the thumb has disappeared. The only alteration in the other fingers is that the anæsthesia of the middle phalanges of the fore and middle fingers extends now only over the radial half of the phalanges, the ulnar half being normal.

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