A TEXTBOOK

OF

NERVOUS DISEASES

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LONDON

J. & A. CHURCHILL

7 Great Marlborough Street

1910

PRINTED BY
SPOTTISWOODE AND CO. LTD., COLCHESTER
LONDON AND ETON

PREFACE

The aim of this book is to provide the practitioner and senior student with a short and practical account of the Diseases of the Nervous System. It is not intended to take the place of the larger and more complete text-books on Nervous Diseases.

In classifying the diseases we have adopted an arrangement based upon practice and personal experience, which we hope may prove useful to those for whom the book is primarily intended.

Attention has especially been given to the clinical description of the several disorders—more particularly from the point of view of diagnosis; but the etiology, pathology, prognosis, and treatment have each received consideration.

The limitation of the size of the book has made necessary the omission of certain of the disorders usually described in treatises upon the nervous system—such as exophthalmic goitre, myxœdema, and acromegaly. For the same reason extensive reference to recent literature has been found impossible.

We desire to record our thanks to our colleagues on the staff of the National Hospital for the Paralysed and Epileptic, Queen Square, London, for permission to make use of and to publish photographs of their cases.

We wish warmly to express our indebtedness to Dr. Farquhar Buzzard for his advice and suggestions in the methods of

classification. We are also indebted to Dr. Logan Turner, of Edinburgh, for revising the paragraphs upon the examination of the ear and its diseases.

Most of the photographs have been prepared by Dr. S. A. K. Wilson, registrar to the hospital; but some have been given by Dr. Byrom Bramwell, Dr. George Gibson, Dr. Gordon Holmes, and Dr. Foster Kennedy. Dr. Gibb has rendered assistance in the preparation of the index.

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HARLEY STREET, LONDON, W.
Jan. 1910.

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LIST OF ILLUSTRATIONS

Immediately in front of the precentral area and corresponding to the posterior ends of the first, second, and third frontal gyri, and a considerable part of the marginal gyrus, is the 'intermediate precentral area' of Campbell.[1] In structure it resembles the precentral area, but contains fewer nerve fibres and no Betz cells. (Figs. 2 and 3.)

The precentral area is that portion of the cerebral cortex in which crude voluntary movements of the body and limbs are represented. Its electrical stimulation is followed by clonic convulsions of the body and limbs on the opposite side. The

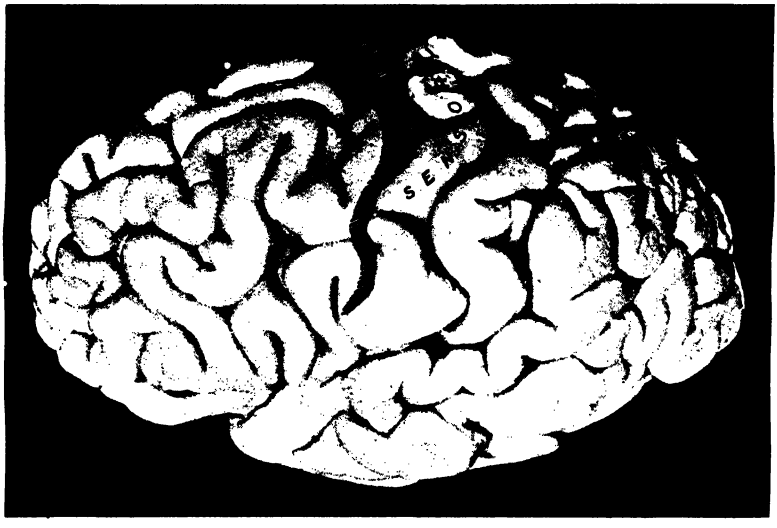


FIG. 1.—The convexity of the left cerebral hemisphere, showing the motor area (shaded) in the front of the fissure of Rolando and the sensory area of the post-central convolution.

'intermediate precentral,' or higher motor (psychomotor) area is regarded as presiding over more specialised or skilled movements than the precentral zone, as in it are contained the centres for articulatory speech and writing.

The *pyramids* or *pyramidal tracts* originate in the grey cortex of the precentral area, and are composed of fibres arising from the large giant cells of that area. These fibres descend through the centrum ovale, form a portion of the corona radiata and reach the internal capsule, where they

[1] Campbell, *Localisation of Cerebral Function*, 1905.

occupy a position partly at, and partly behind, the 'knee' of that structure. The arrangement of the fibres in the internal capsule is such that those which come from the upper cortical areas lie posteriorly, and those from the lower areas anteriorly.

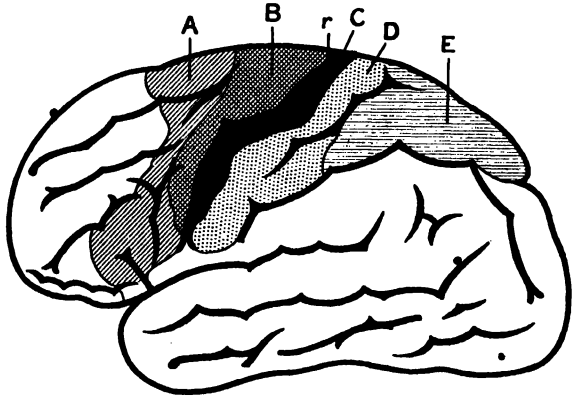


FIG. 2.—The motor and sensory areas of the convexity of the brain according to Campbell. *r*, the fissure of Rolando. *A*, intermediate precentral area. *B*, precentral area. *C*, post-central area. *D*, intermediate post-central area. *E*, parietal area.

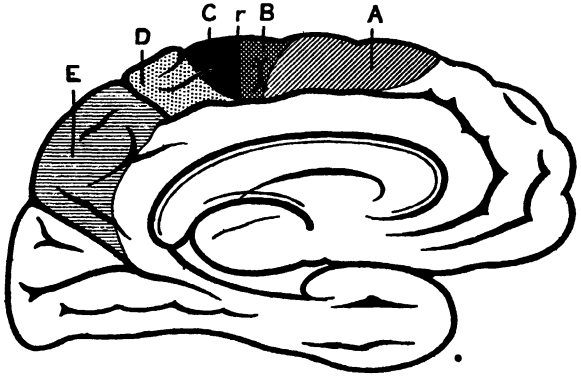


FIG. 3.—The motor and sensory areas upon the mesial surface of the brain, according to Campbell. The lettering indicates the same area as in fig. 2.

Anterior to, and in the region of the 'knee' are the fibres for the eyes, head, tongue, and face; in the posterior limb from before backwards are those for the shoulder, arm, hand, trunk, hip, leg, and foot. The fibres for the leg lie close to those of common and special sensation in the retrolenticular portion

of the internal capsule. From the internal capsule the pyramidal fibres pass downwards into the pes pedunculi, and onwards through the ventral region of the pons Varolii to form the pyramids of the medulla oblongata. On reaching the bulb the two pyramids become more closely approximated than in any previous part of their course.

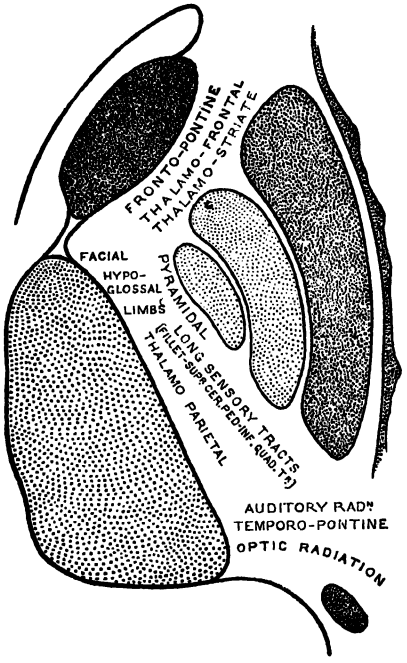


FIG. 4 (from Cunningham's 'Anatomy'). Shows the arrangement and position of the afferent and efferent fibres in the internal capsule.

During their progress through the mid and hind brain a great diminution in the size of the pyramids takes place, owing to a large number of fibres being given off to the motor cranial nerve nuclei, and to other structures, as follows:—

(*a*) To the third and sixth nuclei. It is not exactly known how these nuclei receive pyramidal fibres, whether by special bundles or by means of scattered fibres.

(*b*) To the motor trigeminal, facial, and hypoglossal nuclei, by the 'accessory fillet.'

(*c*) To the nucleus ambiguus, or motor vago-glossopharyngeal nucleus, by 'Pick's bundle.'

(*d*) A ventro-lateral pyramidal bundle has been described (Barnes), but it is doubtful whether it contains pyramidal or other descending fibres.

(*e*) Thalamo-spinal, rubro-spinal, and ponto-spinal bundles. The existence of these tracts may explain the retention of some movement when the pyramids are completely severed, the restitution of function after cerebral lesions with degeneration of the pyramidal tracts and the continuance of artificially induced epileptic convulsions after section of the pyramids.

The great majority of the pyramidal fibres cross at the

decussation of the pyramids to form the lateral, or crossed pyramidal tract of the spinal cord. A relatively small number pass downwards in the antero-internal region of the same side, as the direct pyramidal tract, and decussate, by way of the anterior commissure at succeeding levels, into the grey horns of the opposite side.

The fibres of the crossed pyramidal tract give off collaterals into the grey matter throughout the whole length of the spinal cord, and the tract may be traced as a diminishing structure as far down as the fourth sacral segment. The arborescence of the pyramidal fibres round the anterior cornual cells has not yet been demonstrated, and, according to some observers, they terminate in relation to the cells of the posterior horns.

By means of the pyramidal fibres, the motor cortex of the precentral area is brought into relation with the bulbo-spinal centres and nuclei of all segments of the limbs and body from the eyes to the perinæum.

Owing to the complete decussation of the motor fibres, a destructive lesion of one cerebral hemisphere, involving the pyramidal system, is followed by paralysis of the movements of the opposite side of the body.

2. The peripheral motor system (lower motor neurone)

The peripheral motor system consists of the bulbo-spinal centres, their issuing motor roots, and the motor fibres of the peripheral nerves.

The bulbo-spinal centres are collected into groups of ganglion cells. In the hind brain these collections of cells form the nuclei of the motor cranial nerves, and give origin to their respective motor nerves, or to the motor fibres of the mixed cranial nerves. In the spinal cord the separation of the centres is less defined, although two distinct enlargements are seen corresponding to the upper and lower limbs.

The following is a brief account of the motor nuclei of the cranial nerves and their homologues in the anterior horns.

The *third nucleus* consists of an elongated group of nerve cells lying in the grey matter of the floor of the aqueduct of Sylvius, which connects the third and fourth ventricles. The nucleus has a segmentary character, the several groups of cells

corresponding to separate ocular muscles, or groups of muscles. Quite at its posterior end is a group of cells which would seem to innervate the levator palpebræ superioris, the median groups probably supply the internal and inferior recti muscles, and the lateral group the superior rectus and inferior oblique muscles of the eye. There is also an antero-lateral group of small cells, which was at one time supposed to supply the pupillary fibres and those for the ciliary muscle. The roots of the third nerve pass through the tegment of the crus and issue mesially to the pes cruris at the upper border of the pons Varolii, and proceed into the orbit through the sphenoidal fissure.

The *fourth nucleus* is situated posterior to the third nucleus, of which it is really the distal continuation. The nerve roots decussate after they have left the nucleus in the superior medullary velum and pass forwards into the orbit to supply the superior oblique muscle.

The *sixth nucleus* lies some distance behind the above in the dorsal portion of the tegmentum pontis, under the floor of the fourth ventricle. Its nerve root passes ventrally through the tegment of the pons to issue at its lower margin. It passes forwards to enter the orbit and to supply the external rectus muscle. In the tegmentum pontis the sixth nucleus is enveloped by the issuing root of the seventh nerve.

The oculo-motor nuclei are connected with each other and with the spinal cord by fibres of the posterior longitudinal bundle. In this bundle are fibres which connect the sixth nucleus of one side with the opposite third nucleus, a connexion which subserves the function of conjugate movement of the eyeballs. The sixth nucleus is, therefore, not only the motor nucleus for the homo-lateral external rectus muscle, but is also the centre for the associated action of the external and internal recti of opposite sides.

The *motor nucleus of the fifth nerve* is formed partly by a group of large cells situated in the lateral part of the tegmentum pontis, and partly by a column of cells situated in the Sylvian grey matter—the so-called trophic or descending trigeminal root. Both these sets of cells give fibres to the motor root, which is distributed mainly to the muscles of mastication. (Fig. 5.)

The *seventh nucleus* is a segmented nucleus occupying a

ventral position in the tegmentum pontis. The nerve root passes dorsally towards the floor of the fourth ventricle, and, turning round the sixth nucleus, courses through the tegmentum and issues on the ventro-lateral aspect of the pons. It supplies the facial muscles of expression. (Fig. 5.)

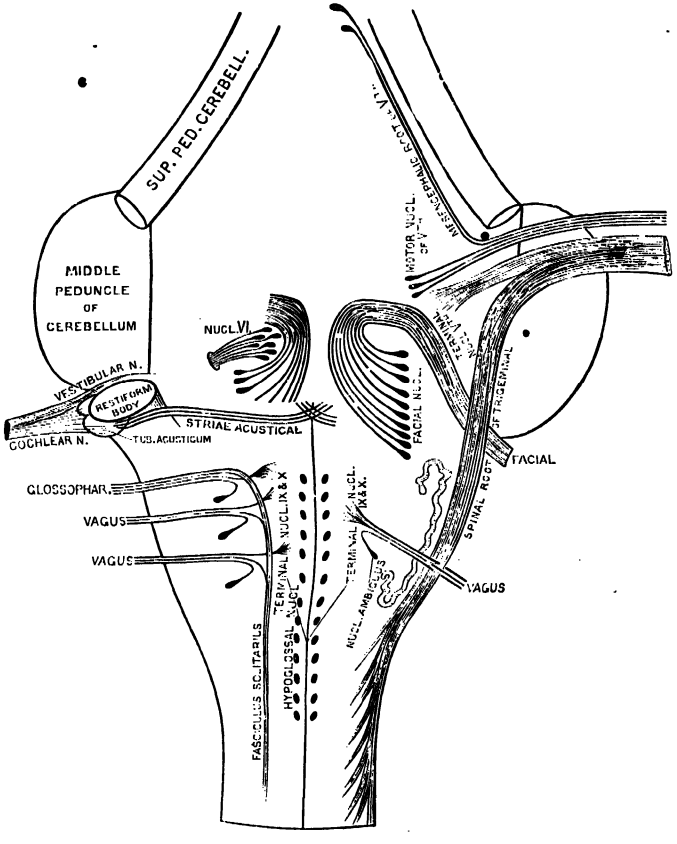


FIG. 5 (from Cunningham's 'Anatomy').—Shows the connexions of the vagal, glossopharyngeal, auditory, facial, abducent, and trigeminal nerves.

The *nucleus ambiguus* is the motor nucleus for the combined vago-glossopharyngeal nerve. It is a long nucleus lying in the reticular formation of the medulla oblongata. Its nerve roots form the motor fibres of the glossopharyngeal and vagus nerves. (Fig. 5.)

The *twelfth nucleus* is a segmented nucleus lying in the dorsal portion of the medulla, and is rich in medullated nerve fibres. Its roots pass through the reticular formation and issue on the anterior surface of the bulb external to the pyramids. (Fig. 5.)

The *nuclei of the anterior horns* presiding over the muscles of the head and neck, trunk and limbs, are situated in the anterior horns throughout the whole extent of the spinal cord.

According to the description given by Lenhossék, the most mesially placed group of cells in the anterior cornual region is commissural, not motor in function. The motor cells lie external to the mesial group. In the upper cervical and dorsal regions of the cord, the motor cells are grouped into a single nucleus: in some places slightly separated from, in others in juxtaposition to, the mesial or commissural group. In the cervical and lumbar enlargements on the other hand, the motor cells become separated into two large and well-defined nuclei, having an antero-lateral and postero-lateral position respectively in the anterior horn. In the lower lumbar and sacral regions a third group has been described occupying a central position.

The nucleus in the intermedio-lateral horn, which is present between the eighth cervical and second lumbar roots, has been found by Bruce[1] to be connected with the sympathetic system.

Of the fibres forming the *anterior roots*, some are coarse and others are fine. It is generally supposed that the fibres of larger calibre pass to the voluntary muscles, while the finer are destined for the sympathetic system by way of the rami communicantes.

The general conclusions which may be derived from numerous studies upon the functions of the motor nuclei in the anterior horns are, first, that muscles and not movements are represented in them; and secondly, that each segment of the cord presides over portions of a number of different muscles. (For further details on this subject, the reader is referred to p. 317.)

[1] Bruce, *Review of Neurology*, 1907.

THE SENSORY SYSTEM

Anatomy of the Sensory System

For purposes of description the sensory system may be divided into three sub-systems:—

1. The lowest, or system of the posterior root ganglion, includes the sensory end-organs, the sensory fibres of the peripheral nerves, and the posterior roots with their extensions into the spinal cord. It also includes the sensory cranial nerves, and their bulbar continuations.

2. The middle or spino-thalamic systems of the cord and the cranial nerves.

3. The highest, or thalamo-cortical system.

The posterior ganglionic system consists of nerve fibres, which have their trophic cells in the ganglia of the posterior spinal roots, or their cranial homologues—Gasserian, geniculate, jugular, petrosal, and vagal.

The sensory fibres pass at the inter-vertebral foramina into the posterior nerve roots of their respective segments.

The central terminations of the spinal and cranial sensory roots will be separately considered.

(1) *Spinal.* The fibres of the posterior nerve roots are primarily divided into two sets, fine and coarse fibres. The fine fibres enter the posterior horn as the zone of Lissauer. The coarse fibres take up a position along the posterior horn of grey matter (the cornu-radicular zone of Marie). This zone is little by little displaced in a mesial direction by the entrance of other nerve roots at successively higher levels. The destination of the coarse fibres is as follows: (*a*) by short fibres terminating in the posterior horn of the same side; (*b*) by medium fibres passing some distance up the posterior columns, and terminating in Clarke's group of cells; and (*c*) by long fibres which pass up the posterior columns to the posterior columnar nuclei. On their passage upwards in the posterior columns, the long fibres, entering from the lumbo-sacral and lower dorsal nerve roots, occupy the postero-median column; those from the upper dorsal and cervical roots ascend in the postero-external column.

(2) *Cranial.* The afferent fibres of the sensory cranial

nerves are arranged in a somewhat different manner, being collected into definite bundles.

(*a*) *The trigeminal nerve.* The centripetal fibres of the Gasserian ganglion on reaching the pons are collected into the spinal root of the trigeminus, which passes distally as far as the second cervical nerve. The fibres of this root terminate in the gelatinous substance of Rolando which lies upon its mesial aspect. (Fig. 5.)

(*b*) The *sensory division of the facial nerve,* or nerve of Wrisberg (pars intermedia), enters the medulla in association with the internal root of the auditory nerve. In a case observed by Ramsay Hunt [1] the fibres terminated in the fasciculus solitarius.

(*c*) The *vago-glossopharyngeal nerve.* The afferent fibres of this nerve divide into two series, one of which forms the fasciculus solitarius, a structure analogous to the spinal root of the trigeminus, and the other terminates in the posterior vago-glossopharyngeal nucleus. The fibres of the fasciculus solitarius end in the contiguous gelatinous substance. (Fig. 5.)

Sensory impressions are conveyed from the spinal cord to the brain by (*a*) the mesial fillet; (*b*) the spino-thalamic system.

The mesial fillet. From the posterior columnar nuclei, in which the posterior columns of the spinal cord terminate, an extensive decussation takes place into the opposite inter-olivary layer. The mesial fillet emerges from this layer, and, passing through the medulla and pons, terminates in the ventro-lateral region of the optic thalamus. It conveys the impressions transmitted by the posterior columns in a cerebral direction.

The spino-thalamic system. This would appear to be the path by which some of the entering posterior root fibres are indirectly continued towards the brain. Fibres, arising in cells situated in the posterior horns, cross to the opposite side by the posterior commissure and ascend in the antero-lateral column of the spinal cord, where they form a series of short systems of ascending fibres, lying mesial and ventral to Gowers's tract. These fibres ascend through the spinal cord, medulla, and pons into the ventral portions of the optic thalamus. In the medulla and pons they receive accessions

[1] Ramsay Hunt, *Journ. Nerv. and Mental Disease*, 1907.

of fibres from the sensory cranial nerves, more especially the trigeminus of the opposite side.

The distribution of the sensory fibres would appear to be as follows:—

1. Some of the fibres are continued for a considerable distance upon the same side as the entering sensory nerves, either as the posterior columns of the cord, or as the descending spinal root of the trigeminus and the vago-glossopharyngeal nerves. (Fig. 6 (1).)

2. Others are continued for a longer or shorter distance upon the same side prior to their decussation, as it is obvious, on the one hand, that the posterior columns in the cervical region cannot contain all the fibres which have passed into them from the lower segments, and, on the other hand, the spinal roots of the trigeminus and vago-glossopharyngeus are structures which diminish in size as they approach their termination. (Fig. 6 (2).)

3. Other fibres decussate at once on entering the spinal cord or the bulb. (Fig. 6 (3).)

Whatever is the immediate disposition of the entering sensory fibres, whether spinal or cranial, it is clear that they all decussate before they reach the upper portion of the hind brain.

The thalamo-cortical system. A great mass of fibres radiates in a cortical direction from the optic thalamus and passes to most regions of the cerebral cortex both of the same and of the opposite side, reaching the latter through the corpus callosum. Which portion of this thalamic projection system belongs to the afferent or sensory pathway under discussion is uncertain; but it would seem possible to differentiate two primary systems, which medullate at different periods (Flechsig): (*a*) A tract of fibres, medullating at the ninth month, passes to the post-central gyrus. This is probably the cortical termination of the mesial fillet. (*b*) A tract, medullating during the first month of extra-uterine life, passes to the falciform lobe. These fibres probably occupy the retrolenticular portion of the internal capsule in close relation to the fibres of the adjacent optic radiation. (Fig. 7.)

The *cortical centres* for common sensation will be considered more particularly under the physiology of the sensory

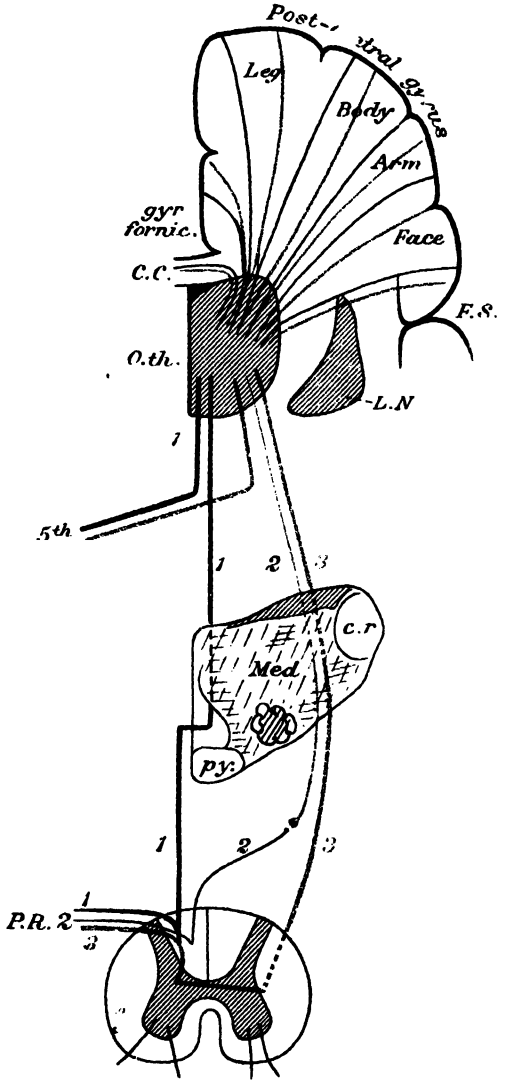


FIG. 6.—Scheme of the sensory system in the spinal cord, and brain. *P.R.*, posterior nerve roots. 1, direct fibres of posterior columns. 2, fibres which pass for a short distance in the posterior columns. 3, the decussating fibres. *Med.*, medulla oblongata. *5th*, trigeminal nerve. *O. th.*, optic thalmus. *C.C.*, corpus callosum. *L.N.*, lenticular nucleus. *F.S.*, Sylvian fissure.

system (p. 18), but it has been shown by Campbell that the post-central gyrus is structurally in harmony with certain other cortical areas, which are known to be sensory in function. The special feature of this zone is the presence of a plexus of nerve fibres of large calibre, having an oblique course and running at right angles to the radiating fasciculi.

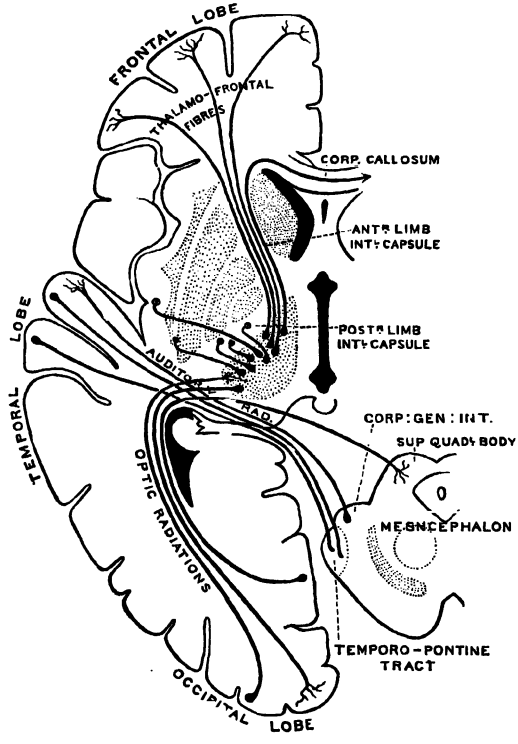


FIG. 7 (from Cunningham's 'Anatomy').—Shows the afferent or thalamo-cortical projection system of fibres.

PHYSIOLOGY OF THE SENSORY SYSTEM

Our conception of the physiology of the sensory system has been enlarged by the recent observations of Head,[1] the general results of whose work have been embodied in the following account.

[1] Head, *Brain*, 1905–1906.

The peripheral nervous system

Head has shown that in the peripheral nervous system there is a physiological grouping of the various forms of sensibility.

1. Epicritic sensibility, consisting of the appreciation of light touch, the finer grades of temperature, cutaneous localisation, and the discrimination of two points of a compass.

2. Protopathic sensibility, or the recognition of painful cutaneous stimuli and the extremes of heat and cold. This form of sensation is unaccompanied by any definite appreciation of locality. The sensibility of the viscera is probably of this type.

3. Deep sensibility, consisting of the sense of passive position and of movement, and the appreciation of pressure, which if excessive may produce pain. The fibres conducting deep sensation run with the motor nerves and are distributed to the muscles, tendons, joints and periosteum.

Section of a peripheral nerve is followed by loss of protopathic sensibility over a variable area, corresponding approximately to the distribution of the nerve on the skin. Epicritic sensibility is abolished over a somewhat larger area.

In the zone of epicritic overlap, the sensibility is characterised by the great discomfort of a painful stimulus, its widespread diffusion, and want of localisation. Deep sensibility is abolished, if the divided nerve contains both motor and sensory fibres.

In cases of nerve injury, not amounting to complete division of the nerve, all forms of sensibility may be affected, but in slight lesions the epicritic loss may be the only type of impaired sensibility.

The above facts also hold good for the limb plexuses and the posterior nerve roots, with this difference—that the nearer the lesion is to the central nervous system the smaller becomes the zone of overlap of epicritic over protopathic loss. In root lesions (as elsewhere shown, p. 104) the protopathic loss may even be more extensive than the epicritic. It has therefore been stated by Head as an axiom that the posterior nerve root is the unit of protopathic, and the peripheral nerve the unit of epicritic sensibility.

The spinal cord

In the discussion of the conduction of sensation in the spinal cord, we enter upon a subject of great complexity.

All varieties of sensation are conducted into the spinal cord through one or more of the posterior root bundles already described. Some forms of sensation are transmitted at once to the opposite side of the spinal cord from the entering posterior roots, and others are conducted upwards along the same side. It is well known that, in consequence of injury or disease of the spinal cord, one form of sensation may be abolished while another may be retained, an observation which indicates that different forms of sensation are conducted along separate tracts. For example, it is not uncommon to find that the sensibility to painful stimuli may be lost, while tactile sensibility is retained; and that if painful sensibility is abolished, the sensibility to heat and cold is also in abeyance.

The conduction of sensation may best be studied by observing the defects of sensibility following partial lesions of the spinal cord in man.

Scheme showing the Brown-Séquard 'Symptom-Complex,' based on Head's Observations

Side of lesion.	*Side opposite lesion.*
Motor paralysis.	No paralysis.
Retention of tactile, light pressure, painful and thermal sensibilities.	Tactile and light pressure sensibilities may or may not be impaired. Painful and thermal sensibilities abolished.
Painful pressure retained.	Painful pressure abolished.
Impairment or abolition of tactile discrimination, and sense of position of limbs.	Retention of sense of position and of tactile discrimination.
Retention of cutaneous localisation.	Cutaneous localisation depends upon the state of tactile sensibility.

From a study of this Table it is apparent: (1) that some forms of sensation (pain and temperature sensibility) cross

soon after their entrance and pass up the opposite side of the cord; (2) that other forms of sensation—the sense of passive position and of movement and tactile discrimination (Head)—do not cross, but pass up on the same side as the entering posterior nerve roots; (3) that tactile sensibility may or may not be abolished on the side opposite the lesion; and (4) that the sense of cutaneous localisation may be dissociated from and remain intact, when the sense of passive position is absent (Head).

In the spinal cord, the varieties of sensory loss are not recognised in the same combinations as those found in lesion of a peripheral mixed nerve. On their entrance into the cord, a rearrangement of the sensory fibres of the posterior roots takes place, so that some forms of sensation cross and pass up the opposite side, while others remain upon the same side. The fibres of the posterior nerve roots, which pass directly up the same side of the cord, are the long extrinsic fibres of the posterior columns. These conduct the impressions subserving the senses of passive position, of movement, and of tactile discrimination.

Of the other posterior root fibres, some were seen to cross indirectly into the ventro-lateral part of the opposite lateral column, and to pass upwards as the spino-thalamic system, which tract is believed by some authorities to conduct the impressions of pain and temperature sensibility. In opposition to this view is the fact that destruction of the gelatinous substance of the posterior horn at the upper end of the cord is followed by, among other phenomena, abolition of contra-lateral painful sensibility—a fact which would point to the posterior horn of grey matter, or the adjacent tissues as conductors of painful sensibility.

The tracts which conduct tactile and pressure sensibilities are mainly crossed, but they do not decussate so soon after their entrance into the cord as those for pain and temperature; hence these impressions are probably conducted for a short distance in the homo-lateral posterior columns, and, higher up, in the ventro-lateral tracts of the opposite side.

We have endeavoured to co-ordinate and arrange these facts in the subjoined Table, which shows: (1) the three main forms of sensibility found in the peripheral nerves; (2) the varieties and kinds of sensation forming the three primary

groups; (3) their position in the spinal cord, whether homo- or contra-lateral to the entering posterior nerve roots; and (4) the tracts in the cord by which the several forms of sensation are probably conducted.

Sensory grouping in the nerves.	Variety of sensation.	Relation in cord to entering root.	Position in columns of cord.
	1. Light touch	Partly crossed Partly uncrossed	Partly homo-lateral posterior column, partly antero-lateral column of opposite side
Epicritic sensibility	2. Discrimination	Uncrossed	Posterior column
	3. Localisation	As for light touch	As for light touch
	4. Moderate shades of temperature	Crossed	(*a*) Spino thalamic system of antero-lateral columns
Protopathic sensibility	5. Extremes of temperature	Crossed	(*b*) Posterior grey horns
	6. Pain	Crossed	
	7. Sense of passive position	Uncrossed	Posterior column
Deep sensibility	8. Contact and pressure	As for light touch	As for light touch
	9. Painful pressure	Crossed	As for pain

The mid- and hindbrain

As already shown, two main routes exist for sensory conduction in these localities:—

1. The mesial fillet, which conducts the sensations conveyed by the contra-lateral posterior columns of the cord. Degeneration of this structure is not followed by loss of tactile and painful sensibility.

2. The spino-thalamic system, which conveys the impressions of touch, pain, and temperature sensibilities from the corresponding tracts in the antero-lateral columns. There are cases on record of hemorrhage and softening of the tegmentum, and of experimental division of this structure in which the fillet was intact, but which were accompanied by anæsthesia and analgesia of the opposite side.

All forms of sensation are gathered together in the

ventro-lateral parts of the optic thalamus, as destruction of this ganglion is followed by complete loss of all forms of cutaneous sensibility upon the opposite side of the body, as well as by loss of the sense of position and of movement of the limbs.

Cortical sensory centres

Recent investigation confirms the early view that motion and sensation have separate centres in the cerebral cortex. There is accumulating clinical evidence that the post-central gyrus is the cortical area for the senses of passive position and of movement, for tactile localisation and discrimination, as well as for cutaneous sensibility.

There is also evidence that the post-central area, like the precentral, is further subdivided into zones corresponding to the different portions of the body. The sensory cortical centres are situated immediately posterior to the corresponding motor centres.

Facts bearing upon the cortical centres for pain and temperature sensibility are less satisfactory. Experimental evidence favours the gyrus fornicatus, but clinical observation supports the view that the sensibilities of pain and temperature have cortical centres in, or immediately behind, the post-central gyrus.

Campbell's observations have shown that the post-central area proper merges posteriorly into an 'intermediate post-central,' which extends into the parietal lobe.

These areas contain higher psychical sensory centres of complex character, in which impressions derived from the various forms of cutaneous and deep sensibility are correlated, and translated into definite conceptions.

THE CEREBELLAR SYSTEM

Anatomy. The cerebellum consists of a large mass of cortical grey matter and several sub-cortical ganglionic structures: the corpus dentatum, the roof nuclei, and probably also the nucleus of Deiters.

The nucleus of Deiters occupies a prominent position upon the dorso-lateral aspect of the pons Varolii. It is characterised by the presence of large multipolar ganglion

cells, similar to those seen in the anterior horns of the spinal cord. It has important relations with the vestibular nerve and the spinal cord.

The cerebellum is connected with the brain stem by afferent and efferent tracts.

The *afferent* cerebellar tracts are the following:—

1. The dorsal or direct spino-cerebellar tract of Flechsig. The fibres of this tract arise as the axis-cylinder processes of the cells of Clarke's group, which is situated in the intermediate grey matter of the spinal cord, mainly in the lower dorsal and upper lumbar regions. Clarke's group is in intimate relation with certain of the posterior root fibres, as after section of the posterior roots, or in association with the posterior columnar degeneration of tabes dorsalis, the medullated fibres of Clarke's group atrophy and disappear. This group of cells therefore appears to be an intermediate station in the afferent spino-cerebellar system. Issuing from Clarke's cell-group, the fibres of the dorsal spino-cerebellar tract take up a position in the postero-lateral periphery of the spinal cord, and ascend to the restiform body. In this structure they are transmitted to the cerebellum and are projected on to the dorsal surface of the middle lobe.

2. The ventral spino-cerebellar tract, forming a portion of the ascending antero-lateral tract of Gowers. The fibres of this tract arise as axis-cylinder processes of cells situated within the posterior horn of the same and the opposite side. These cells also stand in relation to certain of the entering posterior root fibres. In the antero-lateral periphery of the spinal cord the fibres of the ventral spino-cerebellar tract lie external to the descending antero-lateral (vestibular) fibres, and also to the ascending spino-thalamic fibres. The tract passes upwards into the bulb and pons, as high as the root fibres of the fifth cranial nerve. Here it bends backwards into the superior cerebellar peduncle, along which it is transmitted to the cerebellum, and terminates upon the ventral surface of the middle lobe.

3. The vestibular nerve, as described on p. 61.

4. Some fibres would appear to pass from the nuclei of the posterior columns upon the same side, but the existence of this connexion has been denied by recent observers.

5. Fibres coming from the cerebral cortex are supposed

to pass to the pontine nuclei, and to be thence transmitted by way of the middle peduncle to the cerebellum.

6. The olivo-cerebellar connexion.

The *efferent* cerebellar connexions are the following:—

1. The brachium conjunctivum emerges from the corpus dentatum cerebelli and passes by way of the superior cerebellar peduncle to the tegmentum cruris, where it decussates with its fellow from the opposite side. It then passes forwards to terminate partly in the red nucleus and partly in the ventral portions of the optic thalamus. There is no anatomical evidence of a direct passage of fibres from the brachium to the cerebral cortex, although it is surmised that an indirect transference of cerebellar fibres may take place by way of the internal capsule and centrum ovale.

2. The efferent fibres of the middle cerebellar peduncle pass from the corpus dentatum of one cerebellar lobe to the nucleus pontis and tegment of the opposite side.

3. An important indirect cerebellar efferent tract is that which passes from the large-celled nucleus of Deiters through the bulb and occupies the antero-lateral periphery of the spinal cord throughout its whole extent. This tract, known as the *vestibulo-spinal*, or descending antero-lateral tract, degenerates only after destructive lesion of the large cells of Deiters's nucleus, and would appear to be the agent through which the cerebellum exerts a controlling influence upon the trunk and limbs.

Certain *intra-cerebellar* tracts are also present—viz., those which connect the cerebellar cortex with the nucleus dentatus, the roof nuclei, and the nucleus of Deiters.

It will be seen from the above description that the connexions of the cerebellum are partly crossed and partly uncrossed. The crossed connexions are the superior and middle cerebellar peduncles, the ventral spino-cerebellar tract, and the olivo-cerebellar bundle. The uncrossed connexions are the dorsal spino-cerebellar tract, and the vestibulo-spinal tract from the nucleus of Deiters.

Physiology. Considerable divergence of opinion still exists as to the functions of the cerebellum. The effects of its experimental ablation (in whole or in part) have been studied by a number of observers, and the general effects of this operation may be briefly stated.

After removal of the whole cerebellum profound disturbances of equilibration, instability, and inco-ordination of movement of the limbs, trunk, and head are observed. These immediate consequences of the lesion are followed by long-continued unsteadiness of the trunk and limbs on muscular effort, tremor, and nystagmus. These symptoms may persist for an indefinite period, but tend to diminish in course of time.

After removal of one-half of the cerebellum, the persistent symptoms are similar in all respects to those observed after total extirpation, but are confined to the side of the lesion.

The symptoms observed after division of the cerebellar peduncles are similar to those following a unilateral extirpation, but there is a greater tendency to roll round the longitudinal axis towards the side of the lesion. There is also a more marked incurvation of the vertebral axis towards the side of the lesion, and a greater tendency for the homo-lateral limbs to assume an attitude of adduction and flexion, and the contra-lateral limbs one of abduction and extension. In these respects the symptoms are comparable to those following division of the vestibular nerve (p. 62).

Compensation, or restitution of function, occurs to a large extent after severe destructive lesions, but certain permanent signs remain in the shape of cerebellar ataxy, a reeling gait, and an unsteady and ungraduated action of the limbs on voluntary movements.

These permanent symptoms are, in all respects, comparable to what is observed with the slower and less complete lesions of the cerebellum in man. In these cases are found a reeling, unsteady, or drunken gait, and an inco-ordinated or ungraduated action, more especially of the movements of the arm on volitional effort, although definite intention tremors are not commonly seen in lesions limited to the cerebellum.

Some degree of enfeebled muscular tone (hypotonia), affecting more especially the muscles of the neck and back, but also of the limbs has been observed.

True motor paralysis does not occur in consequence of lesion of the cerebellum as such, although some degree of muscular asthenia, or defect in the force of the muscular contraction, has been described by some observers.

Through its superior peduncle the cerebellum may exert

an indirect influence (tonic or trophic) upon the Rolandic area of the opposite side. The connexions of the superior peduncle, however, are more directly with the thalamic, sub-thalamic, and rubral regions of the midbrain. Sherrington [1] has shown that division of both superior peduncles and the tegmentum pontis induces a condition of 'decerebrate rigidity,' not unlike that observed after removal of the entire cerebellum, and Holmes [2] has recorded some cases of tremor having the features of that observed after division of the superior cerebellar peduncle in monkeys, in which the lesion was a tumour involving the sub-thalamic and rubral regions. It is, therefore, not unlikely that the influence of the cerebellum is mainly upon the basal structures just described, and through them by way of the rubro-spinal and vestibulo-spinal tracts upon the musculature of the trunk and limbs.

It has been recently shown (Horsley and Clarke) [3] that the cortex of the cerebellum is electrically inexcitable. The projection of the cerebellar afferent tracts on to the cortex cerebelli favours the view that the whole cortex, and more especially that of the middle lobe, is an afferent receptive centre. According to these observers the intrinsic and para-cerebellar nuclei are the cerebellar efferent mechanisms. Their action when stimulated is tonic; excitation of the intrinsic nuclei provokes homo-lateral movements of the eyes and limbs; and of the para-cerebellar nuclei, extension of the lower limbs and hyper-extension of the neck and trunk.

According to Sherrington, the cerebellum is the 'head ganglion of the proprio-ceptive system,' of which the labyrinth is the most important peripheral organ. Impressions from this organ reach the cerebellum by the vestibular nerve, while those from the joints, tendons, skin, and muscles are conducted by the dorsal and ventral spino-cerebellar tracts.

[1] Sherrington, *Brain*, 1906.
[2] Holmes, *Brain*, 1904.
[3] Horsley and Clarke, *Brain*, 1905

Part II

THE EXAMINATION OF THE NERVOUS SYSTEM IN A CASE OF NERVOUS DISEASE

For the proper investigation of a case of nervous disease a systematic and thorough method of examination should be adopted. The use of a routine plan is desirable, and will save the physician from mistakes in the diagnosis of a class of disease in which the symptoms and signs present many variations. The discrimination of important from unimportant signs is acquired by knowledge, and the value of negative in contrast to positive facts can only be appreciated by experience. Such experience enables the physician to seize upon the essential points of each case, and to extend his examination without neglecting the subordinate features. It should always be kept in mind that the least obvious may be the most important sign.

GENERAL METHOD AND SCHEME OF EXAMINATION

Before commencing the examination of a case of nervous disease, inquiry should be directed to the following:—

1. **Complaint.**
2. **Duration.**
3. **Family history.** The family history of a patient suffering from nervous disorder is important. In the case of functional disorders inquiry should be made particularly into family epilepsy, insanity, alcoholism, and the minor psychoses, such as neurasthenia, psychasthenia, hysteria, morbid fears, obsessions, and tics. In organic nervous diseases inquiry should be directed towards such diathetic states as syphilis, gout, rheumatoid arthritis, rheumatism, tuberculosis, and vascular degeneration.

4. **Personal history.** The history of the patient's health prior to the onset of the illness may throw light upon the malady. This should be investigated in a chronological order commencing with birth, the age at the onset of teething, and whether it was accompanied by convulsions, the age when the patient walked and talked, the diseases of infancy, the incidence of maladies such as bed-wetting, night terrors, somnambulism, epilepsy, chorea, middle ear disease, and tuberculosis. Of illnesses during later years, the more important are influenza, the exanthemata, diphtheria, enteric fever, and acute rheumatism. Venereal infection, more especially syphilis, should be a subject of careful investigation; in women, information on this point can only be obtained by collateral evidence, such as relates to miscarriages. The habits of the patient regarding alcohol, tobacco, exercise, and diet should also be the subject of investigation.

The occupation should be considered with special reference to toxic trades, such as working in lead, phosphorus, mercury, and arsenic; to those which favour the development of the neuroses—such as that of writer, pianist, tailor, or seamstress; and to professions requiring sedentary habits and mental strain and stress. The personal habits and circumstances of the patient also require investigation.

5. **The illness.** The time, the occasion, and the mode of onset of the first symptoms are especially important. The mode of onset is valuable as an indication of the nature of the lesion, and may be the point upon which the diagnosis turns. Inquiry may reveal the existence of previous transitory symptoms, which were regarded as of little or no significance. The course of the malady may be progressive, regressive, or intermittent; or the symptoms may be stationary. The presence or absence of headache, vertigo, vomiting, fits or faints should be noted.

Assigned causes given by the patient or his friends require scrutiny at the hands of the physician, as they have often to be rejected. The physical and mental condition should now be examined.

(1) *The physical condition.* The general physical development, the state of nutrition and complexion of the patient

should be observed, and notice taken of any morbid appearances—such as syphilitic teeth, scars and cicatrices, keratitis, and 'stigmata of degeneration.' These are seen more especially in high, narrow, or deformed palates, in abnormal size or shape of the ears, in cranio-facial asymmetry, in displaced, malformed or badly enamelled teeth, in disproportionate size of the limbs, hare-lip, in anomalies of the fingers, skin, hair, and nails, strabismus and marked errors of refraction—such as high myopia and hypermetropia.

An examination of the skull as to its size, shape, and configuration should never be omitted. Notice should also be taken of scars, nodes, or any other abnormalities which may be present.

(2) *The mental and emotional condition.* The memory both for recent and past events, the power of concentration and attention, and the state of intelligence will be revealed during the examination into the history of the illness by the replies which are given to questions. The physician will also observe such symptoms as depression, exaltation, irritability, excitement, loss of self-control, the presence of delusions or hallucinations, stupor, delirium, and coma.

In many cases, evidence of the slighter degrees of mental change may only be obtained by information derived from the friends or relatives.

Examination of the Special Senses and the Cranial Nerves

Smell

In all cases presenting alterations of the sense of smell, a local or rhinoscopic examination should be made. If the nose is free from local disease, the sense of smell may be tested by the inhalation of odorous substances—such as peppermint, asafœtida, oil of cloves, camphor or musk; each nostril being examined separately. The patient is tested to ascertain whether he smells. If the sense of smell and the ability to distinguish odours are present, the nerves are intact. Ammonia or other substances likely to irritate the branches of the fifth nerve within the nose should not be used.

Taste

The most accurate method of testing the sense of taste lies in stimulating the sensory taste-fibres by a weak galvanic current, applied by twisting two insulated copper wires together, the ends of which are exposed and separated one or two mm. apart.

For ordinary purposes the anterior two-thirds may be tested in the following manner. The patient is told to protrude the tongue, which is then gently cleaned. Substances in the form of white powders—such as quinine, salt or sugar, are then rubbed on to its surface, each side being separately tested, and the patient told to indicate by raising his finger, as soon as he appreciates a sensation of taste, and then to write what he tastes, the tongue not being retracted into the mouth.

To test the posterior third it is necessary to drop strong solutions of these substances on to the back of the tongue.

Vision

The following should be tested and examined :—

(*a*) The acuteness of vision ;

(*b*) The fields of vision ;

(*c*) The optic discs.

As impairment of visual acuteness and of the fields of vision may be due to errors of refraction—myopia, hypermetropia, and astigmatism, or to local diseases of the media or retina—it is important for the examiner to exclude all such factors before assigning any visual defect to nervous causes.

(*a*) The *visual acuteness* for distant objects is tested by means of Snellen's test-types, which are constructed so that a person placed six metres away—at which distance the rays of light are practically parallel—may have his vision expressed in fractions. The numerator records the distance of the patient from the type, the denominator the distance at which each letter should be read by the normal eye. The largest letters should be read under normal conditions at sixty metres ; therefore, if a patient can only read the largest

letters at six metres, the formula is V = $\frac{6}{60}$. If, on the other hand, the patient can read the small type at six metres, V = $\frac{6}{6}$. The test-types should be placed in a good light, and each eye tested separately. If the patient is unable to read any of the letters at six metres, he should be tested to see at what distance he can correctly count fingers, and the result recorded thus : V = fingers at (so many) feet. If he is unable to see fingers or hand movements, his perception of light is tested either by alternate shading and exposure of the eye to light, or, better, by throwing a ray from an ophthalmoscopic mirror upon the eye in a dark room.

For testing the acuteness of near vision, Jaeger's test-types are used. The accommodative power of a person reading the smallest type at the normal distance of from six to eight inches being recorded by the formula: Jaeger 1 at 6–8 inches.

Where impairment of acuteness is due to hypermetropia, myopia, or astigmatism, vision is recorded in the following formula: V = $\frac{6}{24}$, (with stated correction) V = $\frac{6}{9}$ or $\frac{6}{6}$ (as the case may be).

(*b*) *The fields of vision.* The only accurate and scientific method of testing the visual fields is by means of the perimeter, and recording upon charts the result of the investigation. This method, when properly carried out, will reveal not only the existence of concentric limitations of the fields and hemianopsia, but also localised areas of defective vision (scotomata). It is advisable to test not only the field for white, but also for colours. Normally, the field for white is the most extensive, and within this area are the fields for blue, red, and green respectively.

For practical purposes, a general idea of the visual fields may be obtained by the following test. The patient is placed with his back to the light, each eye being examined separately. The examiner sits about two feet from the patient and directs him to look steadily into whichever of the observer's eyes is opposite the one to be examined. Thus, when the left eye is being examined he will fix the examiner's right eye. The examiner then brings his finger towards the centre from various parts of the periphery, and, by asking the patient to state *as soon as he sees the finger*, he obtains an approximate idea of the size of the visual fields.

If the state of the patient renders such an examination impossible, the fields may be tested by quickly bringing the hand in from various parts of the periphery towards the eye, and a note made as to whether blinking occurs when the eye is threatened. If hemianopsia is present, no response is obtained when the finger is brought in from the blind field.

The various defects of the visual field are:—

1. Concentric contraction, or diminution of the peripheral field.

2. Hemianopsia, or loss of vision in one-half of the visual field. This may be horizontal or vertical. The horizontal forms are (*a*) homonymous hemianopsia, or blindness of the corresponding lateral halves of the visual fields; (*b*) heteronymous hemianopsia, or blindness of the opposite lateral fields (binasal or bitemporal).

The vertical forms are superior, or blindness of the upper half, and inferior, or blindness of the lower half of the fields.

3. Quadrantic hemianopsia, or blindness of the corresponding sectors of the visual fields.

4. Scotoma, central when the macular region is blind, annular when round the fixation point, paracentral when to one side, and eccentric when in the periphery.

(*c*) *The state of the optic discs.* This is investigated by the ophthalmoscope. The chief points to be observed are the colour, the presence or absence of swelling, the state of the margin, and the condition of the central cup. In primary atrophy, the disc is pale, or a parchment white or grey white appearance, the edges are well defined, and the lamina cribrosa is clearly seen at the bottom of the central cup. Normally, the outer half of the disc is paler than the inner, and the diagnosis of atrophy should be based not upon pallor alone, but in conjunction with visual defect.

In secondary optic atrophy, the disc is pale, the cup is filled up, the margins blurred, and the arteries small and enveloped by connective tissue.

In optic neuritis the disc is swollen and hyperæmic, the margins blurred, the veins engorged, and the arteries and veins may be buried in exudation. New vessels may be seen on the surface of the exudation and hemorrhages may be scattered about.

In all cases the state of the fundus oculi with reference to the retinal vessels, the yellow spot, and the choroid should be examined.

Hearing

In all cases of deafness an otoscopic examination should be made, for details of which the reader is referred to special works upon the diseases of the ear.

In neurological practice it is important to test the hearing in all cases presenting symptoms of deafness, tinnitus, or vertigo, more especially with a view to determine whether deafness arises from disease of the sound-conducting or of the sound-perceiving mechanism of the ear.

Auditory acuteness is tested by several methods; reliance should not be placed on any single test, but on a combination of several, of which the following are the most important:—

1. *The voice.* Although the raised, the conversational, and the whispered voice may all be used, the last is the most important. The room should be quiet and the patient placed as far away from the wall as possible. The ear which is not being examined should be closed. The whispered voice ought to be heard in a quiet room at from seven to ten yards, but the test should be carried out at varying distances.

Acuteness of hearing may be measured and expressed by a formula, in which the numerator denotes the distance at which the voice is heard by the patient, and the denominator the distance at which it would be heard by a normal ear.

2. *The watch.* This is not a satisfactory mode of testing hearing, as it only gives the auditory acuteness for feeble sounds. The distance at which a particular watch is normally heard should be known. When used, it is preferably brought in from a distance, the patient being asked to indicate when he hears it.

3. *The tuning-fork.* Forks of low and high pitch should be used. The note produced by a vibrating tuning-fork is heard both through the air and the bones of the skull. In normal states of the ear, air conduction is better than bone conduction.

In disorders of the sound-conducting apparatus—'obstructive deafness'—the tuning-fork is heard better through the bone than through the air (BC > AC). Conversely, in affections of the sound-perceiving apparatus—'nerve deafness'—the normal reaction, in which the fork is heard through the air better than through the bone, is present (AC > BC). These reactions may be investigated by Weber's and Rinné's tests.

Weber's test. If in a unilateral ear affection the stem of a vibrating tuning-fork be placed mesially upon the skull, and the note heard in the deaf ear, the sound-conducting apparatus is probably at fault ('middle ear deafness'). If referred to the normal ear, the lesion is in the sound-perceiving apparatus ('internal ear deafness'). If in a bilateral ear affection the tuning-fork, placed mesially, is referred to the less deaf ear, it is evidence in favour of an internal ear lesion in the more deaf ear.

Rinné's test. Normally the vibrating tuning-fork is heard when held in front of the external auditory meatus, after it has ceased to be heard upon the mastoid process. This is the positive Rinné response. Rinné + in a deaf ear is evidence of an affection of the sound-perceiving apparatus ('internal ear deafness'). If in a deaf ear the fork is no longer heard by air conduction, after it has ceased to be heard *per osseum* (Rinné −), it is evidence of an affection of the sound-conducting apparatus ('middle ear deafness').

Schwabach's test. Here the observer compares his own bone conduction, which must be normal, with that of the patient. When the vibrating tuning-fork placed on the mastoid process of the patient has ceased to be heard by him, but is still heard when placed upon the skull of the observer, an affection of the sound-perceiving apparatus is suspected—bone conduction is shortened. On the other hand, if the patient hears the fork after the observer has ceased to do so, an affection of the sound-conducting apparatus is suggested—bone conduction is lengthened.

In differentiating between affections of the sound-conducting and sound-perceiving apparatus, it is of value to make use of tuning-forks of high and low pitch. In lesions of the former, the low tones are lost first; in lesions of the latter, the perception of high tones is lost.

The preceding observations and their clinical significance have been summarised in the following Table :—

TABLE (MODIFIED FROM CHAVASSE AND JOUBERT) SHOWING THE CHIEF PHENOMENA OBSERVED ON TESTING THE SENSE OF HEARING

Tests.	Affections of middle ear.	Affections of labyrinth.	Combined conduction and perception affections.	In old people.
Whispered voice	Heard badly	Heard badly	Variable	Variable
Watch by air	Fair perception	Heard badly	Variable	Fair perception
Watch by bone	Good perception	Not heard	Variable	Not heard
Tuning-fork / Air	Diminished	High tones lost	Duration diminished	Well heard
Tuning-fork \ Bone	Normal	Duration diminished	Diminished or lost	Not heard
Weber's test	BC > AC	AC > BC	Variable	AC > BC
Rinné's test	Negative	Positive	Variable	Positive

The Vestibular Apparatus

The following tests may be applied :—

1. *Rombergism.* The patient is made to stand with the eyes closed, first on both feet placed together and then on each foot separately, and his ability or inability to stand is noted. In a unilateral ear lesion, he may tend to sway or fall towards the affected side, but his swaying movements may be of an indefinite character.

2. *Gait in walking.* The patient is made to 'walk the line' with the eyes open and closed, and his tendency to sway or deviate is observed. He may deviate towards the affected side. There may be a tendency to broaden the base of support or even to assume a degree of ataxic gait.

3. *Execution of movements requiring co-ordinate control*—e.g.

hopping forwards and backwards with the eyes open and closed. There is an inability to carry out such movements without swaying or loss of co-ordination.

4. *Rotation tests.* The patient is seated upon an easily rotating chair, which is made to revolve at the rate of ten complete revolutions in twenty seconds. Rotation is stopped abruptly and the patient is directed to turn the eyes in extreme deviation—first towards the side from which rotation has been made, and then in the direction of the rotation. The vestibular organs in both ears are simultaneously tested, but the nystagmus produced after stopping rotation, tests more definitely the apparatus on the side from which the rotation has been made. After rotation, there is horizontal nystagmus towards the side from which rotation has been made when the eyes are deviated to that side. There is an absence of nystagmus when the eyes are deviated in the direction of rotation. The duration of the nystagmus, when the vestibular organs are normal, is twenty-five seconds or more.

If the right labyrinth be injured or destroyed, and the patient be rotated from left to right, the nystagmus produced after stopping rotation being mainly due to the left labyrinth will continue for twenty-five seconds, while that produced after rotating from right to left may only continue for ten or fifteen seconds or less.

5. *Caloric tests.* When the normal ear is syringed with water below the body temperature, 65°–80° F., the head being held erect, there is horizontal and rotatory nystagmus on fixing the eyes to the opposite side. If water be used above the body temperature, 110°–118° F., horizontal and rotatory nystagmus occurs on fixing the eyes to the same side. Here each vestibular apparatus is tested separately. If it is destroyed, or the vestibular nerve paralysed, there is as a rule no nystagmus on syringing.

6. *Galvanic tests.* By holding one electrode in the hand and placing the other upon the mastoid process, currents of 10-25 milliampères may be used. When the kathode is applied to the normal ear, rotatory nystagmus is produced on fixing the eyes to the same side. When the anode is applied, there is rotatory nystagmus on fixing the eyes to the opposite side.

III., IV., VI.—Third, fourth, and sixth nerves

In testing ocular movements the eyes should be examined separately and together. The patient is told to fix his eyes upon the examiner's finger, which is held at about three feet away and moved in upward, downward, and lateral directions. Convergence is tested by approximating the finger towards the eyes. Impairment of the ocular movements and any abnormal movement—such as nystagmus—will thus be discerned; the character and direction of such movements should also be observed.

Paralysis of an individual ocular muscle may show itself by strabismus—when the eye is at rest—and by deficient movement in the direction of its normal action. In minor degrees of paralysis these signs may not be evident to the observer, although the patient may complain of double vision in a specified direction. If this is the case, it is necessary to apply the *candle test* in order to ascertain the relation and position of the false image. In this test, which is carried out in a dark room, one of the eyes is covered by a coloured glass, and the head is fixed to prevent movement. A candle held about two or three yards away from the patient is moved in the plane of action of each muscle, and the patient is told to indicate the position of the coloured and white flames, the relative position of the two images, their relation to and separation from each other. *The false image is seen by the paralysed eye and is displaced in the direction of traction of the paralysed muscle.*

The position of the images, their relation to each other, and the symptoms of paralysis of individual muscles are described on pp. 73 to 75.

The pupils. Normally, the pupil is round or slightly oval, with a regular outline, and placed centrally in the iris. The two pupils are of equal size, and measure from two to four mm. in diameter; they are larger in myopic and smaller in hypermetropic persons.

Irregularity of outline may result from local conditions—such as synechiæ or from a defect of innervation.

Eccentricity of the pupil may be congenital, or may result from central nervous lesions ensuing in later life.

When examining the size of the pupils, the amount of

light falling on each eye should be the same. Inequality of the pupils may be due to refractive errors, to local inflammatory changes, to paralysis or stimulation of the cervical sympathetic nerve on one side, to paralysis of the third cranial nerve, or to unilateral optic nerve atrophy.

Myosis, or small pupil, may be unilateral or bilateral. In myosis, from paralysis of the sympathetic, slight ptosis is present, the reflexes to light and accommodation are preserved, but lost to painful stimulation; cocaine produces no dilatation, but atropine causes a permanent mydriasis.

Mydriasis, or dilated pupil, may also be uni- or bilateral. If due to paralysis of the third nerve, all the pupillary reflexes are lost; if on the other hand it be due to stimulation of the cervical sympathetic nerve, the reflexes are preserved and atropine causes an increase of the dilatation.

Pupillary reactions. (*a*) Reaction to convergence and accommodation is tested by making the patient look from a distant to a near object when a pupillary contraction occurs.

(*b*) Reaction to light. In testing the light-reaction the patient should be placed so as to face the window, each eye being tested separately, the one not under observation being covered by the examiner's hand. Both the direct and the consensual reactions are tested. A pupil inactive to light may respond on convergence or with accommodation (Argyll-Robertson phenomenon). The pupil dilates normally on stimulation of the skin of the neck (cilio-spinal reflex).

In cases of hemianopsia the reaction of the pupil should be examined by throwing light respectively upon the seeing and the blind halves of the visual field. To do this satisfactorily a fine pencil of light is required. If the pupil fails to respond only when light is thrown upon the blind half of the retina, it signifies that the lesion is situated in the chiasma or optic tract. This is the hemiopic pupillary reaction.

V.—Fifth or trigeminal nerve

Motor. The voluntary action of the temporal and masseter muscles is tested by asking the patient to clench the teeth, and the action of the external pterygoid muscles by asking the patient to open his mouth against resistance. In cases of unilateral palsy the jaw deviates to the paralysed side and

the patient is unable to move the jaw to the non-paralysed side. An inability to depress the lower jaw signifies paralysis of the internal pterygoid muscles. Degenerative changes in the muscles are ascertained by the presence of atrophy and altered reactions to faradic and galvanic excitation.

Sensory. Sensation is tested as detailed later under the examination of the sensory phenomena elsewhere in the body (p. 39). It is also necessary to test the sensibility of the cornea and the mucous membranes of the mouth, nose, palate, and tongue.

VII.—Seventh or facial nerve

The face should be examined first in repose, and any asymmetry noted. Slight asymmetry is not important. In paralysis the face may be drawn to one side, either by over-action of non-paralysed muscles, or by contracture of paralysed muscles. The voluntary movements should next be tested by such actions as elevation of the eyebrows, frowning, closure of the eyelids, showing the teeth, whistling, and pouting. If paralysis is present, these movements will be impaired. The emotional movements—such as occur in smiling, laughing, and crying—should be observed. Differences between the degree of affection of the upper and lower parts of the face are also to be noted.

The electrical reactions of the muscles, both to faradic and galvanic stimulation, should be tested in all cases of facial paralysis (p. 51).

IX., X.—Ninth and tenth nerves. Vago-glossopharyngeal nerve

The points to be investigated in connexion with this nerve are the movements of the pharyngeal muscles, the soft palate, and the vocal cords; and in addition the action of the pulmonary, cardiac, and gastric centres.

1. *Pharynx and soft palate.* The signs of paralysis are—(1) inability to swallow, (2) regurgitation of fluids through the nose, and (3) an absence of movement of elevation of the soft palate when the patient is asked to open his mouth and say, 'Ah.'

In unilateral lesions little impairment of swallowing occurs,

but the palate will be observed to be drawn upwards to the non-paralysed side on phonation.

The sensory functions are tested (*a*) as to taste on the posterior third of the tongue, and (*b*) as to common sensation over the soft palate, pharynx, and fauces. The palato-pharyngeal reflex is tested by tickling the fauces.

2. *The larynx.* Any changes in the voice should be noted, also the ability to cough and phonate, and the presence of stridor. In all cases presenting such symptoms, a laryngoscopic examination ought to be made. The symptoms of the several forms of laryngeal paralysis and the corresponding laryngoscopic appearances are detailed on p. 92.

3. Alterations in the *pulmonary, cardiac,* and *gastric functions*—such as slow or altered respiration, rapid or slow heart's action, vomiting and hiccough—can only be referred to nerve disturbances in the absence of organic disease or other obvious cause.

XII.—Twelfth nerve. Hypoglossal nerve

This is tested by asking the patient to protrude the tongue and to put it into his cheeks. Loss of these movements may be unaccompanied by wasting of the tongue, when due to a lesion above the nucleus. The orbicularis oris may be affected in association with nuclear palsy of the tongue. If the paralysis is unilateral, the tongue on protrusion deviates to the paralysed side. In nerve lesions proper the tongue is paralysed on one or both sides, and the paralysis is accompanied by wasting and furrowing of its surface. The commencement of the act of swallowing is impaired, and articulation is defective.

Examination of the Motor System

The examination of the motor system of the trunk and limbs is carried out to ascertain :—

1. *The general condition of the muscles.* Observation should be made upon the physique of the patient, his muscular development in relation to his occupation and pursuits, the presence of deformities, and the attitude and position of the limbs at rest and on movement.

2. *The presence or absence of tremor, spasm, and of convulsive or other abnormal movements.* The character of

such movements should be observed, their range, rhythm, and constancy; whether they are irregular, purposive or spasmodic; the muscles affected and the influence of rest, action, or emotion upon them, and whether they are under the control of the will.

3. *The power of the various groups of muscles.* This includes the range as well as the strength of muscular action. The patient should be asked to perform the ordinary movements of the limbs, trunk, and head. Should any weakness or inability to perform them be noted, the examiner should feel the muscles during their attempt at movement, with a view to ascertain whether they are really contracting. The state of the antagonistic muscles should also be noted, as in cases of functional paralysis the contraction of the antagonistic muscles can be felt before that of the prime movers.

In testing the defects of the muscular power, it is important to remember that, in cerebral paralysis, the loss of power is due to paralysis of voluntary movement, while in spinal and peripheral paralysis, the loss of power depends upon paralysis of individual muscles or groups of muscles.

Thus in hemiplegia (cerebral paralysis), the examination consists, in the first place, in asking the patient to perform all the voluntary movements, commencing with the proximal and proceeding to the distal joints. The trunk movements should also be tested. The points to be observed in this examination are:—

(*a*) The ability or inability to perform the movements.
(*b*) The strength of the movements.
(*c*) The range of movement.
(*d*) The condition of the joints.

In the peripheral type of paralysis, the examination should be conducted along similar lines:—

(*a*) The patient's ability to perform movements.

(*b*) Where inability exists, the action of individual muscles concerned in any movement should be investigated. This includes not only the prime movers, but the antagonists and the synergic muscles.

(*c*) The degree of paralysis of the individual muscles is ascertained by observing whether any movement is produced, or whether any contraction can be felt.

The strength of the muscular movements of the hand may

be tested by means of the dynamometer, when a comparative record is required; but the most practical and efficacious procedure is to make the patient perform movements against the resistance of the examiner's hand, when a good idea may be obtained not only of the power, but also of the effort made by the patient to carry out the movement. Associated movements should also be observed.

4. *The condition and size of the muscles.* The examiner will observe whether there is increase or abnormal development of any muscles; whether muscles are wasted or diminished in size, and whether such wasting is accompanied by flaccidity or spasticity; whether fibrillary tremor is present or not; and whether any muscular contractures are giving rise to deformities of the limb. It ought to be noted also, whether the deformity is due to contracture of paralysed muscles—as in old-standing facial palsy; to over-action of non-paralysed antagonistic muscles—as in infantile paralysis; or to the over-action of the stronger muscles—as in the flexor contracture of an old-standing hemiplegia. The mechanical irritability of the muscles, or their contraction to direct percussion, should also be noted.

5. *Muscular tone.* The limbs being relaxed should be passively moved at the several joints, and observations made as to whether the normal flexibility of the limbs is present. In cases with increased tone (hypertonia) some degree of muscular resistance is felt; in greater degree, this is known as spasticity, and if severe it may be so great as not to be passively overcome. In loss or diminution of tone (hypotonia) the movements at the joints are in excess of normal (flaccidity). In extreme degrees the limbs may be made to assume abnormal positions as in locomotor ataxia. As a rule, hypertonia is associated with active, and hypotonia with absent, tendon jerks.

6. *Co-ordination of muscular action.* This is tested by asking the patient to touch, for example, the tip of the nose, or to approximate the index fingers of the two hands, to walk along a straight line, or to place the heel of one foot on the knee of the other leg, and so on. Finer movements may be tested by the act of writing. Tests should also be applied with the eyes closed, observation being made as to whether this induces or increases any want of co-ordination. Care should

be taken to discriminate between the unsteadiness caused by tremor and muscular weakness and that due to inco-ordination.

7. *Electrical reaction.* The faradic irritability, as being the more important, should first be tested. If diminished or lost, the reaction to galvanism should be investigated, first as regards the character of the reaction, whether brisk, feeble, or sluggish; and secondly, as to the polar reactions.

(For a fuller account of the electrical reactions the reader is referred to p. 50.)

Examination of the Sensory System

The sensory system is tested with a view to the investigation of the following points:—

1. Whether sensibility is normal, impaired, or lost.
2. The area or areas over which impairment or loss is detected.
3. The quality of the sensory impairment or loss.
4. The degree of the sensory impairment.
5. The existence of hyperæsthesia.

1. *Is sensibility impaired or lost?* A routine examination should be made in every case, including the following tests: cotton wool, pin-prick, finger touch, deep pressure, localisation and sense of position. If any of these sensibilities is defective or lost, a more minute and detailed investigation should be undertaken.

In cases where the patient's mental condition precludes a full examination, such tests ought to be employed as will give an approximate idea of the state of the sensory functions.

2. *The areas of defective sensibility.* The examination will show whether the defects are universal, unilateral, limited to one extremity, to a segment or portion of a limb, irregular or crossed, bilateral without inclusion of the face, extending up to a definite segmental level, or bilateral affecting the extremities or segments of the limbs, or whether the defects are of root or nerve distribution.

3. *The quality of the sensory loss.* The recent work of Head has shown that the various forms of sensibility are grouped differently in the peripheral nerves and spinal cord (p. 14). In some cases the lesion may be obviously spinal or peripheral, when the investigation of sensibility should at once be undertaken along the appropriate lines. The only

routine method of investigation is the examination of every form of sensibility. We will therefore describe separately the method of testing each.

1. Cutaneous sensibility, including:—
   (*a*) Tactile sensibility;
   (*b*) Discrimination of the two points of a compass;
   (*c*) Moderate degrees of heat and cold;
   (*d*) Cutaneous pain;
   (*e*) Extremes of heat and cold.
2. Deep sensibility, including:—
   (*a*) Contact or pressure;
   (*b*) Painful pressure;
   (*c*) Sense of position, passive and active;
   (*d*) Vibration.
3. Localisation.
4. Stereognosis.

1. Cutaneous sensibility

Tactile sensibility. This is tested by means of cotton wool rolled to a fine point. The patient should be shown what the testing medium is, and then told to close his eyes and indicate when and where he is touched. Where the epidermis is thick, or the sensibility generally of a low order, a camel's-hair brush may be substituted for the cotton wool.

Tactile discrimination. This is tested by means of the blunt points of a compass separated from each other at known intervals. As this form of sensibility varies widely in different localities, it is necessary to apply the test according to the normal standard of the region.

Care must be taken to apply the two points simultaneously, and a record obtained from a given area by touching the patient a definite number of times. Head has constructed a graphic formula by using the number of times touched by the double points as the numerator, and by the single points as the denominator. The distance between the compass-points must be kept constant during each test.

It is advisable, where possible, to use the corresponding skin area as the normal standard or control.

Moderate degrees of temperature. Considerable variations exist in different persons as to what are moderate shades of temperature; but a temperature between 25° C.

and 40° C. may be employed for the purposes of this test. Test tubes containing water of known and constant temperatures may be employed, and the patient, with his eyes closed, is asked to state whether touched with the warm or the cold tube.

Cutaneous pain. In testing this, care ought to be taken not to exercise pressure. It may be tested either by gently pulling the hairs, or by using the point of a steel pin, which should not be too sharp. The patient is asked to state whether the pin-point communicates a painful sensation or not. He should not be asked to say whether the head or the point of the pin is used, because, if tactile sensibility is preserved, it is easy to discriminate between them. If painful sensibility is impaired or abolished, the pin-point is recognised merely as a tactile impression. Any delay in the appreciation of pain should be noted.

Extremes of heat and cold. (Heat above 45° C. and cold below 20° C.) The patient should be asked whether he recognises them, whether they give rise to painful impressions, or whether they are confused with each other.

2. Deep sensibility

Contact or light pressure. For testing this a stimulus sufficient to produce displacement of the skin is required—such as the application of the finger to the surface of the skin.

Deep pain or painful pressure. Pressure exerted with sufficient force gives rise to a painful sensation in the deeper tissues—such as muscles or bones. This is tested by pinching the muscles.

For the measurement of painful pressure, Cattell has devised an instrument—the algometer—which, by means of a spring and a graduated scale, records the amount of pressure applied.

Sense of position—passive and active. This is a complex form of sensibility. In testing passive position, the limb is relaxed and the joints moved passively, care being taken that in effecting these movements, only the lateral surfaces of the limb are grasped, as this reduces to a minimum the aid given by cutaneous impressions. The patient, whose eyes are closed, is then asked to state the position in which he thinks the limb is placed; or he may be asked to place the corresponding limb in the like position. In doubtful cases the patient may

be asked to look at the position of the limb and to state whether the position is what he imagined it to be. The joint sensibility is that concerned mainly in passive movements of the limbs.

In testing the sense of movement (active position) the patient is asked to carry out some definite movement, such as approximating the points of the index fingers, or touching the nose with the finger, and the deviation or error observed. In uncomplicated cases, this is some measure of the amount of interference with the sense of movement and of the joint sense. But as this test calls into play motor and cerebellar functions, the examination of the sense of passive position alone is to be preferred.

Vibration. This is tested by means of a large tuning-fork placed upon the bones of the limbs, particularly over certain prominences—such as the styloid prominences, the malleoli or the sternum. The patient is asked to state whether he feels the vibration.

3. Localisation

In testing localisation it is important to ask the patient, if possible, to indicate the spot touched. In certain cases it is permissible to allow him to look at the limb before he points to the spot. The errors of localisation should be noted: whether distal or proximal, pre- or post-axial. Erroneous recognition as regards the limb, or side of the body (allocheiria) should also be noted.

4. Stereognosis

This is the ability to recognise without aid of vision the shape, size, and consistence of objects placed in the hands. Care should be taken not to diagnose astereognosis unless all forms of primary sensibility are retained (p. 251).

Examination of the Reflexes

The reflexes are of three kinds: the superficial, obtained by stimulation of the skin; the deep, obtained by striking the tendon of a muscle under slight tension; and the organic, which are concerned in swallowing, micturition, and defæcation.

The examination of the reflexes is of importance in two

ways: first, because their deviation from the normal may be direct evidence of organic disease of the nervous system; and second, because such deviation may indicate the site of the lesion.

In the normal healthy state, reflex action is unimpaired. Alterations in the reflexes may either be in the direction of increase or diminution.

Superficial reflexes. Exaggeration of these reflexes may be found in neurotic persons, and in conditions of hyperæsthesia, but is not significant of organic disease. On the other hand, impairment or abolition of the reflexes is of great importance. The superficial reflexes are lost in:—

1. Local abdominal conditions—such as a fat or pendulous belly.

2. Local inflammatory conditions of the abdominal organs.

3. Local injuries of nerves and root lesions, implying an interference with the reflex arc.

4. Some hysterical conditions, especially in association with hysterical anæsthesia. In our experience loss of the superficial reflexes is uncommon in functional conditions, and is more characteristic of an organic lesion of the pyramidal tracts.

5. Organic affections of the pyramidal system.

It is important to note in all cases, whether the alteration in these reflexes is uni- or bilateral, and whether the impairment is associated with changes in the deep reflexes.

For example, in organic hemiplegia, the superficial reflexes —especially the abdominal and epigastric—are diminished or lost on the hemiplegic side. This is associated with a definite increase of the deep reflexes upon the same side, and by an alteration in the character of the plantar reflex, which tends to revert from the normal flexor to an extensor type.

In cases of compression paraplegia, for example, at the level of the ninth dorsal segment, the superficial reflexes will be found normal above that level, but abolished below. This abolition will be associated with an increase of the deep reflexes and an extensor plantar response on both sides.

The superficial reflexes commonly examined are:—

Corneo-conjunctival: elicited by touching lightly, or blowing upon the cornea. The result is closure of the eyelids

Pharyngeal: elicited by touching the posterior wall of the pharynx, when a contraction of the pharyngeal muscles results.

Palatal: elicited by touching the soft palate. The result is retraction of the soft palate and drawing up of the uvula.

Scapular: elicited by stroking the skin between the shoulder blades. The result is contraction of the scapular muscles.

Bulbo-cavernosus: elicited by pinching the dorsum of the penis. The result is contraction of the bulbous part of the urethra.

Anal: pricking the skin over the perinæum evokes contraction of the anal sphincter.

Epigastric and *abdominal*: obtained by gently stroking the skin of the abdomen along the costal margin and the outer border of the rectus abdominalis muscle.

Cremasteric: tested by stroking the skin along the inner side of the thigh. The result is drawing up of the testicle.

Gluteal: by stroking the skin along the gluteal fold, the gluteal muscles contract.

The plantar reflex. In conditions of health, stroking the outer margin of the sole of the foot, or across the balls of the toes, produces inversion of the foot, flexion of the small toes, and of the big toe at the metatarso-phalangeal joint, and sometimes flexion of the thigh at the hip.

In testing this reflex it is essential to have the leg slightly everted at the hip joint, flexed at the knee, and relaxed at the ankle joint. The sole of the foot also should be warm and free from moisture. The important feature of this reflex is the movement of the great toe. A voluntary withdrawal or twitching of the foot, which occurs in sensitive persons, should not be mistaken for a reflex action. The variations from the normal commonly observed are of two kinds:—

1. *Abolition of all movement.* This occurs in coma, in certain hysterical conditions; in lesions of the reflex arc of the first sacral segment, with the sensory disturbances involving the sole of the foot in tabes; with paralysis of the muscles subserving the toes in acute poliomyelitis; in complete transverse lesions of the cord, and when the feet are cold.

2. *Extensor response, or extension of the big toe.* The substitution of an entirely different action, characterised by eversion of the foot and extension of the great toe is direct evidence of interference with the pyramidal system. It is

important to realise that such extensor response is not a loss of a superficial reflex, but merely a reversion to the normal reflex obtained in infants before they have learned to walk, and before the pyramidal fibres have received their medullary sheaths.

The paradoxical plantar reflex[1] is elicited by pressing deeply on the calf muscles, the patient lying on his back with the muscles of the leg relaxed. The result is an extensor movement of all the toes. This reflex is sometimes obtained when the plantar response is flexor, but is not present in health.

Oppenheim's reflex is elicited by pressing the thumb upon the leg just behind the inner border of the upper end of the tibia and firmly moving it towards the ankle. In normal cases the foot is inverted and the great toe flexed; but in lesions of the upper motor neurone the foot may be everted and the great toe extended. Extension may occur in cases where the plantar response is indefinite or even flexor.

Deep reflexes. A mere increase of the deep or tendon reflexes may, or may not, be of significance. Exaggeration is significant of organic disease (1) if it is present upon one side, (2) if below a definite level (the reflexes above remaining normal), (3) if associated with spasticity, or with changes in the superficial and plantar reflexes.

Loss of the deep reflexes is of great clinical importance, for it is practically certain that this phenomenon is never present as a normal condition. Their abolition therefore indicates organic affection of some part of the reflex arc—either the afferent limb, the motor cells of the anterior horns, the efferent limb, or the muscles. They are also abolished temporarily in acute lesions of the cerebellum, and in complete transverse lesions of the spinal cord above the lumbar enlargement.

The deep reflexes are tested by placing the limb in such a position as to relax the muscle whose tendon jerk is to be elicited. The muscle is then put under slight tension, and by tapping the tendon sharply with the finger or a small hammer, a muscular contraction of greater or less degree results. This test really indicates the state of the muscular tone, which depends in large part upon the integrity of the reflex arc.

The deep reflexes commonly examined and the methods of testing are as follows: —

[1] Gordon, *American Medicine*, 1904.

The jaw jerk. The patient opens the mouth, and the examiner slightly depressing the chin with his finger, so as to stretch the masseter muscles; taps the jaw, thereby inducing a contraction of the masseter muscles.

Scapular. Tapping the vertebral border of the scapula causes a retraction of the shoulder.

Triceps. The arm is semi-flexed at the elbow joint, and the tendon is struck just above its insertion into the olecranon process.

Biceps. This is obtained by flexing the elbow joint, placing the thumb upon the tendon of the biceps and slightly stretching the muscle. The thumb is then struck and the biceps contracts sharply.

Supinator and wrist jerks. The arm is supported with the elbow slightly flexed and the radius struck just above the styloid process.

Knee jerk. The patient is seated on a chair with his feet upon the floor. The knee being slightly flexed so as to relax the rectus femoris muscle, a contraction of the quadriceps is obtained on striking the patellar tendon. The simplest and most satisfactory method of reinforcing the knee jerk is to make the patient, when seated on a chair, extend the leg with the heel and toe remaining upon the floor. When the maximum extension in this position has been reached, he is asked to press the toes firmly on to the floor without raising his heel, and the tendon is sharply struck.

The *knee clonus* is obtained best by extending the leg with the patella freely movable. The tendon is then put upon the stretch by suddenly depressing the patella.

Ankle jerk. The examiner grasps the foot, the ankle joint being relaxed, and the leg everted at the hip and flexed at the knee. The tendo Achilles is stretched by slightly dorsiflexing the foot. The tendon is then sharply struck.

Should this method fail, the patient is made to kneel upon a chair and the tendon struck. Reinforcement is obtained by asking the patient to let his body sink down so that the buttocks approximate to the heels. By this method the hamstrings and calf muscles are relaxed.

Ankle clonus. This is obtained with the leg in the position for the Achilles jerk, the foot is grasped by the examiner's hand and suddenly dorsiflexed.

Organic reflexes. Those usually examined are:—

1. *Vesical.* The patients should be asked whether they have any difficulty in passing or in holding their water; whether they have any delay or hesitancy in the act of micturition. If there is any difficulty in passing water, local conditions (urethral, vesical and urinary) should be excluded.

If the patients complain of frequency of micturition or inability to hold their water, the examination should be made to determine (1) whether there is distension of the bladder, or (2) whether there is loss of sphincter tone, or (3) whether there is loss of control over the sphincter.

In women incontinence of urine, especially on sudden effort, may be due to local causes.

In all cases of nervous disease, where there is any possibility of interference with the sphincter control, the physician should not be satisfied with the patient's statements, but should himself determine – by percussion, or if necessary by passing a catheter—whether the bladder is distended.

2. *Rectal.* Inquiry should be made as to the existence of constipation, or loss of control over the action of the bowels. In the latter case, the state of the anal sphincter should be digitally examined, so as to ascertain whether it contracts. If the anus is patulous, there is interference with the reflex arc through the fourth and fifth sacral segments. If loss of control only occurs in the administration of purgatives, an impairment of cerebral control over the spinal reflex is indicated.

3. *Swallowing.* This is examined by testing the patient's ability to swallow fluids, semi-solids and solids.

4. *Sexual.* In certain cases it may be necessary to make inquiry as to the functions of the sexual organs; whether erection is possible or not, and ejaculation premature, delayed, or absent.

The reflexes affected in association with the sympathetic nervous system are:

Cilio-spinal. Pinching the skin of the neck provokes dilatation of the pupil.

Scrotal. Cold applied to the scrotum induces contraction of the dartos muscle.

Vesical, rectal, uterine. Stimulation of the bladder, rectum and uterus result in contraction of the bladder, rectum and

uterus. These reflexes may take place independently of the central nervous system. They may be initiated by voluntary effort.

Internal anal. A finger passed into the rectum is gripped by the contraction of the internal sphincter muscle.

Having completed the examination of the nervous system, the other systems should be examined *seriatim*. The absence of signs of organic disease of the nervous system makes this the more necessary, as nervous symptoms may be the earliest expression of derangement or disease of some other part of the body. Disease of the nervous system may be directly dependent upon disease of other systems, especially the cardio-vascular and urinary. Disease of the nervous system may also exist synchronously with, but independent of disease of other systems.

Lumbar Puncture: the Cerebro-spinal Fluid

The examination of the cerebro-spinal fluid is of considerable value in diagnosis. The fluid is obtained by a lumbar puncture. For this purpose the patient is placed upon his left side, or sits in a chair, with his back exposed to the operator; in either case the head and shoulders are bent well forwards. The space between the laminæ of the fourth and fifth lumbar vertebræ is preferably chosen for the insertion of the needle, on account of the absence of the spinal cord in this situation. The needle is passed in a slightly upward and inward direction about half an inch to one side of the mesial line, and pushed on until the point is felt to be free. The fluid is allowed to escape drop by drop to the extent of 3 to 5 c.cm. The needle is then withdrawn and the wound covered with a collodion dressing.

If the puncture is effected under strict antiseptic precautions and the patient kept in bed for twenty-four hours, no risk attends this operation. In many cases, however, even when precautions are taken, the patient may experience headache of a peculiarly disagreeable type when he sits up. This may depend upon the quantity taken away, which should not exceed 5 c.cm. In cases of intracranial tumour with great increase of intracranial pressure there is a risk of sudden collapse and death.

Two methods are used for the cytological examination of the fluid. A known quantity is centrifugalised for a definite time, some of the deposit transferred to a slide, fixed and stained, and the number of cells counted under the field of the objective. In the other method the fluid is handled as in the counting of blood, a few drops being placed in the cell of a Thomas-Zeiss slide, and the number of cells counted and estimated per cubic millimetre.

The *normal fluid* is a clear watery liquid of low specific gravity and alkaline reaction, and contains a small percentage of chlorides, some serum globulin, a trace of cholin, and a substance which reduces Fehling's solution.

Albumen is not normally present, but may be found in considerable quantity in cases of meningitis.

The quantity of cholin normally present may be increased in organic nervous disorders—a fact which has been used in the differential diagnosis of otherwise doubtful cases of organic and functional disease.

The cytology of the cerebro-spinal fluid. Only two or three lymphocytes are found in the centrifuged deposit of normal fluid. In many pathological conditions, the cells are materially increased. They are of two kinds:—

(*a*) Lymphocytes or mono-nucleated cells.

(*b*) Polymorpho-nuclear cells.

The lymphocytes are about the size of a red blood corpuscle. They contain one large deeply staining nucleus, surrounded by a rim of protoplasm.

The polymorpho-nuclear cells are larger, with a lobulated or horse-shoe shaped nucleus, and a large amount of protoplasm containing a fine granular material.

In addition, large endothelial cells may be seen, containing an irregularly shaped nucleus and much protoplasm.

The factors which determine a mono- or polymorpho-nuclear exudation are not sufficiently known, but it would seem as if the former is the effect of a toxic condition, while the latter is attributable to an acute inflammatory process associated with the presence of micro-organisms.

An increase of the lymphocytes is found in cerebro-spinal syphilis, in the parasyphilitic diseases—tabes dorsalis and general paralysis—in the early stages of tuberculous

meningitis, in post-basic meningitis, and in the convalescent or chronic stage of cerebro-spinal fever.

The polymorpho-nuclear cells are found in the acute forms of meningitis, including the rapid cases of tuberculous meningitis, and in cerebro-spinal fever during the acute period of the symptoms.

The bacteriology of the cerebro-spinal fluid. A bacteriological examination should be carried out in conjunction with the cytological. The presence of a specific micro-organism confirms a diagnosis which may have been still doubtful from the cell examination alone.

Electrical Examination

Normally a contraction is obtained when an electrode, attached to either a faradic or a galvanic battery, is applied to a muscle and the circuit closed. As this reaction may be modified or lost in certain diseases of the nervous system, the electrical reactions of the muscles are of some importance in the clinical examination of a case in which muscular weakness or atrophy is present.

Reactions in health

1. *Faradic contractility.* The faradic contractility of a muscle is obtained by applying the electrode over the muscle to be tested, more especially over the motor point or that portion of the muscle into which the motor nerve passes. The normal reaction is a brisk, well-sustained muscular contraction.

2. *Galvanic contractility.* Muscular contraction is only obtained when the circuit is closed and opened; none is observed during the continued flow of the current. As there are two poles, four series of contractions may be observed: two at closing of the circuit (positive and negative poles) and two at opening of the circuit (positive and negative poles). These are represented usually by letters, thus :—

K.C.C. = kathodal closing contraction.
A.C.C. = anodal closing contraction.
A.O.C. = anodal opening contraction.
K.O.C. = kathodal opening contraction.

The above order is that found in health, but owing to the

very strong current required to give the opening contractions their examination is usually dispensed with.

The reaction in health may therefore be stated as follows :—

Nerve. Reaction to faradism and to galvanism.

Muscle. Reaction to faradism, brisk and well sustained.
Reaction to galvanism, short and sharp.

$$K.C.C. > A.C.C. : A.O.C. > K.O.C.$$

Reactions in disease of the lower motor neurone

In certain states of muscular wasting—lesions of the lower motor neurone—an important alteration in the electrical irritability of nerve and muscle is obtained, known as the reaction of degeneration. This consists of a loss or impairment of the faradic irritability, in association with a modification of the contraction to galvanism, both quantitatively and qualitatively. The quantitative change is found in the replacement of the short, sharp reaction of health by a slow and sluggish muscular response. The qualitative change is seen in the equality of the K.C.C. and A.C.C., or the more rapid response of the A.C.C. If the strong current can be interrupted the K.O.C. will be obtained more easily than the A.O.C.

The reaction of degeneration (R.D.) is therefore as follows :—

Nerve. Reaction to both faradism and galvanism lost.

Muscle. Reaction to faradism impaired or lost.
Reaction to galvanism, slow and sluggish.

$$A.C.C. > K.C.C. ; \text{ or } K.C.C. = A.C.C. ; K.O.C. > A.O.C.$$

All degrees of incompleteness are seen from mere diminution of faradic excitability to the fully developed reaction of degeneration.

The state of the electrical reactions is a guide to prognosis.

Reactions in other diseases

In certain diseases—the myopathies, myasthenia gravis, tetany, and myotonia congenita (Thomsen's disease), the electrical reactions show distinctive and characteristic changes, which are detailed later in the descriptions of these disorders.

Part III

THE SPECIAL SENSES

SMELL

The **olfactory system** consists of: (*a*) the olfactory bulb, into which the olfactory nerves from the Schneiderian mucous membrane enter through the cribriform plate of the ethmoid bone. (*b*) The olfactory tract, which divides into two roots—an external passing towards the apex of the temporal lobe and the hippocampal lobule, and a mesial which crosses to the opposite side by the anterior commissure. (*c*) A cortical centre, which appears to be located in the lobus pyriformis or uncinate gyrus, and to some extent in the anterior part of the gyrus fornicatus.

Anosmia, or loss of the sense of smell, is a rare symptom of nervous disease. It is due more commonly to disease within the nose.

It may arise from the following causes:—

(*a*) Local conditions within the nasal cavities—such as enlargement of the turbinated bones, polypi, and fœtid atrophic rhinitis.

(*b*) Arrest of secretion and 'trophic' changes in cases of paralysis of the trigeminal nerve.

(*c*) Fracture of the base of the skull involving the olfactory bulb or tract in the anterior fossa.

(*d*) Local intracranial disease—such as meningitis, tumour of the frontal lobe pressing upon the olfactory tract, and tumours of the pituitary region.

(*e*) Hysteria, in association with other hysterical stigmata, such as hemi-anæsthesia (p. 527).

Parosmia, or perverted sense of smell, may be found in association with local nasal disease. We have seen it as

an isolated and troublesome after-effect of influenza. Subjective sensations of smell of central origin occur in cases of tumour involving the cortical centre, situated in the uncinate region of the temporo-sphenoidal lobe. They are also found as the aura in some cases of idiopathic epilepsy. Hallucinations of smell are occasionally met with in the insane.

Subjective sensations of smell are generally of an unpleasant character.

TASTE

Nerve fibres subserving the sense of taste are distributed over the tongue, soft palate, and palatine arches.

The course of the taste-fibres between the periphery and the brain is complicated, and is still the subject of discussion. The following facts appear to be proven: (*a*) The taste-fibres for the anterior two-thirds of the tongue are distributed in the lingual branch of the fifth nerve. From this nerve the major portion pass into the chorda tympani and run along with the facial nerve in the aqueductus Fallopii, as far as the geniculate ganglion. From this they pass directly through the sensory root of the seventh to the pons Varolii.

Entering the medulla oblongata by the sensory root of the facial nerve, and probably also by the roots of the glosso-pharyngeal, the fibres of taste pass upwards, but do not proceed along with the other sensory fibres to the internal capsule. They find their way by an as yet unknown route to the anterior end of the temporo-sphenoidal lobe, where the cortical centre for taste has been located in close relation to the olfactory centre. The histological researches of Campbell favour the anterior part of the island of Reil as a probable gustatory cortical centre.

(*b*) The taste-fibres, from the posterior third of the tongue, are distributed in the glosso-pharyngeal nerve. Some fibres may be transmitted to the brain by the roots of this nerve, but others course through the nerve of Jacobson and the small superficial petrosal nerve to the otic ganglion on the third division of the fifth nerve.

(*c*) The fibres from the soft palate and palatine arches pass through the Vidian nerve and great superficial petrosal to the geniculate ganglion. Excision of the Gasserian ganglion

is only rarely followed by loss of taste over the half of the tongue, and this is not a permanent defect.

The statistical observations of Cushing[1] and Davies,[2] based upon the studies of thirty-nine cases of excision of the Gasserian ganglion for trigeminal neuralgia, showed that only two of these had loss of taste, four impaired sense of taste, and thirty-three no impairment of taste after this operation. It would therefore appear as if the glosso-pharyngeal roots and the sensory root of the facial were the only transmitters of taste to the brain.

Ageusia, or loss of taste, occurs in peripheral facial paralysis, and in hysteria along with hemi-anæsthesia and interference with the other special senses. It may also be present in an incomplete form in catarrhal conditions of the mouth and tongue, and sometimes after influenza. When unilateral the patient may be unaware of its existence.

Parageusia, or perverted taste, is present in local disorders of the mouth, and subjective sensations of taste may arise from tumours of the uncinate region.

VISION

Anatomy. The visual system may be divided into two segments: a peripheral, comprising the retina, optic nerves, chiasma, optic tracts and their terminations in the basal ganglia—anterior quadrigeminal body, pulvinar thalami and external geniculate body; and a central, consisting of the nuclei in the pulvinar, and external geniculate body, the optic radiations and the cortical centres for sight.

The *optic nerves* are composed of three bundles of fibres: (*a*) a direct, or uncrossed, bundle lying externally, (*b*) a crossed bundle lying internally, and (*c*) a central, foveal or papillo-macular bundle. At the chiasma the crossed bundles, which come from the nasal halves of the retina decussate; but the direct bundles, coming from the temporal halves, pass backwards on the same side. In consequence of this arrangement the fibres from the homonymous halves of both retinæ run together in the optic tract. (Fig. 8.)

Some optic tract fibres may be traced into the superficial

[1] Cushing, *Bull. Johns Hopkins Hosp.*, 1903.
[2] Morriston Davies, *Brain*, 1907.

layers of the anterior quadrigeminal bodies. These probably subserve the reflex movements of the pupil (the optico-pupillary fibres). The majority of the optic tract fibres enter the external geniculate body and the pulvinar thalami optici.

From these ganglia a large mass of fibres is projected towards the cortex of the occipital lobe, as the *optic radiations*

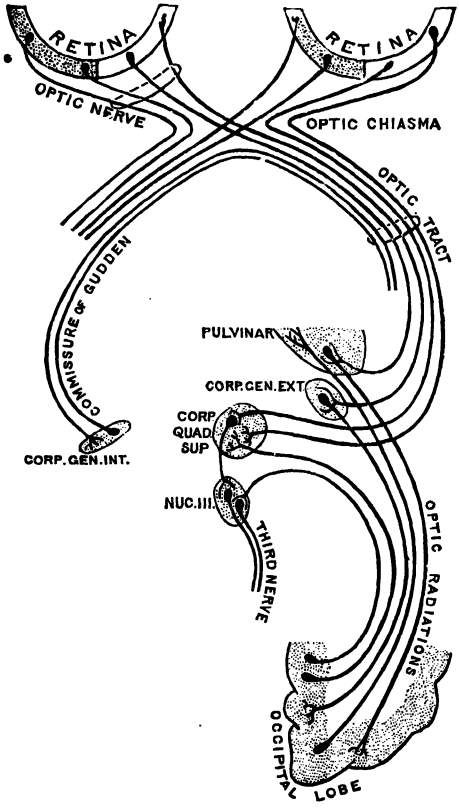


FIG. 8 (from Cunningham's 'Anatomy').—Showing the composition and connexions of the fibres of the optic nerves and tracts.

of Gratiolet. In their course towards the cortex these fibres pass through the retrolenticular portion of the internal capsule, in juxtaposition to, but behind, the sensory thalamo-cortical fibres; and farther on they lie subjacent to the angular gyrus and in close relation to the superior temporal convolution, mixing with the fibres of the inferior longitudinal fasciculus.

The optic radiations are made up of three distinct sets of fibres: (*a*) a bundle already medullated at birth, passing from the external geniculate body to the lips of the calcarine fissure. This forms the true radiation of the visual fibres according to Flechsig and Henschen.

(*b*) A system passing to the adjacent angular, cuneal, lingual, and fusiform gyri.

(*c*) An efferent or corticifugal tract, passing from the occipital lobe to the middle layers of the anterior quadrigeminal body.

The cortical visual centres. Henschen[1] has located the cortical half-vision, or visuo-sensory centre on the mesial aspect of the occipital lobe in the lips of the calcarine fissure. According to von Monakow and others, this centre occupies the lingual lobe, the cuneus, and a portion of the external aspect at the occipital pole. In other words, the half-vision centre occupies that portion of the lobe corresponding to the distribution of the 'line of Gennari,' which is in part formed by the fibres of the optic radiation and partly by fibres from the opposite occipital lobe (Mott).

In association with the primary or half-vision centre, Campbell and others have described a higher, or 'psycho-visual' centre, occupying the external surface of the occipital lobe and the remainder of the cuneus, and bounded in front approximately by the parieto-occipital fissure. (Figs. 9 and 11.)

The occipital lobe has extensive connexions with other portions of the brain, such as the opposite occipito-angular region by way of the splenium corporis callosi, with the frontal lobe by the superior longitudinal fasciculus and the tapetum, and with the temporo-angular region by the inferior longitudinal fasciculus.

The last connexion is an especially important one, as the angular gyrus, with its subcortical longitudinal fasciculus and the second occipital gyrus, subserve the recognition of written or printed language. This region corresponds to 'Wernicke's area.'

The frequent coexistence of homonymous hemi-anopsia with 'sub-cortical alexia' is due to the close relation which the longitudinal fasciculus has with the optic radiations, an

[1] Henschen, *Patholog. des Gehirns*, 1892.

intermingling of the fibres of both structures taking place in the region of the angular gyrus.

Physiology. Owing to the decussation of the fibres from the corresponding nasal halves of the retina (temporal fields) and the uncrossed position of those from the temporal halves (nasal fields), each optic tract contains fibres from homonymous retinal segments. The right optic tract, for example, transmits the visual fibres from the right halves of both retinæ, corresponding to the left homonymous visual fields. In this manner each half of the retina is represented in the corresponding basal ganglia, optic radiation, and half-vision cortical centre. Hence a destructive lesion of any of these

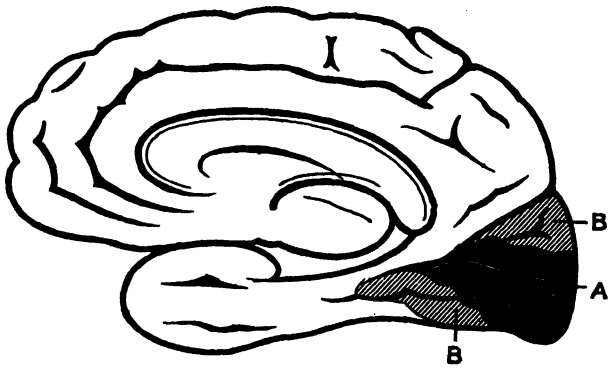


FIG. 9 (after Campbell). Showing the distribution of the cortical visual centre upon the mesial aspect of the hemisphere. *A*, primary visual centre. *B*, visuo-psychical centre.

structures on the right side will occasion, not blindness of the opposite (left) eye, but blindness of the homonymous right halves of both eyes, with loss of the visual fields on the left side. (Fig. 8.)

In homonymous hemi-anopsia from blindness of the corresponding halves of both retinæ due to cerebral disease, the line of demarcation rarely passes through the fixation point. This point corresponds to the distribution of the fibres of the papillo-macular bundle, which we have seen passes into both optic tracts.

The macular region therefore appears to have a bilateral cortical representation; but it is uncertain whether this lies in the calcarine area, as believed by Henschen, or in a higher

visual centre in the angular gyrus, as stated by Ferrier, or generally over the occipito-angular cortex, according to Monakow.

Sector or quadrantic defects of the visual fields—where corresponding retinal quadrants are rendered blind, with loss of vision in the corresponding portions of the visual fields—are sometimes observed. Beevor and Collier [1] state that the line of separation between the visual centres (corresponding to the upper and lower quadrants of the visual field) is probably the calcarine fissure; the lower visual field being represented in the upper or supra-calcarine area, and vice versa.

Diseases of the Peripheral Visual System

The reader is referred to works on ophthalmology for an account of diseases of the retina and local orbital conditions.

The optic nerve

A lesion of the optic nerve results in impairment or loss of vision upon the affected side.

The chief conditions affecting the optic nerve are optic neuritis and optic atrophy.

Optic neuritis. The common cause of optic neuritis is intra-cranial tumour or abscess, especially when associated with marked increase of intracranial pressure. It may also be found in renal disease (acute or chronic), in profound anæmia, certain toxic states, lead encephalopathy, cerebral syphilis, and acute infective disorders. It is occasionally found in association with acute myelitis in the cervical region.

In the early stages, blurring of the edges of the disc and hyperæmia of the optic papilla are seen. It is important to differentiate the early changes from somewhat similar appearances observed in some cases of hypermetropia; and it should be borne in mind that a slight blurring of the inner margin of the disc is a physiological condition in many eyes. As the neuritis increases, the hyperæmia and congestion become more intense, the disc is swollen and of a reddish colour, the central cup is filled in, the veins distended and the arteries smaller than normal. In severe cases the swelling may be as

[1] Beevor and Collier, *Brain*, 1904.

much as 7 diopters, and the surrounding fundus œdematous; the vessels present the same features as in the earlier stages, but extensive exudations may be observed either in patches or extending along the vessels, and occasionally obliterating portions of them. Hemorrhages may be seen scattered about on the disc and in its vicinity, and the general appearance may resemble that of albuminuric retinitis. As the swelling subsides the congestion lessens, the disc becomes pale, but the central cup remains filled in, and newly formed connective tissue may be seen surrounding the blood-vessels. The edges of the disc are blurred in places, and the arteries are small and tortuous.

Symptoms. Vision may or may not be impaired—normal vision being compatible with a considerable degree of swelling. The earliest symptoms are mistiness and blurring of sight, which may at first be intermittent but later become constant, and associated with definite impairment of vision. In the later stages, when optic atrophy has supervened, defective visual acuteness is present, and contraction of the visual field may be detected. Blindness is rare during the acute stage, but supervenes with the advent of complete optic nerve atrophy. The disappearance of the pupillary light reflex is coincidental with the loss of light perception.

The only *treatment* of any service is the operation of decompression, especially when optic neuritis is due to intracranial causes (p. 277).

Optic atrophy may arise as a primary condition or follow upon optic neuritis or local injury to the nerve. Several varieties of primary atrophy are described: (*a*) degenerative atrophy—as in the hereditary type of Leber and amaurotic family idiocy; (*b*) as a local phenomenon in general diseases—such as tabes dorsalis, general paralysis, and disseminated sclerosis; (*c*) from local lesions of the optic nerve—such as fracture of the base of the skull, tumour pressing directly on the nerve, and retrobulbar neuritis.

In the *primary optic atrophy*, which occurs in tabes dorsalis, the disc is grey-white in colour with clean-cut edges. The cup is not filled in, the lamina cribrosa is well seen, and there is little alteration in the calibre of the retinal blood-vessels. It is a bilateral condition, progressive in its course, and leading eventually to complete blindness. In some cases, however, a

small central zone of relatively good vision may persist for a long time, which would seem to be due to preservation of the central or papillo-macular bundle of optic nerve fibres.

A form of optic atrophy, probably arising from the presence of a local sclerotic patch in the nerve or tract, and frequently bilateral, is found in disseminated sclerosis. It does not present the greyish whiteness of the tabetic atrophy, but in cases of old standing may result in complete blindness. In the early stages of the disease, temporary attacks of amblyopia may occur without any obvious ophthalmoscopic signs, but in these cases some degree of optic atrophy usually develops in the later stages.

Secondary atrophy. Should optic neuritis not resolve under appropriate treatment, the natural course of events is towards destruction of the nerve fibres, atrophy of the optic disc, and impairment or loss of vision. In this condition the disc is white, the margins present a blurred appearance, the arteries are much reduced in size, and white lines are seen along the retinal blood-vessels.

Embolism or thrombosis of the central artery of the retina. This may result from a general infective malady, such as ulcerative endocarditis, or from local arterial disease. Thrombosis of the central artery on one side has been observed along with hemiplegia of the opposite side of the body. It is a rare association, resulting from extension of thrombosis from the internal carotid into the ophthalmic artery. In consequence of embolism or thrombosis of the central artery, anæmia of the retina, œdema, hemorrhages and atrophy of the optic disc are found.

The *symptoms* are sudden impairment, or loss of vision, from which a recovery may to a large extent take place.

Optic chiasma

The chiasma may be involved by tumours or syphilitic growths in the pituitary fossa, by the pressure of a dilated third ventricle, as in hydrocephalus and in some cases of acromegaly. It may also be affected by local lesions, as in disseminated sclerosis or from the pressure of atheromatous arteries.

The symptoms of lesion of the mesial part of the chiasma are blindness of the nasal halves of the retina, with loss of

the temporal fields on both sides (bitemporal hemi-anopsia). Lesions of the external surface only cause blindness of the temporal half of the retina (nasal hemi-anopsia). (Fig. 8.)

Optic tract

Lesion of the optic tract gives rise to homonymous hemi-anopsia to the opposite side.

The chief point by which a lesion of the optic tract may be distinguished from one affecting the optic radiations is that the pupil does not contract when light is thrown on the blind halves of the retina.

HEARING AND EQUILIBRATION

The eighth nerve trunk is composed of two separate nerves —the vestibular nerve from the semicircular canals, and the auditory proper or cochlear nerve. These two divisions have separate origins and terminations and different functions.

1. The vestibular nerve

Anatomy. The fibres of this nerve arise in the bipolar cells of the ganglion vestibulare, whose peripheral processes pass to the lining membrane of the semicircular canals. The central prolongations, or axones combine to form the anterior, mesial or vestibular root of the eighth nerve, which enters the pons Varolii between the restiform body and the ascending trigeminal root. The pontine nuclei, to which these fibres pass, are not exactly known, but vestibular root-fibres may be traced, after section of the eighth nerve distal to the accessory auditory ganglion, into the mesial vestibular nucleus, the nucleus of Deiters and the dorsal vestibular nucleus of Bechterew. Some nerve fibres also appear to come into direct relation with the roof nuclei of the middle lobe, and perhaps with the cortex of the lateral lobe of the cerebellum (Cajal). A direct connexion between the vestibular nerve and the nucleus of the sixth cranial nerve has also been observed. (Fig. 10.)

The main projection system from these nuclei is an indirect one to the cerebellar cortex of the middle and lateral lobes. There is a subsidiary system by way of the internal arcuate fibres of the pons.

Physiology. The vestibular nerve is the path by which impressions from the semicircular canals reach the brain, and more especially the cerebellum. Lesions of the semicircular canals in animals are followed by profound disturbances of equilibration. It has also been experimentally proved that section of the vestibular nerve results in similar well-marked

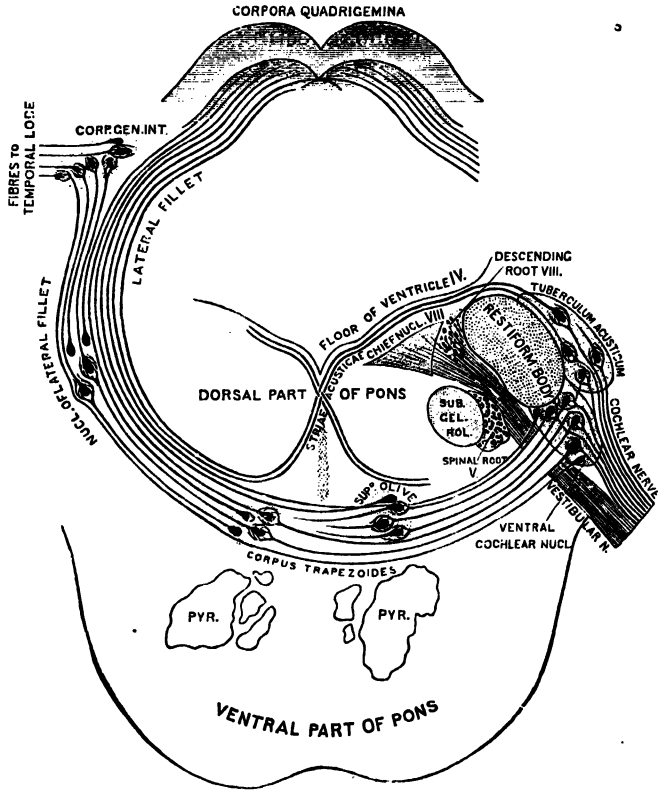


FIG. 10 (from Cunningham's 'Anatomy').—Showing the central connexions of the cochlear and vestibular divisions of the eighth nerve.

disturbances. After experimental section of this nerve, the animal rolls many times in succession towards the side of the lesion. Apparently with a view to counteract this vertiginous condition, the limbs upon the side of the divided nerve are extended and abducted from the body, and those on the opposite side are flexed and adducted. These effects are,

however, of a temporary character, the animal eventually regaining its equilibrium, although never completely.

Similarly in man, lesions of the labyrinth and of the vestibular nerve are associated with vertiginous attacks and disturbance of equilibration.

According to Sherrington,[1] the labyrinth is 'the receptor organ' of the head segments, which, in association with other receptive organs of the limbs and trunk, form a proprio-ceptive system for the maintenance of the equilibrium, the head ganglion of which system is the cerebellum.

2. The cochlear nerve

The cells of origin of this nerve are situated in the ganglion spirale. They are of a bipolar type, the peripheral processes of which terminate freely between the epithelial cells of the organ of Corti. The central processes form the posterior, lateral, or cochlear root of the eighth nerve. Many of the fibres terminate in the accessory auditory nucleus or auditory ganglion, which is situated on the trunk of the nerve outside the pons, while the remainder pass dorso-externally over the restiform body to terminate in the so-called *tuberculum acusticum*, which lies on the dorso-lateral surface of the medulla. It is probable that the auditory ganglion and the acoustic tubercle are parts of the same structure, to which the term 'cochlear end-nucleus' has been applied, as section of the auditory nerve distal to this structure is not followed by degeneration of the central conducting fibres. To obtain degeneration of these fibres, the cochlear nerve requires to be severed proximal to the auditory ganglion, and by this means the central auditory tract may be in part demonstrated.

The central auditory tract is formed of the fibres of the corpus trapezoideum, and includes the superior olivary bodies and both lateral fillets, but more particularly that of the crossed side. The tract is continued proximally within the tegmentum cruris as far as the internal geniculate body. The posterior quadrigeminal bodies would appear to be ganglia accessory to, rather than of, the central auditory tract. From the internal geniculate body fibres pass to the temporal lobe by way of the retro-lenticular portion of the internal capsule (figs. 7 and 10), and its destruction has been stated to cause deafness.

[1] Sherrington, *Brain*, 1900.

There is a complementary corticifugal tract, which passes from the first temporal gyrus, through the centrum ovale, and the lateral pontine system of the crus, to terminate in the upper portion of the pons—the temporo-pontine tract. (Fig. 7.)

Each ear stands in functional relation with both superior temporal gyri, as a unilateral lesion of the cortical auditory centre does not destroy hearing in the opposite ear alone, but probably impairs it on both sides. This is not surprising in view of the existence of extensive commissural connexions between the two auditory tracts, both in the trapezoid body, the quadrigeminal region, and the internal geniculate bodies.

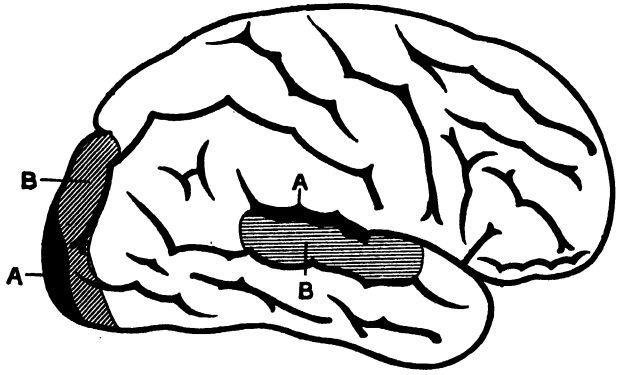


FIG. 11 (after Campbell).—This figure shows the cortical representation on the convexity of the centres for hearing and vision. Temporal lobe: *A*, primary auditory centre; *B*, audito-psychical area. Occipital lobe: *A*, primary visual centre; *B*, visuo-psychical area.

The cortical auditory centres. It has long been held that the cortical auditory centre lies in the superior temporal convolution, but the researches of Campbell would seem to place the primary auditory centre, or centre for the reception of simple auditory impressions, in the transverse temporal gyri of Heschl, which occupy the posterior end of the superior surface of the first temporal gyrus within the Sylvian fissure. These are hidden from view until the lips of the Sylvian fissure are separated. As in the case of the other cortical sensory areas, the primary auditory, or audito-sensory centre, is partly surrounded by an audito-psychical centre, which lies, according to Campbell, in the cortex of the

convexity of the hind end of the first temporal convolution. In this audito-psychical centre—in right-handed persons, on the left side of the brain—the auditory speech-centre is situated. Lesion of this area is associated with 'word-deafness,' or the loss of the interpretation of spoken language, and the power of recalling words and names. (Fig. 11.)

The auditory cortical centre has certain subcortical connexions, of which the fasciculus longitudinalis inferior, connecting it with the primary visual centre, and the fasciculus longitudinalis superior, connecting it with the inferior frontal gyrus, are the most important.

Diseases of the Eighth Nerve and its Vestibular System

The symptoms of paralysis or irritation of the eighth nerve consist of disturbances both of the sense of hearing and of equilibration.

The nerve is locally affected in two places: (*a*) in the subdural space; (*b*) in the labyrinth (cochlea and semicircular canals).

Nerve deafness

1. The common cause of nerve deafness is lesion of the internal ear or labyrinth. This may either be a primary disease of the labyrinth, to which the term Ménière's disease has been applied, or it may be secondary to an old-standing suppurative affection or sclerosis of the middle ear, when the term 'Ménière's symptom-complex' is applied to the condition.

2. Nerve deafness may arise from paralysis of the auditory nerve at the base of the brain. This is commonly due to a tumour growing either from the sheath of the nerve, or from the periosteum around the internal auditory meatus, involving the nerve. The deafness in these cases is an early symptom and may persist, with slight tinnitus and sometimes vertiginous attacks, for a considerable period before the onset of other symptoms signifying pressure upon adjacent structures. These symptoms are an incomplete degree of peripheral facial paralysis, unilateral cerebellar symptoms, and occasionally an associated anæsthesia over the distribution of the trigeminal nerve (see p. 267).

3. Nerve deafness may arise from cortical and subcortical lesions. In cases of this nature the lesion is usually softening or new growth, involving the temporal lobe and more especially the first temporal gyrus. A few cases are on record in which deafness has been due to softening of the first temporal gyrus on both sides. In unilateral lesions, involving the temporal lobe upon the left side, word-deafness is the more common symptom, and this may or may not be accompanied by impaired hearing to ordinary auditory impressions.

The absence of any marked degree of deafness in unilateral cortical lesions is due to the extensive bilateral distribution of the auditory fibres in the cerebral cortex.

Acute labyrinthitis—Ménière's disease

Ménière's disease is, in its true form, an acute destruction of the labyrinth, arising either from inflammation (acute labyrinthitis) or from hemorrhage into the labyrinth in the course of arterio-sclerosis or syphilis.

In consequence of this destruction characteristic symptoms are found. These are intense vertigo, vomiting, a feeling as of rotation of the body round its axis, profound disturbance of equilibration, and a well-marked nystagmus towards the unaffected side. The patient is of necessity confined to bed and assumes a characteristic attitude, lying upon the sound side.

The severe symptoms last from two to three days, and then gradually subside; but nystagmus may persist for a longer period, and pronounced movements of the head may induce a feeling of vertigo. Eventually all the irritative symptoms pass away, leaving only unilateral deafness and tinnitus aurium. Acute Ménière's disease, as above described, is a rare malady.

Chronic labyrinthitis—auditory vertigo—'Ménière's symptom-complex'

This is either a primary condition or secondary to disease of the middle ear.

1. The primary disease occurs mainly during early adult and adult life. It is often of a progressive character, and may be ascribed to constitutional causes—such as gout, senile degeneration, and arterio-sclerosis.

Symptoms. The symptoms of this variety show all degrees of severity, from attacks of severe vertigo, impelling the patient forcibly on to the ground and followed by vomiting, to slight vertiginous sensations, which may or may not be succeeded by nausea. In the majority of cases, more or less 'nerve deafness' and some form of subjective tinnitus are invariably present. As a rule, little difficulty is experienced in the diagnosis of the severe type of the malady; but in cases in which the aural symptoms are only slightly developed, the distinction between attacks of minor labyrinthrine vertigo and minor epileptic seizures is not easy. The presence, however, of labyrinthine symptoms favours the diagnosis of the local condition.

The symptoms are either unilateral or bilateral, with one ear more affected than the other. The deafness may be so slight as not to have been recognised by the patient, and the tinnitus is of a hissing, pulsating, or humming character. The attacks of vertigo have usually been preceded for some months, or a year or more, by tinnitus and deafness.

The disease is not necessarily progressive, although symptoms may persist for long periods. Suitable treatment may keep the vertiginous attacks in abeyance, and recovery eventually ensue.

2. Deafness, tinnitus, and vertigo form a frequent combination in those who have previously suffered from old-standing middle ear disease, either suppurative or sclerotic.

The symptoms are of gradual onset: the first attack of giddiness having been preceded for an indefinite period by tinnitus and deafness, having the features of middle ear deafness.

Tinnitus aurium is probably the most distressing symptom, owing to the persistence and severity of the sounds. In some cases it is so severe as to be actually painful. The sounds vary enormously—hissing, knocking, pulsating, musical, as of a train in a tunnel, humming, &c.

Vertigo occurs either in the form of seizures, in which the patient is hurled suddenly to the ground, or of a sensation as if he has been struck on the head. The sensation may be either that of turning to one or other side, or of external objects moving in one or other direction. Frequently combined with occasional vertiginous paroxysms is some degree

of instability in walking, so that the patient may stagger like a drunken man.

In old-standing cases, head sensations are a common and persistent symptom. They are a feeling of oppression on the vertex, a sensation of fullness or of throbbing, and headache.

Diagnosis. The Ménière group of symptoms presents little difficulty in diagnosis, when the labyrinthine deafness and tinnitus are associated with severe attacks of vertigo. Should, however, minor and temporary vertiginous seizures be present, without subsequent vomiting or nausea, the deafness and tinnitus being partial or slight, it may not be easy to differentiate the condition from minor epilepsy. In epilepsy some interference with consciousness is always present. There would, however, appear to be some cases in which the labyrinthine disease is associated with attacks of minor epilepsy, and others in which some interference with consciousness is a feature of the labyrinthine vertigo. The distinction between the two disorders can only be made by a careful study of the associated phenomena: impairment of memory and mental changes in epilepsy, progressive deafness and tinnitus in labyrinthine disease.

In cases presenting profound unilateral 'nerve-deafness' with tinnitus and occasionally slight vertiginous attacks, the possibility of the existence of a tumour of the auditory nerve should be kept in mind (p. 267).

Treatment. Bromide of potassium is the most useful remedy in the treatment both of Ménière's disease and of Ménière's symptoms. It should be administered, as in the treatment of epilepsy, over long periods of time. In most cases its administration reduces the number and severity of the attacks, and in many arrests them entirely. Hydrobromic acid, though favoured by many physicians, has not been found so satisfactory as the bromide salts.

If a specific cause is at work in any particular case, it should be combated either by the iodides, or by alkalies, colchicum, or mild purgation.

Quinine and salicylate of soda have been recommended in cases where the bromides are of little use, but in our experience their administration has been invariably attended by an aggravation of the symptoms.

Local counter-irritation, or the application of a seton behind the ear, may be applied in conjunction with medicinal treatment.

In severe cases—more especially if unilateral, progressive, and with painful tinnitus—operative destruction of the labyrinth or division of the auditory nerve has been recently practised.

Part IV

THE CRANIAL NERVES

The paralyses of the cranial nerves are of importance and interest not only as indications of local disease, but as evidence of localising value in intracranial disease.

THE OCULO-MOTOR NERVES

These are the third, fourth, and sixth cranial nerves. They are purely motor in function.

Etiology. (1) Paralysis of the third, fourth, and sixth cranial nerves may be due to traumata of the bones of the skull involving the sphenoidal fissure, periostitis, tumours, and inflammatory conditions within the orbital cavity.

2. The nerves may be involved within the skull, in their course between the brain and the foramina of exit, by new growths or inflammatory conditions, of which the commonest are syphilis and tubercle. They may also be affected in consequence of a general increase of intracranial pressure. The long intracranial course of the sixth nerves renders them especially liable to suffer from this cause. They may also be paralysed as a result of neuritis—rheumatic and perhaps other forms.

3. Toxic causes—such as diphtheria and ptomaine poisoning—may involve either the nerves or their nuclei of origin.

4. The nerves or the nuclei may be implicated within the pons and crura cerebri by new growths, localised vascular lesions (thrombotic or hemorrhagic); and in polio-encephalitis either superior affecting the grey matter of the aqueduct of Sylvius and third ventricle, or inferior affecting the grey matter of the fourth ventricle and aqueduct of Sylvius. The

nuclei may also be involved in a general polio-myelitis and encephalitis, and from hemorrhage into the third and fourth ventricles. The nuclei may be affected by chronic degenerative processes, either limited to the nuclei of the ocular muscles, or in association with similar changes in the bulbar nuclei.

5. Ocular paralyses may occur during the course of some chronic nervous disorders, especially tabes dorsalis and disseminated sclerosis.

6. Ocular affections, limited to the conjugate movements of the eyes, may be due to cortical and subcortical paralysis or irritation.

7. Ocular paralyses may also be found in association with migraine, myasthenia gravis, and hysteria.

8. Congenital ocular paralyses.

Symptomatology. The symptoms of ocular paralyses are of two kinds: (*a*) subjective, or those complained of by the patient, and (*b*) objective, or those recognised by the physician.

The subjective symptoms are:—

1. *Diplopia,* or double vision, is the result of strabismus. In certain positions two images are seen—the true and the false image; the latter always being observed by the paralysed eye. The two images ought to be described by the patient as regards their inclination to and degree of separation from each other, the direction in which this increases, and the elevation of the false image above or below the true.

The inclination of the false image depends on the degree of rotation of the eyeballs; and its separation from the true image is in proportion to the amount of paralysis present.

Homonymous diplopia is that in which the false image is seen on the same side as the paralysed eye. It is characteristic of paralysis of the abductor muscles of the globes—viz., the obliques and the external rectus.

Diplopia is said to be crossed when the false image is seen on the side of the sound eye. This is characteristic of palsy of the adductors—the internal, superior and inferior recti.

If the paralysed muscle is also an elevator of the eyeball, the false image is higher than the true; if a depressor, it is on a lower plane than the true.

In cases of divergent strabismus, therefore, diplopia

is crossed; in cases of convergent strabismus, diplopia is homonymous.

2. *Giddiness* and *erroneous projection* are only apparent when the action of the healthy muscle exceeds that of the affected muscle. They are most marked in recent cases.

A vertiginous condition may be induced when the patient looks only with the paralysed eye, owing to nystagmoid jerkings occasioned by attempts to keep the image on the macula. The patient may in consequence receive the impression that he himself is oscillating.

FIG. 12.—Paralysis of the left internal rectus muscle, showing external strabismus of the left eyeball.

The objective signs are:—

1. *Strabismus*, or squint. This is of two kinds:

(*a*) True or concomitant strabismus.

(*b*) Paralytic strabismus.

According to the deviation of the visual axes, strabismus is said to be convergent, divergent, oblique, superior, or inferior.

2. *Deviation of the ocular axes.* This is of two kinds:

Primary deviation is that which occurs in the paralysed eye on attempting to fix an object when the normal eye is covered; *secondary deviation* is that observed in the sound eye, and occurs from overaction of the corresponding muscle when the patient is asked to fix with the paralysed eye, the sound eye being covered.

3. *Limitation of movement* of the eyeball is in the direction of the paralysed muscle.

4. *Abnormal attitudes of the head.* In order to avoid or minimise double vision, the head is held in positions according to the paralysis present. In palsy of the adductors of the eyeball the head is directed to the sound side, and in palsy of the abductors, to the affected side. In paralysis of the elevators the head is thrown backwards, and in that of the depressors, forwards and downwards.

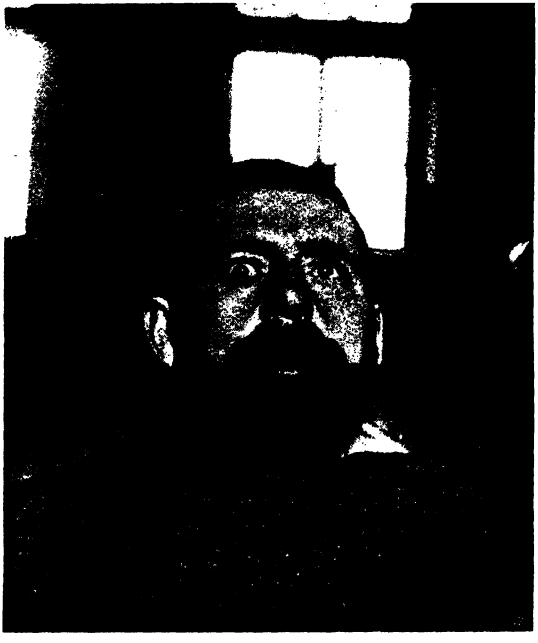


FIG. 13.—Paralysis of the right superior rectus muscle, showing the absence of upward movement of the right eyeball on looking upwards.

Paralysis of individual ocular muscles

The investigation of paralysis of the individual ocular muscles is facilitated by considering them as either adductors or abductors of the eyeball.

The adductors of the eyes are the internal, superior, and inferior recti muscles, supplied by the third nerve.

Internal rectus. This is solely an adductor muscle. If the muscle on the right side is paralysed the diplopia is

crossed, separation of the images occurs on looking to the left, the images are parallel and on the same level. (Fig. 12.)

Superior rectus. This is an elevator and also an adductor muscle. In paralysis the diplopia is crossed, on looking upwards the false image is higher than the true, and its separation from the true is greater above. The head is held backwards and inclined to the healthy side. (Fig. 13.)

FIG. 14.—Paralysis of the right external rectus muscle, showing internal strabismus of the right eyeball.

Inferior rectus. This is an adductor and depressor muscle. In paralysis the diplopia is crossed on looking downwards, and the separation of the images is greater below the horizontal. The head is slightly flexed towards the healthy side.

The abductors of the eyes are the external recti and the superior and inferior oblique muscles, supplied respectively by the sixth, fourth, and third nerves.

External rectus. This is purely an abductor muscle. The diplopia is homonymous, the images are parallel and on the same level. The separation of the images increases on

looking to the paralysed side. The face is turned towards the sound side. The eye is deviated inwards. (Fig. 14.)

Superior oblique. This muscle is both an abductor and a depressor of the globes. It moves the cornea downwards and outwards. The diplopia is found on looking downwards, and is homonymous; the false image is lower than the true, and the greater separation is at the lower ends. On looking downwards and inwards the difference in the height of the images increases; on looking downwards and outwards the obliquity increases. The head is turned downwards and the chin

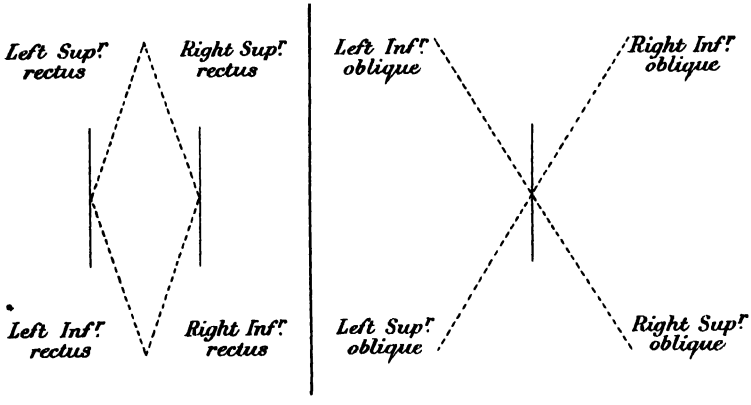


FIG. 15.—Werner's 'Artificial Memory' for the position of the false images in the ocular paralyses. On the left side the position of the true and false images is shown in palsy of the recti muscles; on the right side their position in palsy of the obliques. The continuous line represents the true image, the broken line the false image. For example, in paralysis of the right superior rectus, the false image is crossed, it is higher than the true, it is most marked on looking upwards, and the separation of the images is most marked above.

inclined towards the healthy side. Patients with this paralysis complain of confused vision when going upstairs.

Inferior oblique. It is an elevator and an abductor. It carries the cornea upwards and outwards. The diplopia is homonymous, is seen on looking upwards, the false image is higher than the true, and the greatest separation is above. The difference in the vertical separation increases on looking upwards, and in the horizontal on looking upwards and outwards. The paralysis of movement is in an upward and outward direction; the head is tilted backwards and the chin tilted towards the sound side.

Paralysis of individual ocular nerves

In complete paralysis of the *third nerve* there is palsy of all the external ocular muscles (except the superior oblique and the external rectus), the levator palpebræ superioris, the sphincter pupillæ, and the ciliary muscle. The eye cannot be rotated upwards, or directly inwards or downwards, but a slight downward and inward movement can be effected by the superior oblique. The eye tends to be pulled outwards (divergent strabismus) by the unopposed action of the external rectus muscle. The pupil is at first of medium size, but later dilates owing to the unopposed action of the dilator pupillæ. The light reaction and the power of accommodation are lost. If the paralysis is complete, there is no diplopia owing to the ptosis or drooping of the upper eyelid. To counteract the ptosis there is an overaction of the corresponding frontalis muscle. (Figs. 16 and 17.)

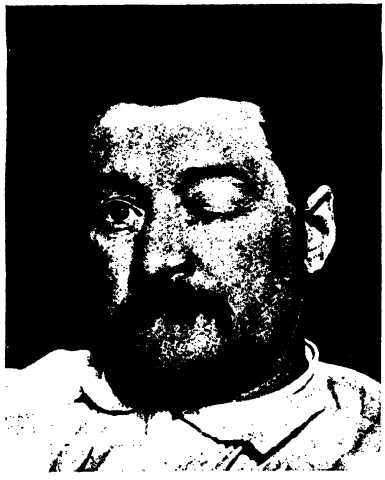


Fig. 16.—Paralysis of the left third nerve, showing complete ptosis of the left upper lid.

In partial paralysis any one of the individual muscles supplied by the nerve may be affected. As a general rule there is some degree of ptosis and some interference with the pupillary reactions.

The symptoms of paralysis of the *fourth nerve* are those already described for the superior oblique muscle.

Paralysis of the *sixth nerve* presents the symptoms already described under palsy of the external rectus muscle.

Recurring ocular paralysis

This is a rare form of ocular palsy, in which, after an attack of severe headache accompanied by vomiting, paralysis of an ocular nerve is temporarily induced.

The paralysis is usually of the third nerve, but the sixth may also be affected. Occasionally the palsy only involves the levator palpebræ or the internal ocular muscles. (Fig. 18.)

In typical examples of this affection, recurring headaches are succeeded by oculo-motor paralysis of variable duration, from a few days up to several weeks. As the malady progresses, the duration of the paralysis is prolonged until no recovery takes place, and the periodic headaches make no alteration in the degree of the palsy.

In all cases of this character, submitted to post-mortem examination, an organic lesion—tumour or exudation—has been detected involving the trunk of the affected nerve.

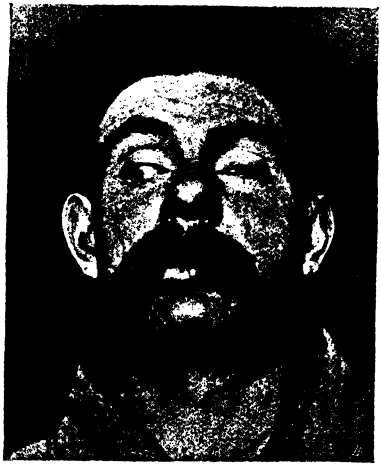


FIG. 17.—A case of partial bilateral paralysis of the third nerve. Note the double divergent strabismus, partial ptosis, and overaction of the frontalis muscles.

The pupillary light-reflex

The afferent fibres for the pupillary light-reflex are probably distinct from the visual fibres, and pass along the optic nerve, through the chiasma, into the optic tract (p. 54).

From the optic tract the pupillary fibres enter the quadrigeminal region, and thence are conducted by way of Meynert's 'fountain decussation' to the third nerve nucleus (Harris).[1] From this locality efferent fibres are transmitted through the ciliary nerves to the ciliary ganglion, and thence to the sphincter iridis muscle. It has recently been shown that the mechanism presiding over the sphincter action of the

[1] Harris, *Brain*, 1904.

iris lies in the ciliary ganglion and not in the oculo-motor nucleus (Bach).[1]

The pupil-dilating fibres pass downwards through the pons, medulla oblongata, and cervical portion of the spinal cord, probably in the gelatinous substance or adjacent posterior horn. They pass from the cord through the anterior roots of the first and second dorsal nerves and enter the inferior cervical ganglion of the cervical sympathetic, and are transmitted by the sensory root of the fifth nerve, the ophthalmic branch, and the long ciliary nerves to the iris muscle. They do not pass into the ciliary ganglion.

FIG. 18.—A case of recurring paralysis affecting the nerve to the left internal rectus muscle.

Loss of the light-reaction, with retention of pupillary contraction on convergence and accommodation, is known as the Argyll-Robertson reaction. The observations of Marina[2] show that this reaction would appear to be due to degenerative changes in the ciliary ganglion. In lesion of the roots of the third nerve, or of the nerve trunk itself, the pupil is dilated and inactive on convergence. Although usually a bilateral condition, it may be found on one side only.

The converse of the Argyll-Robertson phenomenon is occasionally seen—viz., retention of the pupillary light-reaction with loss of contraction on convergence.

[1] Bach, *Zeitsch. für Augenheilkunde*, 1904.
[2] Marina, *Annal. di Nervologia*, 1901.

Conjugate movements of the eyes

Lateral conjugate movements of the globes are produced by the associated action of the internal rectus muscle of one side, and the external rectus of the other. Both muscles, for the purposes of this movement, are innervated from the nucleus of the sixth nerve, connecting fibres passing from this nucleus to the opposite third nucleus. Interference with this movement may therefore be due to lesion (*a*) of the fibres which connect the cortical centre in the frontal lobe with the sixth nucleus, (*b*) of the sixth nucleus, or (*c*) the commissural fibres between it and the third nucleus.

A unilateral cerebral lesion, if irritative, causes conjugate deviation of the eyes to the opposite side; if destructive there is paralysis of conjugate movement to the opposite side, and there may be, if the lesion is recent, conjugate deviation of the eyes to the same side as the lesion, due to the over-action of the unaffected cortical centres of the opposite hemisphere.

Paralysis of conjugate movement, arising from a lesion in the pons Varolii, involving the sixth nucleus, is sometimes associated with facial palsy of the peripheral type upon the same side. The explanation of this association is topographical, the nucleus of the sixth nerve being surrounded by the intramedullary root of the seventh nerve. This association is absent in lesion involving the commissural fibres.

The other conjugate movements, upward and downward, are innervated from the third nerve nucleus and are rarely paralysed alone, although defect of the upward movements is found as a symptom of lesion in the neighbourhood of the quadrigeminal bodies.

Nystagmus

Nystagmus is a condition in which on conjugate movements of the eyes, the ocular deviation is poorly sustained, and is effected by a succession of slow or quick, coarse or fine, rotatory or oscillatory movements of varying range.

Nystagmus may arise from many causes, of which the following are the chief: —

1. Interference with the co-ordinating centres and connexions of the ocular muscles—

(*a*) In the cerebellum. Its features are described on p. 263.

(*b*) In the semicircular canals. Its features are described on p. 32.

(*c*) In organic diseases of the midbrain and pons.

(*d*) As a temporary symptom in some cerebral lesions.

(*e*) In organic diseases—such as disseminated sclerosis and Friedreich's ataxy.

(*f*) In paresis of the ocular muscles in peripheral neuritis.

2. In conditions associated with interference with vision—such as optic atrophy, errors of refraction, and albinism.

3. From strain and fatigue of the ocular muscles, as in high myopia, and miner's nystagmus.

4. As a congenital condition of central origin, as in spasmus nutans.

5. Spontaneous nystagmus. In this the movements are irregular, slow, oscillating, and wandering. They may be increased on conjugate movement. When present during rest, they are due to an inability to fix the eyes.

THE TRIGEMINAL NERVE

The fifth nerve consists of two primary divisions—the motor and the sensory—analogous to the motor and sensory roots of the spinal nerves. Situated upon the sensory division is the Gasserian ganglion, from which the three sensory branches of the nerve spring. Its deep origin and connexions have been described on pp. 6 and 10.

Sensory distribution. There is only slight overlap between the distribution of this nerve and that of the adjacent cervical plexus. The sensory loss following removal of the Gasserian ganglion is considerably less than the anatomical distribution of the nerve upon the face. The areas of epicritic and protopathic loss are almost identical, the former being slightly the larger and having an irregular posterior margin, as shown in fig. 19 (Davies). Excluded from the area of trigeminal distribution is notably the skin of the external auditory meatus, the auricle, and the major portion of the lower jaw. The area of loss of deep sensibility probably corresponds to that of

protopathic anæsthesia. The trigeminal distribution over the mucous membranes includes the nostril and half of the tongue as far back as the circumvallate papillæ, the margin of anæsthesia passing outwards along the line of these papillæ to the anterior pillar of the fauces, and hence along the centre of the soft and hard palates to the upper lip (Sherren). The fibres

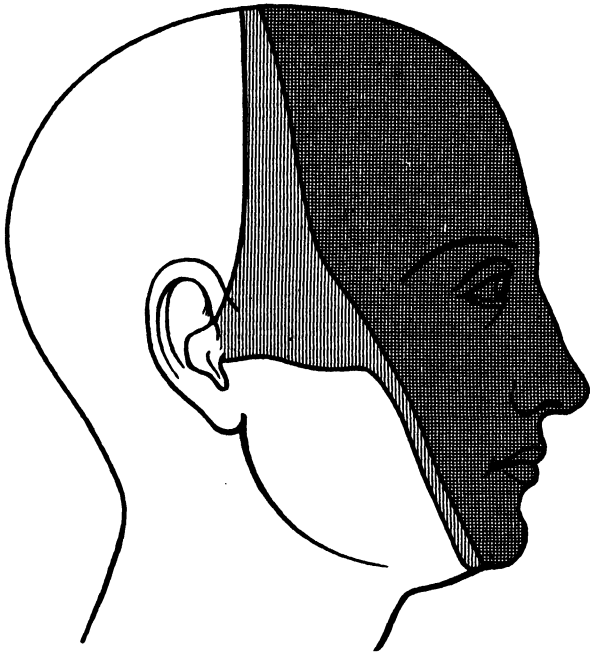


FIG. 19.—Chart showing the area of anæsthesia following excision of the Gasserian ganglion. The deeply shaded portion represents the area of both epicritic and protopathic loss; the lightly shaded shows the overlap of the epicritic loss (Davies).

subserving deep sensibility of the tongue are probably conveyed in the hypoglossal nerve.

The *causes* of trigeminal paralysis are either central within the pons, or peripheral, affecting the nerve roots. Central paralysis, both motor and sensory, may result from tumours or other lesions involving the central connexions of the nerves. The motor nucleus may be the seat of degenerative changes in bulbar paralysis. The intramedullary portion of the sensory root may be involved in tabes and syringomyelia.

Peripheral paralysis is usually associated with pressure upon the roots by new growths, or chronic inflammatory meningeal conditions.

The sensory root may be involved alone in cases of herpes of the Gasserian ganglion, and rarely the ganglion itself may be destroyed without any affection of the motor root.

Motor paralysis is limited to the muscles which move the lower jaw. Paralysis with atrophy and degenerative

FIG. 20.—Paralysis of the motor division of the left fifth nerve. The lower jaw deviates to the side of paralysis, when the mouth is opened.

electrical changes are observed in the temporal and masseter muscles. Paralysis of the pterygoid muscles is shown by an inability to move the lower jaw to the healthy side, and a deviation of the jaw to the paralysed side when the mouth is opened. (Fig. 20.)

Sensory paralysis. Loss or impairment of the epicritic, protopathic, and deep sensibilities is found over the cutaneous distribution of the nerve. The mucous membrane of the

tongue becomes dry, furred, and covered with the débris of food. The inside of the cheek may show ulcerated patches, where it has been unintentionally bitten during mastication. The mucous membrane of the nose is dry from an absence of secretion, and the sense of smell is impaired. Trophic ulcers may, or may not, develop upon the cornea.

The pupil on the side of the lesion is usually smaller than the normal one. Herpes may be occasionally observed over the distribution of the fifth nerve, but is usually confined to the supra-orbital, nasal, and supra-trochlear branches. The cornea is also sometimes involved in this condition.

The symptom known as *neuro-paralytic keratitis* is not an essential accompaniment of paralysis of the sensory root. Its presence would seem to be caused by irritation of the sensory fibres of the root, or of the Gasserian ganglion. In surgical removal of the ganglion, it may be prevented by closure of the lids during, and for a few days subsequent to, the operation, provided always that no septic irritation occurs at the base of the brain. We hold that its existence is evidence of a neuritic or inflammatory affection of the nerve rather than of paralysis.

There is no evidence that the trigeminal nerve contains either vaso-motor or trophic fibres for the skin of the face, nor is there any alteration in lachrymal or salivary secretion, after section of the nerve.

THE FACIAL NERVE

The facial is a mixed nerve, whose motor division is the portio dura, and whose sensory division is the pars intermedia, or nerve of Wrisberg, which takes origin in the ganglion geniculi facialis.

The motor division supplies all the facial muscles of expression, the stylo-hyoid and posterior belly of the digastric, the platysma, and the stapedius muscles. The sensory root is distributed to a skin area corresponding to the anterior surface of the external ear and the external auditory meatus. This is the Zoster area affected by irritative lesions of the geniculate ganglion (Ramsay Hunt). In the nerve of Wrisberg are also contained taste fibres from the corda tympani.

The whole of the facial nerve enters the internal auditory meatus in company with the auditory nerve. Thence it

passes through the aqueductus Fallopii, traverses the roof of the middle ear and issues from the temporal bone through the stylo-mastoid foramen. On the face it is divided roughly into three main branches: the oculo-facial, supplying the frontalis, corrugator supercilii, and the orbicularis palpebrarum muscles, the oro-facial to the orbicularis oris, and the mid-facial to the remainder of the muscles of expression.

Facial paralysis

This is the most common type of cranial nerve palsy, and is due to both peripheral and central causes.

Etiology. Of peripheral causation the most frequent is the so-called 'rheumatic,' or refrigeration paralysis. Eighty-eight per cent. of our cases arose from this cause, the remainder being due to suppurative middle ear disease, pressure from the forceps at birth, or in consequence of operations upon the mastoid bone. Other peripheral causes of facial palsy are basal syphilitic meningitis and the pressure of tumours in the cerebellar pontine angle.

Peripheral facial paralyses of rheumatic origin have a certain seasonal incidence. In our series the months of April, October, and November gave the greatest frequency—the cases amounting to nearly one-half of the total number. This would suggest an infective causation, corresponding in many ways to the seasonal incidence of acute poliomyelitis. The occasional occurrence of the malady in families, or amongst those who are working in the same locality, also supports this view.

Pathology. The pathological change underlying this form of facial palsy is a parenchymatous neuritis, with disintegration of the medullary sheaths. It is most apparent in the peripheral distribution of the nerve and at the distal end of the Fallopian canal, with a lessening intensity as the nerve is traced towards the geniculate ganglion.

Facial paralysis is favoured by a congenital narrowing of the Fallopian canal and stylo-mastoid foramen. Gowers has described the relative frequency of peripheral facial palsy in hemiatrophia facialis, in which the facial bones are atrophied.

Symptoms. In the common type of facial palsy, the muscles of one side of the face are completely paralysed. The patient is unable to frown or raise the eyebrows, to close the

eyelids, to whistle, or to smile. The naso-labial fold is obliterated, and in old-standing cases the angle of the mouth may hang down in a pouch-like fashion. All the wrinkles and furrows of the face on the paralysed side are erased, in

FIG. 21A.—A case of old-standing, right-sided, peripheral facial paralysis. Note the inability to close the right eyelids, the pouch-like appearance of the right side of the face, and the absence of the naso-labial fold.

consequence of which it assumes an expressionless, mask-like appearance. (Fig. 21A.)

In eating and drinking, food collects in the paralysed cheek, and fluid may run out at the angle of the mouth; tears roll down the face from weakness of the lower lid, which is sometimes everted into a condition of ectropion, with resulting conjunctivitis.

The tongue may present a false appearance of being protruded to the healthy side. The sensibility of the face is

not impaired, though pain—usually behind the ear—is a frequent symptom in the early stages.

The sense of taste may, or may not, be abolished on the anterior two-thirds of the tongue on the paralysed side. Taste may not be affected at all in mild cases of paralysis, owing to the limitation of the neuritis to the nerve distribution

FIG. 21B.—Depicts an old case of facial palsy of the left side showing paralysis on voluntary movement.

upon the face. In more severe cases, on the other hand, taste is usually lost early in the disease, owing to neuritic extension along the bony canal, involving the corda tympani.

Hearing is often implicated. If the facial neuritis is secondary to suppurative otitis media, air conduction is abolished, and the Rinné reaction is negative; but in cases where the nerve to the stapedius is paralysed, an increased sense of hearing (hyperacusis) may be present, especially to musical sounds.

In severe forms of facial palsy, the muscles show the

reaction of degeneration in from a week to ten days after the onset. In milder cases, a diminution of faradic irritability only is detected.

Peripheral facial palsy is usually one-sided, but a bilateral palsy is occasionally observed. The two sides are rarely simultaneously affected, and one side is more completely paralysed than the other. (Figs. 22 and 23.)

FIG. 22.

FIG. 23.

A case of bilateral facial paralysis.—Fig. 22, when at rest; fig. 23, on attempt at movement. Observe that the left side is more paralysed than the right. The mask-like appearance and absence of expression are well shown.

In the ordinary mild type of the disease, complete recovery ought to occur. But a number of cases of a more severe character are not uncommon, in which *secondary contracture* ensues. This condition is often accompanied by a feeling of stiffness of the face, and in states of repose, the contracture of the paralysed muscles may induce a false appearance of weakness on the healthy side. In these cases, attempts to close the eyelids will result in partial failure and in an over-action of the levator anguli oris, so that the mouth will be pulled forcibly over to the paralysed side; on showing the teeth the mouth is pulled to the non-paralysed side.

Facial palsy may coexist with *herpes zoster* of the auricle, external auditory meatus, and sometimes of the superficial cervical plexus, in whole or in part. In severe cases of this character deafness and symptoms of labyrinthine affection

may also be present. This 'syndrome' would appear to be due to an acute infective inflammation of the geniculate ganglion with secondary implication of the adjacent facial and auditory nerves in the internal auditory meatus (Ramsay Hunt). (Fig. 24.)

As facial paralysis may arise from destructive lesion any-

Fig. 24.—A case of right-sided facial palsy with herpes of the superficial cervical plexus. The herpetic vesicles have been artificially stained black.

where between the cerebral cortex and the facial musculature, several clinical types are observed.

1. A *cortical* and *subcortical palsy*, in which the oculo-facial muscles are relatively less involved than those about the angle of the mouth. The upper group is, however, impaired, for although the eyelids may be closed voluntarily, the patient may be unable to keep them closed against resistance.

In bilateral paresis—such as occurs in the pseudo-bulbar palsies—there is usually an overaction of the frontalis muscle, giving rise to a characteristic transverse furrowing of the forehead, and an associated paresis of the tongue

movements, defective articulation, and paralysis of the limbs. The electrical irritability of the muscles is unchanged.

2. *Nuclear paralysis* is usually of the chronic type and rarely occurs alone, being found in association with atrophic paralysis of the bulbar and motor trigeminal nerves, and with atrophy of the small muscles of the hands, arms, and shoulders. The lower part of the face, more particularly the orbicularis oris, is mainly affected and contrasts in a striking manner with the over-action of the frontalis. In these cases electrical alterations are of a quantitative rather than a qualitative character.

3. *Root paralysis.* The facial nerve root is not infrequently involved by tumours of the tegment of the pons and of the posterior fossa. In these cases the palsy is of the peripheral type, and is rarely complete. It is usually associated with palsy of the adjacent trigeminal and auditory nerves. The sense of taste on both the front and back of the tongue on the paralysed side may be impaired or lost.

4. *Facial paralysis of a peripheral type* is observed in diphtheritic, alcoholic, and lead neuritis. Weakness of the facial muscles is also seen in the myopathies and myasthenia gravis.

The **prognosis** of the refrigeration paralysis is based upon a study of the electrical reactions. If from a week to ten days after the onset there is only quantitative diminution of faradic excitability in the paralysed muscles, recovery will take place in from three to four weeks, or less. Should the faradic reaction be abolished with qualitative galvanic alterations, recovery may be indefinitely delayed. If these reactions persist over many months, ultimate complete recovery is doubtful, although some degree of return of movement is probable with contracture of the paralysed muscles.

A favourable type of facial palsy is that arising from temporary pressure upon the facial nerve accidentally inflicted during the performance of the complete mastoid operation.

Treatment. In mild cases a daily application of the faradic current will assist the natural tendency to resolution and recovery. In cases in which the faradic reaction is abolished, the application of the continuous current for a

prolonged period is essential. Massage of the facial muscles in all cases is of undoubted value in tending to preserve the muscular nutrition and the lines and contour of the face, and to prevent contracture of the paralysed muscles. On the return of some faradic excitability, combined galvano-faradism may be usefully adopted. In cases arising from otitis media, the cure of the aural condition is essential, and this may, if necessary, be followed by an anastomosis of the peripheral end of the cut facial nerve with the hypoglossal nerve.

Facial spasm is described under The Tics, on p. 588.

THE GLOSSOPHARYNGEAL AND VAGUS NERVES

The nerves distinguished by these names are in reality portions of one large mixed nerve, having a common origin for their motor and a common termination for their sensory fibres in the medulla oblongata. The nerve roots, as they issue from and enter the medulla, form a series of fasciculi between the restiform body and the inferior olive. To the uppermost of these roots the term 'glossopharyngeal' has been applied, and to the middle 'vagus.' To the lowest the term 'accessory' has been applied under a misapprehension: they being in reality the most distal of the vagal roots. The term 'accessory' was originally given by Willis to the nerve which springs from the spinal cord, enters the skull through the foramen magnum, and issues therefrom in company with the vagi through the jugular foramen.

The deep origin and connexions have been described on pp. 7 and 10.

The peripheral distribution of the vago-glossopharyngeal nerve is very extensive, embracing such structures as the soft palate, the vocal cords, the tongue (in part), the pharynx and œsophagus, the lungs, heart, stomach, and to some extent also the intestines.

Etiology. This nerve may suffer paralysis, in whole or in part, from lesions of:—

(*a*) Its medullary nuclei: as in bulbar palsy, tabes, syringomyelia, and disseminated sclerosis.

(*b*) Its intracranial roots: as in lesions of the meninges,

intracranial tumours, aneurisms, and disease of the bones of the base of the skull.

(*c*) Its extracranial course: from tumours, injuries, and other sources of pressure. The vagus nerve, particularly its recurrent laryngeal branch, is especially prone to compression on the left side from aneurism of the arch of the aorta, and from mediastinal growths and tuberculous disease of the apex of the right lung.

(*d*) Certain poisons and general infective disorders affect the vagus as part of an extensive peripheral neuritis. Of these may be mentioned alcohol, lead, arsenic, diphtheria, and influenza.

Symptoms. The symptoms vary according to the position of the lesion; but may be separated, for descriptive purposes, into those referred to (*a*) the glossopharyngeal nerve, and (*b*) the vagus nerve.

1. Glossopharyngeal paralysis

There is no case on record of isolated palsy of this nerve. The trunk of the nerve contains the fibres of taste for the posterior third of the tongue, but it is uncertain how far taste fibres are contained in the roots. Owing to a branch which is given off from the petrous ganglion, the nerve gives fibres to the tympanic plexus for the supply of the mucous membrane of the tympanum and the Eustachian tube. The pharyngeal branches fuse with similar branches of the vagus to form the pharyngeal plexus, which is both motor and sensory for the superior constrictor muscle and mucous membrane of the pharynx. Symptoms of glossopharyngeal palsy, therefore, are difficulty in swallowing, anæsthesia of the upper pharynx, and loss of taste over the posterior third of the tongue.

2. Vagus paralysis

The larynx is supplied by two branches of the vagus nerve; the superior laryngeal, which is sensory for the mucous membrane of the larynx above the vocal cords and motor for the cricothyroid and epiglottideus muscles; and the recurrent laryngeal, which supplies the remaining muscles and the mucous membrane below the vocal cords and the trachea.

The character and symptoms of the several forms of laryngeal paralyses are indicated in the following table :—

Paralysis.	Symptoms.	State of Vocal Cord.	Causes.
Complete bilateral palsy	Aphonia, stridor, inability to cough	Motionless in the cadaveric position	Organic only, as in tabes
Complete unilateral palsy	Voice hoarse, cough impossible, stridor on deep inspiration	Paralysed and motionless in cadaveric position	Organic, usually pressure on recurrent laryngeal nerve
Bilateral abductor palsy	Voice not affected, inspiratory stridor, cough normal	Cords approximate on phonation. Do not separate in inspiration	Organic, and indicates early palsy
Unilateral abductor palsy	Voice and cough not affected. May be slight inspiratory stridor	Cords approximate on phonation. Paralysed cord, immobile on inspiration	Early organic disease
Bilateral adductor palsy	Aphonia	Inspiratory movement normal. Imperfect movement on attempted phonation.	Functional

In the earlier stages of pressure upon the vagus, and in nuclear degeneration, the first symptom to be observed is paralysis of the abductor mechanism (Semon). This is a well-recognised clinical phenomenon, and may be due to the fact that the abductor and adductor fibres occupy distinct and separate bundles in the nerve.

Other symptoms are found in paralysis of the soft palate, either on one or both sides, according to the position of the lesion and anæsthesia of the laryngeal mucous membrane.

In bilateral affections of the vagi, there is irregularity in and acceleration of the heart's action. This is due to paralysis of the cardio-inhibitory mechanism derived from the upper vagal nerve roots. Symptoms referred to the œsophagus, stomach, intestine, and lungs, although theoretically possible, and stated to exist by some writers, are vague and not sufficiently authenticated to be regarded as characteristic.

THE HYPOGLOSSAL NERVE

This nerve is frequently paralysed in cerebral disorders. It is a common accompaniment of hemiplegia, from lesion of the supranuclear fibres. The nucleus is involved in bulbar palsy, and may be in tabes and syringomyelia. The nerve roots are affected in basal meningeal lesions and in caries of the occipito-atloid region. In the neck the nerve may

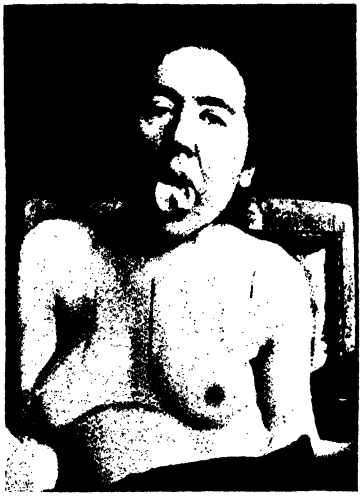


FIG. 25.— A case of syringo-bulbia, showing paralysis and atrophy of the left side of the tongue.

FIG. 26.— Bilateral paralysis of the tongue from an intramedullary lesion affecting the roots of the hypoglossal nerves.

be injured by wounds or by the pressure of tumours. (Figs. 25 and 26.)

Symptoms. In supra-nuclear paralysis of this nerve the tongue is protruded towards the paralysed side in a curved fashion. It can be moved from side to side within the mouth, but there may be some interference with the formation of the lingual consonants. If the lesion is nuclear, or infra-nuclear, the tongue shows marked wasting either on one or both sides; the mucous membrane is wrinkled and fibrillary tremors of its muscles may be detected. In bilateral palsy it

may not be possible to protrude it beyond the dental arch. In these cases a difficulty in chewing and swallowing is usually superadded. (Fig. 26.)

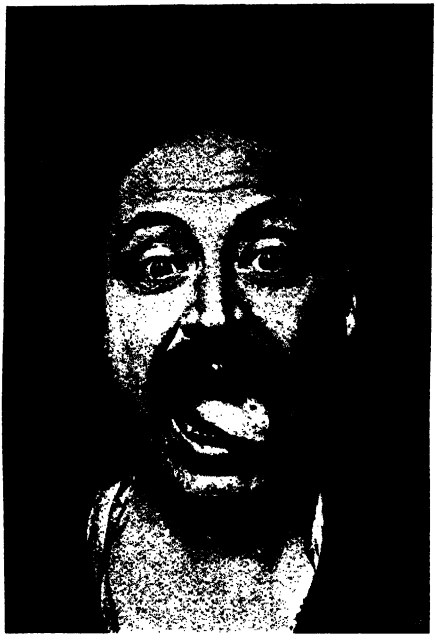


FIG. 27.—Unilateral paralysis of the tongue, which is protruded to the left side or the side of paralysis.

SPINAL ACCESSORY NERVE

This nerve supplies the sterno-mastoid and the upper third of the trapezius muscles.

It may be paralysed in consequence of disease of the upper cervical region as a part of a progressive muscular atrophy, and in caries of the cervical vertebræ, especially affecting the occipito-atloid and atlo-axoid joints.

Its paralysis is seen in impairment of the normal prominence and outline of the neck, by wasting of the upper part of the trapezius and by drooping of the shoulder. Paralysis of the sterno-mastoid alone is shown by an inability to turn the head to the opposite side, while on depression of the chin,

the head deviates towards the sound side. Palsy of the trapezius alone is demonstrated by an inability to shrug the shoulder.

Under this heading mention may be made of a permanent contraction of the sterno-mastoid, giving rise to a form of

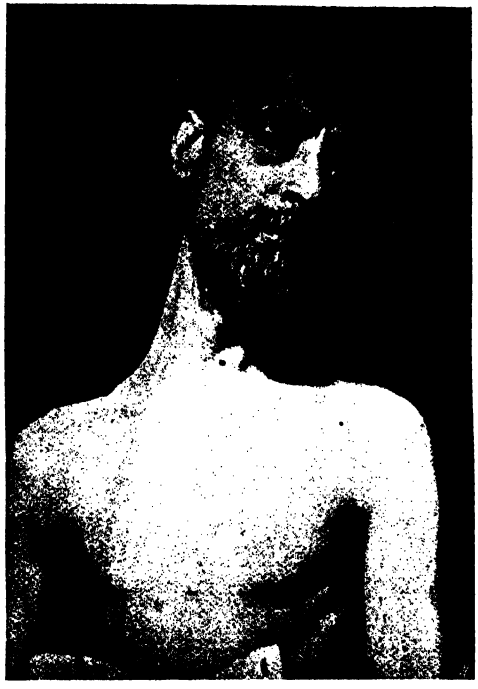


FIG. 28.—Paralysis of the right spinal accessory nerve, showing the absence of the normal outline of the neck, with drooping of the shoulder.

'wry neck.' This condition may be congenital from intrauterine injury, or follow upon injury to the spinal accessory nerve or sterno-mastoid muscle during delivery, or in the case of adults from any traumatism about the neck. The symptoms are: an inability to turn the head towards the side of the paralysed muscle, and a fixation of the head in the direction of the normal action of the sterno-mastoid muscle.

Part V

THE PERIPHERAL NERVOUS SYSTEM

STRUCTURE OF THE PERIPHERAL NERVES

A cross-section of a peripheral mixed nerve shows (*a*) an epineurium or nerve sheath; (*b*) a perineurium, which surrounds bundles of nerve fibres and gives off (*c*) an endoneurium, which surrounds the individual nerve fibres.

The nerve sheath contains fat, blood-vessels, and small nerves (nervi nervorum). Small blood-vessels also pass with the perineurium to supply nerve fibres. In the peri- and endoneurium are lymphatic channels which communicate with the general lymphatic system.

The spinal nerves consist mainly of medullated fibres, but some probably contain non-medullated fibres. The medullated fibres are composed of an axis-cylinder, a medullary sheath, and a neurilemma sheath. The axis-cylinder which lies near the centre of the fibre is formed of fine fibrillæ. The medullary sheath surrounds the axis-cylinder, and is a homogeneous refractile structure composed of myeline. When degenerating, this breaks up into droplets, which stain black with osmic acid. The myeline sheath is surrounded by the neurilemma sheath, a structureless elastic membrane. At certain places the myeline is absent or much attenuated, so that the axis-cylinder appears to be directly surrounded by the neurilemma sheath. These are known as the nodes of Ranvier, and between each pair of nodes is the neurilemma nucleus, which is supposed to possess a nutritive influence over the nerve fibre. In addition to these nodes obliquely placed incisures of the myeline sheath, known as Lantermann's incisures, are present.

Non-medullated or grey fibres are found chiefly in the sympathetic and olfactory nerves.

Chapter I

LESIONS OF THE PERIPHERAL NERVES

Etiology. Local lesions of the peripheral nerves may arise from the following causes :—

1. *Local inflammatory conditions*—such as syphilis, gout, and rheumatism.

2. *Wounds*—incised, lacerated, gunshot, and operative. Accidental injuries are specially common about the wrist, while gunshot injuries are more frequent in the sciatic and musculo-spiral nerves. Injuries to nerves may also be inflicted during an operation. The facial nerve, the cervical plexus, and the spinal accessory nerve are especially liable to injury during operations upon the ear and in the neck.

3. *Stretching and tearing of the nerve.* This may result from excessive traction—such as may arise from traction exerted during delivery, from a fall upon the shoulder or side of the head, from manipulations of joints under anæsthesia, from the breaking down of contractures, or from excessive traction upon the arm, as in operations upon the breast or axilla.

4. *Pressure.* Pressure upon nerves may be due to tumours, dislocations, fractures, callus, fibrous adhesions around or in the neighbourhood of nerves, and to the pressure of bandages, splints, and crutches.

Nerve injuries found in association with fractures may be primary, and due either to direct laceration of the nerve by a fragment or splinter of bone penetrating the nerve, or to the actual violence which leads to the fracture causing traction on or contusion of the nerve. This injury may occur at some distance from the fracture.

Secondary lesions of nerves may occur at a late period when the nerve sheath is irritated or involved by callus or fibrous tissue.

Many nerve lesions may, without causing any solution of the anatomical continuity of the nerve, give rise to a temporary or permanent loss of function. Physiologically, the function of the nerve may be impaired or abolished; anatomically, the nerve may be completely or partially severed. It

is obvious that the anatomical and physiological conditions do not necessarily correspond: as a nerve which is structurally continuous may lose its conductivity and be functionally destroyed from the presence of various local causes such as callus in the neighbourhood of a fracture or cicatricial tissue.

Symptomatology. The symptoms of peripheral paralysis, whatever the cause, may be motor, sensory, trophic, and vaso-motor.

Motor symptoms:

1. Atrophic paralysis of the flaccid type.
2. Abolition of the tendon reflexes.
3. Electrical alterations characteristic of the reaction of degeneration (p. 51).
4. If deformities and contractures exist, they are due to the over-action of non-paralysed muscles. These deformities are of late development.

Sensory symptoms. The sensory symptoms are both subjective and objective. The former consist of numbness, tingling, 'pins-and-needles,' or of sensations of burning pain in the distribution of the affected nerve or plexus. Subjective sensory symptoms are usually accompanied by pain or tenderness on pressure along the nerve trunk. On the other hand, in some forms of compression neuritis, no pain or tenderness may be present.

The objective signs depend largely upon the nature of the nerve lesion. In acute inflammatory lesions, sensory symptoms are well marked; on the other hand, if the nerve is injured by slow compression, the motor symptoms are early and pronounced, and may not be accompanied by sensory symptoms, except in the later stages. The area of objective sensory loss resulting from an isolated nerve lesion is not coterminous with the anatomical skin distribution of the nerve, because of the overlap existing between adjacent nerve areas. The area of sensory loss is therefore contained within the anatomical distribution of the nerve, and consists of a larger area in which there is epicritic loss, and a less extensive zone of protopathic loss (p. 14). The sensibility to contact and deep pressure and the sense of passive position and of movement are retained if the cutaneous branches only are involved. In the area between the margins of protopathic

and epicritic loss, sensibility is modified in that painful stimuli cause greater discomfort than normal, and the pain which results is widely diffused and cannot be localised.

Trophic symptoms. These consist of atrophy of the skin, which becomes thin and glossy; the development of ulcers and sores; atrophy of bones and joints, and falling out of the hair and nails. According to Head these changes are associated with loss of the protopathic sensibility. They are therefore only present when the nerve is severely affected. As in slight injuries and minor degrees of neuritis, the sensory loss may consist merely of epicritic anæsthesia.

Recovery of sensation after injury to nerves, or after reunion of a divided nerve, takes place first in the protopathic and subsequently in the epicritic sensibilities. With the restitution of the former, there is a rapid recovery and healing of ulcers, sores, and other trophic phenomena (Head).

Vaso-motor and secretory symptoms. There may be redness of the skin with local rise of temperature and hyperidriosis. In the later stages blueness, diminution of local temperature, and dryness of the skin are present. Œdema is not a common feature.

Diagnosis. A differential diagnosis has to be made between peripheral nerve lesions and *Arthritic muscular paralysis* arising from lesions of joints. In the latter, only minor degenerative electrical changes are detected in the muscles; there is no sensory loss or impairment, and the distribution of the paralysis is in direct relation to the joint injury and not to that of any nerve.

Another condition requiring to be distinguished from peripheral nerve lesion is *Ischæmic paralysis.* This results from the too tight application of bandages or splints, causing swelling of the free extremity of the limb, associated with great pain and tenderness. At a later stage there are rigidity and hardness of the muscles, loss of voluntary power, and limitation of passive movement. The electrical reactions are normal and there is no objective sensory loss.

In cases where a nerve is paralysed by slow compression, such as may result from the presence of a cervical rib or local overgrowth of bone, the condition must be distinguished from *Syringomyelia.* In pressure paralyses the motor and sensory

impairment is limited to the distribution of the affected roots or nerves and is peripheral in type. In syringomyelia the paralysis is often bilateral, the sensory loss dissociated and characteristic of a central cord lesion; and, further, the symptoms progress showing involvement of other regions of the cord and interference with the spinal tracts. In all doubtful cases an X-ray examination should be made.

A word may be said as to the differential diagnosis from *Hysterical paralysis* with contracture. In this affection there is no change in the electrical excitability of the muscles, and the sensory loss shows hysterical features described on a later page (p. 535). Hysterical contractures, moreover, differ essentially in appearance from those found in peripheral lesions.

Prognosis. The prognosis as regards recovery from peripheral nerve paralyses depends entirely upon the nature of the lesion and the degree of injury to the nerve fibres.

In slight cases—such as result from pressure, blows, or minor degrees of traction, in which the electrical excitability is only slightly or not at all impaired—recovery may be expected to take place completely and within a few weeks.

In severer cases—associated with the reaction of degeneration and wasting of muscles—progress may be slow, and recovery only occur after many months.

If the nerve is divided, completely or incompletely, or severely lacerated, excision of the divided ends and suture of the nerve ought to be undertaken. An important question, and one often hard to answer, is for how long a period after the receipt of an injury is recovery of the nerve possible. According to Sherren,[1] there is no reason why good results may not be obtained after an interval of three or even more years. In cases of this nature the condition of the paralysed muscles should be carefully investigated, as being more likely to influence the prognosis than any other factor. If galvanic and mechanical excitability of the muscles are abolished, recovery of muscular power is highly improbable.

[1] Sherren, *Injuries of Nerves*, 1909.

Sherren recommends re-suture of the nerves, even in cases in which a return of motor power is unlikely, with a view to the restoration of protopathic sensibility, and the consequent resolution of trophic sores and ulcers.

In cases, on the other hand, in which the galvanic irritability is retained, although polar changes may be present, re-suture of the nerve should always be undertaken.

If the suture be successful, recovery takes place in the following order:—

(*a*) Protopathic sensibility begins to return in from six to sixteen weeks, and is complete in from four to twelve months.

(*b*) Epicritic sensibility is re-established in from twelve to eighteen months.

(*c*) Recovery of the motor functions varies, being more rapid the nearer the lesion is to the periphery of the limb; the time required is from one to two years.

Treatment. The treatment of nerve paralyses is conducted upon general lines. In the first place, it is necessary to maintain the nutrition of the muscle fibres. This is partly done by means of the faradic or galvanic currents, according to the form of electrical stimulation to which the muscles respond; and partly by massage and passive movements at the joints. So far as the muscular weakness permits, these may be aided by movements against resistance.

Secondly, deformities and contractures of the paralysed parts should be prevented by passive movements at the joints, as already mentioned, and by the aid of carefully adjusted splints, and apparatus designed to aid the action of paralysed muscles and to prevent the over-action of the non-paralysed.

Thirdly, care should be taken to prevent accidental bruises, pressure over bony prominences, and the contact of substances which may irritate the skin.

Fourthly, suture of the injured nerve should be undertaken in suitable cases, and treatment on the above-mentioned lines carried out until the return of the sensory and motor functions renders it unnecessary.

In cases of slow compression, as in cervical rib, the treatment consists of removal of the cause.

Lesions of Special Nerves

The Phrenic Nerve

The phrenic nerve arises from the third, fourth, and fifth cervical nerves and supplies the diaphragm.

The phrenic nerve may be paralysed by lesions of the third and fourth cervical segments and by implication of the third and fourth cervical nerve roots from trauma, hemorrhage, syphilitic meningitis, tumours, tuberculous and malignant

Fig. 29.—Shows the winging of the scapula in a case of paralysis of the right serratus magnus muscle. Both arms are extended horizontally in front of the patient.

disease of the vertebræ. It may be involved in its course through injury, or from tumours situated in the neck or thorax, but such causes are rare. It may also be implicated in neuritis, especially in the post-diphtheritic, alcoholic, and toxic varieties, but rarely in lead neuritis.

The resulting paralysis is shown by an absence of descent of the diaphragm on inspiration, more particularly on deep inspiratory efforts. If the paralysis is complete, there may be

retraction of the epigastric region and the lower border of the liver. In expiration, a bulging of the hypochondria, protrusion of the stomach, and downward displacement of the liver are observed. Even slight exertion may be accompanied by considerable breathlessness and distress. Expectoration, coughing, and defæcation may also be hindered. Unilateral paralysis gives rise to little discomfort.

Prognosis. This is largely dependent upon the cause of the paralysis. Its occurrence as a complication is serious in multiple neuritis, but not necessarily fatal; and in the non-fatal cases, complete recovery usually occurs.

The Long Thoracic Nerve

This nerve arises from the fourth, fifth, and sixth cervical nerves, and supplies the serratus magnus muscle.

It is paralysed alone as a result of traumatism, common causes of which are injuries to the shoulder and wounds in the axilla and supra-scapular region, from carrying weights upon the shoulder and from over-exertion, especially of elevation of the arm. It is also paralysed in acute infective disorders. Its palsy is more common on the right side than the left, and more frequent in men than in women.

FIG. 30.—Paralysis of the serratus magnus muscle, viewed from the side.

A characteristic picture is presented by paralysis of the serratus magnus. When the arm is at rest the scapula is higher than normal, and its inner border is approximated to the vertebræ, especially at its lower end, so that it lies

obliquely upwards and outwards. On carrying the arm forwards, the inner border of the scapula wings outwards from the chest. If the arm is abducted into the horizontal position, the shoulder blade approaches the vertebral column, its inner border is raised, and the rhomboids and trapezius muscles are displaced backwards. The arm cannot be raised above the horizontal, as from the lack of scapular rotation the necessary fixation of the shoulder does not take place. If the arm is carried forwards, pressure applied on it in a backward direction causes displacement of the shoulder blade. (Figs. 29 and 30.)

The paralysis is accompanied by pain in the neck and shoulder, but no objective sensory loss is present.

The prognosis is on the whole good, but recovery may be delayed for many months.

Chapter II

PARALYSES OF THE UPPER LIMB

Paralysis of the nerves of the upper limb may be described according as the lesion involves:—

1. The nerve roots.
2. The brachial plexus.
3. The individual nerves.

1. Lesions of the Roots of the Brachial Plexus

The *motor* symptoms of lesion of the nerve roots of the upper limb correspond to those following disease or destruction of the related segments of the spinal cord, and are recorded on a subsequent page (p. 317).

The *sensory* loss covers a definite area of skin over the hand, forearm, and upper arm, and has been accurately delimitated after division of the posterior roots of the brachial plexus.

The accompanying chart (fig. 31) shows the distribution of the epicritic and protopathic loss following division of the posterior roots from the sixth cervical to the second dorsal inclusive. It will be observed that the area of protopathic is larger than that of epicritic loss.

2. Lesions of the Brachial Plexus

The brachial plexus is formed by the anterior primary divisions of the five nerves from the fifth cervical to the first dorsal inclusive. The fifth and sixth nerves constitute the upper trunk, the seventh the middle trunk, and the eighth cervical and first dorsal the lower trunk of the plexus. (Fig. 32.)

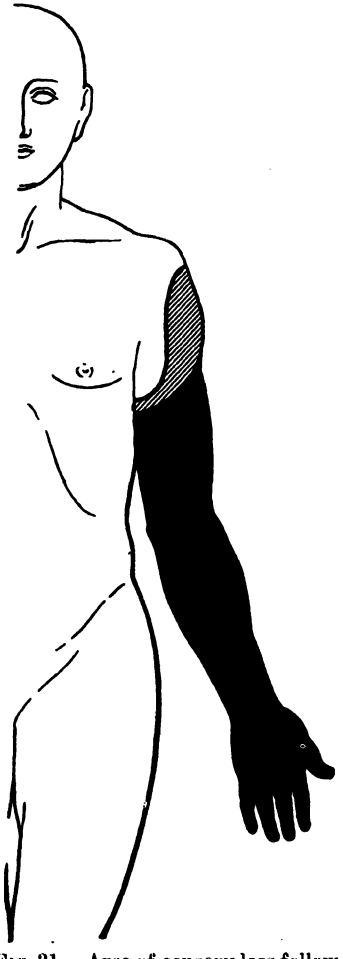


Fig. 31.—Area of sensory loss following section of the posterior nerve roots from the sixth cervical to the second dorsal inclusive. The black area corresponds to the zone of protopathic, epicritic, and deep sensory loss; the shaded represents the overlap of protopathic loss.

Etiology. The chief cause of lesion of the brachial plexus is undue violence or traction applied to the arm, neck, or shoulder. Of this nature are the obstetrical paralyses, arising from traction upon the arm during the process of delivery, paralyses arising from traction upon the arms in connexion with injuries about the shoulder joint, and paralyses following operations upon the breast and axilla, when the arm is forcibly abducted and maintained in an unnatural position. Gunshot wounds or other accidental injuries of the plexus are rare. The plexus may also be implicated in dislocations of the humerus, and by tumours or aneurismal dilatation in the posterior triangle of the neck. A toxic or infective neuritis of the plexus, though rare, has also been described.

Traction injuries of the plexus are due to tearing of the nerve sheaths, sometimes accompanied by hemorrhage within the sheath, and in severe

instances to an actual rupture of the nerve fibres. In less severe cases, however, the lesion would appear to consist merely of over-stretching of the nerves without any serious solution of their continuity.

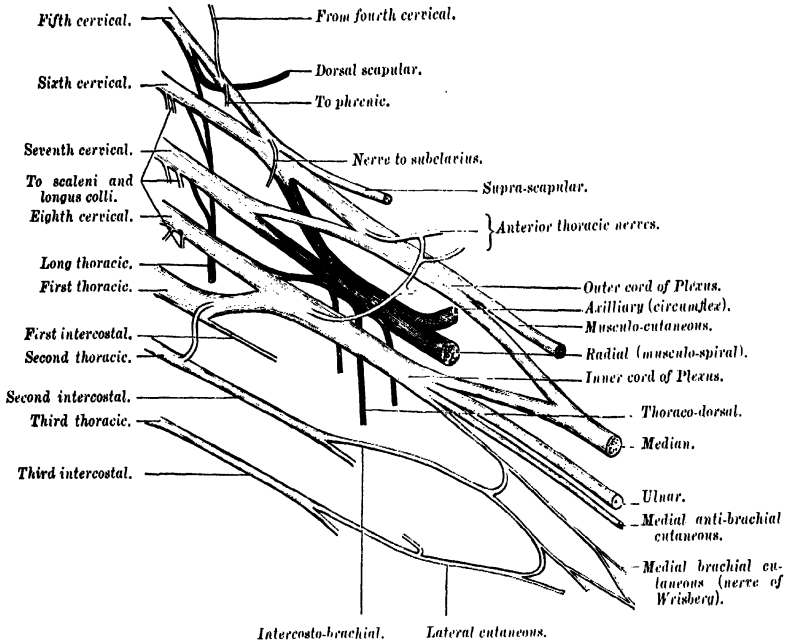


FIG. 32 (from Morris's 'Anatomy').—Diagram of a common form of brachial plexus. The posterior cord of the plexus is darkly shaded.

In obstetrical paralysis, and in paralysis resulting from malposition of the arm during operations, the upper trunk of the plexus is more commonly the seat of lesion. On the other hand, the lower trunks receive the chief brunt of the traction when the arm is outstretched, as in trying to save the body when falling from a height.

Symptoms. The symptoms of lesion of the brachial plexus may be described according as they are due to interference with:—

(1) The whole plexus.
(2) The upper trunk.
(3) The middle trunk.
(4) The lower trunk.
(5) The cords of the plexus.

(1) The whole brachial plexus

In lesions of the whole brachial plexus the entire musculature of the arm is paralysed, with the exception of the serratus magnus, rhomboids, and levator anguli scapulæ. In most cases the pectoral muscles are also paralysed. The

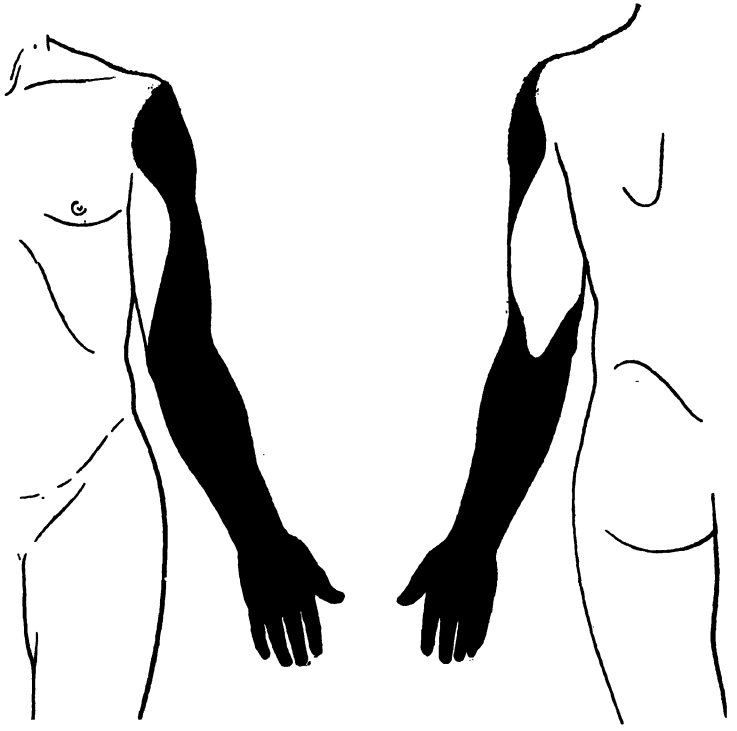


FIG. 33.—The distribution of the sensory loss after lesion of the brachial plexus (Head).

paralysis rarely remains total, and the residual paralysis presents the features of either the upper or lower arm type.

According to Sherren the *sensory symptoms* consist of epicritic and protopathic loss over the whole of the forearm and hand, and the outer surface of the upper arm in its lower two-thirds. The tip of the shoulder and the inner surface of the upper arm are sensitive, owing to the intact condition of

Sensation, both the epicritic and protopathic varieties, is impaired along the ulnar border of the hand and forearm.

In some cases an associated palsy of the sympathetic fibres is demonstrable. When this occurs, the nerve roots have probably been torn or otherwise injured near the spinal cord.

Cervical ribs. The symptoms occasioned by the presence

FIG. 35.—Showing the atrophy of the hand and forearm in a case of cervical rib on the right side.

of a supernumerary or cervical rib may be explained as the result of pressure upon the lower trunk of the plexus. In this condition the motor symptoms consist mainly of paralysis and atrophy of the intrinsic muscles of the hand, with an anæsthetic area along the ulnar border of the forearm and hand. Subjective sensations of pain, often of a severe character, with numbness and tingling, are also described. The absence of symptoms pointing to lesion of the sympathetic fibres (narrowing of the palpebral fissure and constriction of

the pupil) would appear to place the lesion in the lower trunk rather than in the roots of the plexus. (Fig. 35.)

The condition is more commonly met with clinically in women than in men, and more often affects the right side. The symptoms do not usually appear until early adult life.

The diagnosis is rendered especially easy by the use of the Röntgen rays, and the treatment consists in removing the exostosis or additional rib.

(5) The cords of the brachial plexus

Each trunk of the brachial plexus divides into an anterior and a posterior branch. All the posterior branches unite to form the *posterior cord*, the anterior branches of the upper and middle trunks unite to form the *outer cord*, while the anterior branch of the lower trunk forms the *inner cord* (see fig. 32). In this way an important rearrangement of the nerve fibres in the trunks of the plexus is brought about.

The *outer cord* of the plexus contains the fibres of the upper trunk, with the exception of those for the supra- and infra-spinatus muscles. It receives from the middle trunk the nerve fibres for the pronators and long flexors of the wrist and fingers. It eventually gives origin to the external anterior thoracic nerve, the musculo-cutaneous nerve, the nerve to the coraco-brachialis, and the outer head of the median. The *symptoms* of lesion of the outer cord, therefore, consist of paralysis of the pectoralis major, the biceps, brachialis anticus (partly), the coraco-brachialis, the pronator teres, flexor carpi radialis, and flexor sublimis digitorum. Sensation is impaired over an area on the outer surface of the forearm, whose anterior boundary is well defined, but whose posterior boundary fades into the area supplied by the descending branch of the musculo-spiral nerve (Sherren).

The *posterior cord* of the plexus, in addition to containing some of the nerve fibres of the middle trunk, receives fibres from the upper trunk for the deltoid, teres minor, and sub-scapularis muscles, and also some from the lower trunk. It gives origin to the subscapular, circumflex, and musculo-spiral nerves. The *symptoms* of lesion of the posterior cord are as follows: paralysis of the subscápularis, of the deltoid and teres minor, of the triceps, brachialis anticus

(partly), supinators, long and short extensors of the wrist and fingers, and the extensors of the thumb and index finger.

The skin areas affected correspond to the cutaneous distribution of the circumflex and musculo-spiral nerves elsewhere described.

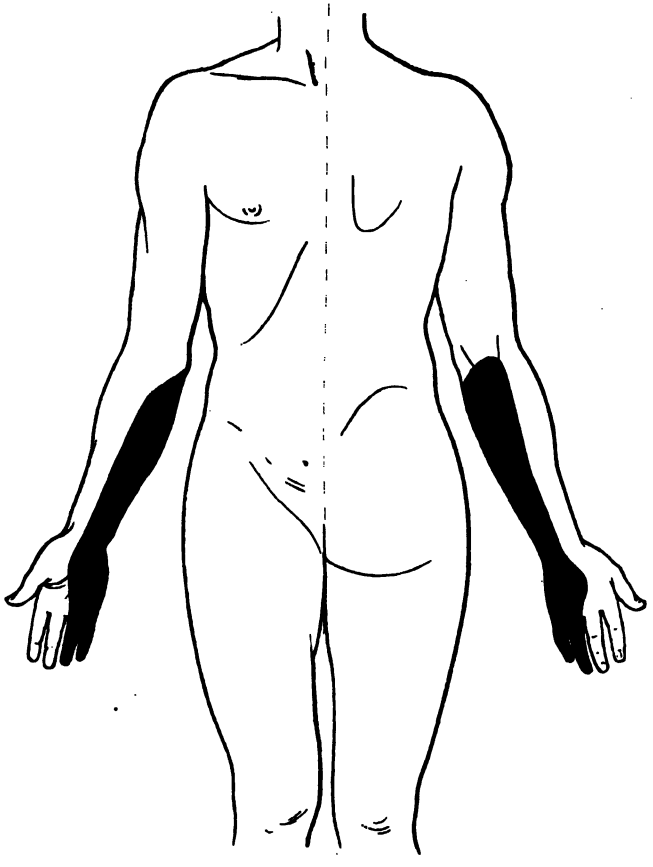


FIG. 36.—The area of sensory loss following lesion of the inner cord of the brachial plexus (Head). The front of the hand and forearm is shown on the left, the back on the right side of the figure.

The *inner cord* of the plexus contains the remaining nerve fibres of the lower trunk. It gives origin to the internal anterior thoracic, the internal cutaneous, the inner head of the median and the ulnar nerves. A lesion is characterised by paralysis of the pectoralis major, flexor profundus digitorum, flexor longus pollicis, flexor carpi ulnaris, the lumbricales and

interossei, and the special muscles of the thumb and little finger. The sensory loss corresponds to the area of ulnar anæsthesia upon the inner side of the forearm and hand. (Fig. 36.)

The Circumflex Nerve

This nerve arises from the posterior cord of the brachial plexus, and supplies the deltoid and teres minor muscles; it also sends branches to the shoulder joint.

The commonest cause of paralysis is a fall on or injury to the shoulder, especially with dislocation of the shoulder joint. Amongst general causes, lead poisoning and diabetes may induce an isolated palsy. This may also occur in typhoid fever.

As the muscles are primarily concerned in elevation and outward rotation of the upper arm, these movements are paralysed in lesions of the circumflex. There is an area of epicritic and protopathic loss over the insertion of the deltoid, and the upper half of the outer side of the arm. Arthritic changes in the shoulder joint are sometimes found.

It is commonly affected along with the *supra-scapular nerve*, which arises from the upper trunk of the plexus, and is distributed to the supra- and infra-spinatus muscles. Its skin area corresponds to the outer border of the scapula, contiguous with that of the circumflex.

The Musculo-Spiral Nerve

This nerve arises from the posterior cord of the brachial plexus and supplies the following muscles: triceps, brachialis anticus, supinator longus, extensor carpi radialis and ulnaris, extensor communis digitorum, and the extensors of the thumb and little finger. Through its radial and external cutaneous branches it supplies the skin on the radial side of the back of the wrist, thumb, and hand.

Etiology. Paralysis of the musculo-spiral nerve is an important feature in lesion of the posterior cord of the brachial plexus (p. 111), in which case the circumflex and subscapular nerves are also involved.

Isolated paralysis of the nerve is common. It may be implicated in the upper part of the arm by the pressure of

a crutch, by traction on the arm and in the 'sleep palsies' following alcoholic intoxication. In these cases the lesion is above the origin of the nerve to the triceps muscle. Below the origin of this nerve the chief causes of palsy are fractures in the lower third of the arm, gunshot or other wounds, dislocation of the head of the radius and operations upon the arm.

The nerve may also be paralysed from infective causes during the course of enteric fever, the puerperium and articular

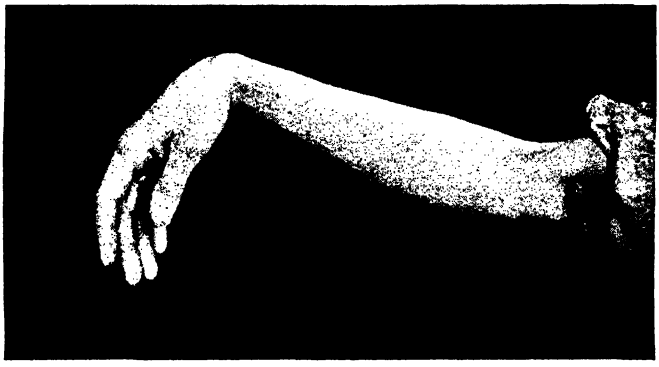


FIG. 37.—Showing drop wrist in a case of musculo-spiral paralysis. The prominence on the dorsum of the wrists is well seen in the photograph.

rheumatism. Paralysis of the nerve also forms an element in the multiple peripheral paralyses of lead, arsenic, and alcoholic neuritis. In the case of lead palsy the supinator longus muscle is rarely affected—a point of some importance in differential diagnosis.

Symptoms. The *motor* symptoms are seen in wrist and fingerdrop, and in the event of the lesion being above the origin of the nerve to the triceps, in inability to extend the forearm at the elbow. The appearance of the hand and forearm is characteristic. The patient is unable to extend the wrist or the fingers. This is especially observed in the two ulnar fingers, and least in the index finger. Attempts at wrist extension produce some degree of flexion, owing to the unopposed action of the synergic flexor muscles. There is often a prominence at the back of the wrist from a partial dislocation of the carpus, due to the weakness of the extensors and the stretching of the dorsal ligaments. The

supinator longus is always paralysed, and the triceps also, if the lesion is high up in the upper arm. (Figs. 37 and 38.)

The *sensory* symptoms depend upon the position of the lesion. For the purpose of studying the sensory loss the nerve may be divided into three parts:—

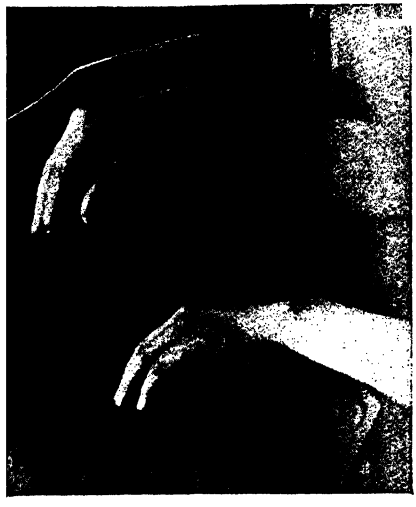


FIG. 38.—Bilateral drop wrist due to pressure on both musculo-spiral nerves from the use of crutches.

(*a*) Lesion above the origin of the external cutaneous nerve. When the lesion is in the upper arm, epicritic, protopathic, and deep sensibilities are affected over a skin area on the dorsum of the hand corresponding to the two radial fingers, and upon the back and outer side of the thumb. (Fig. 39.) The area of epicritic is more extensive than that of protopathic loss.

(*b*) In lesion below the origin of the external cutaneous branch (paralysis of the radial nerve) either no sensory impairment is detected, or only epicritic loss upon the outer side of the thumb.

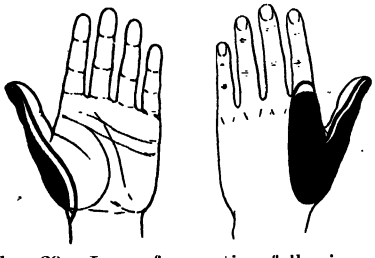


FIG. 39.—Loss of sensation following a lesion of the radial and external cutaneous branches of the musculo-spiral nerve. The total area of loss is contained within the continuous thick line. The black is the area of protopathic and epicritic loss, the zone between that and the continuous line is the area of epicritic overlap. (After Head.)

(*c*) On the other hand, lesion of the external cutaneous and radial branches is followed by an epicritic and protopathic loss corresponding to that described after lesion of the nerve trunk (Head), but deep sensibility is not affected.

The conclusions to be drawn from these observations are, that the external cutaneous nerve and its branches are the

main paths for both epicritic and protopathic sensibility to the skin of the hand supplied by the musculo-spiral nerve; that deep sensibility is transmitted by the posterior interosseous nerve; and that the radial nerve only conveys impressions of epicritic sensibility.

The **prognosis** is favourable for recovery in most cases. In the simple pressure cases, with little or no electrical alterations, recovery is rapid. If the reaction of degeneration is present, recovery may be delayed for many months. In gunshot wounds, or other solutions of the anatomical continuity of the nerve fibres, resection of the ends of the nerve and suture may be necessary, in which event cure may not ensue until after a year, even in favourable cases; while in cases complicated with much wasting and degenerative changes, complete recovery may never take place.

The **treatment** is based on the general indications given on p. 101. A glove, designed to support the wrist and keep both it and the fingers extended, greatly hastens recovery, and enables the patient to make use of the hand in grasping objects.

The Median Nerve

The median nerve arises by two heads derived from the outer and inner cords of the brachial plexus. It supplies the following muscles: flexor profundus digitorum, flexor longus pollicis, flexor carpi radialis, flexor sublimis digitorum, the pronators, the two outer lumbricals, the abductor and opponens pollicis, and the short head of the flexor brevis pollicis.

Etiology. The median nerve may be paralysed from accidental wounds in the neighbourhood of the wrist joint, but is also involved in other parts of its course by fractures in the upper arm or forearm. It may be paralysed in carpenters and professional golfers as a form of occupation or craft palsy.

Symptoms. The symptoms depend upon the position of the lesion whether:—

(*a*) At or above the elbow.

(*b*) At or about the wrist.

(*a*) In lesion of the nerve at or above the elbow joint, the motor loss is chiefly seen in weakness and immobility of the thumb and index finger. There is inability to flex the

terminal phalanx of the thumb, or to flex the index finger except at the metacarpo-phalangeal joint. Pronation is feeble.

Epicritic loss corresponds to an area bounded on the palmar surface of the hand by a line drawn through the axis of the ring finger, and extending to the limit of musculo-spiral loss along the palmar axis of the thumb. The line then passes along the free margin of the first interosseous space, and up the index finger until it reaches the first interphalangeal joint, when it passes across the middle finger and half the ring finger to the boundary of ulnar anæsthesia in the axis of the ring finger. (Fig. 40.)

The protopathic loss varies from a small area over the tips of the fingers to one almost coextensive with the epicritic anæsthesia. Deep sensibility is retained.

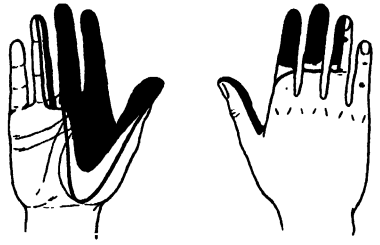


FIG. 40.—Shows the area of sensory loss following lesion of the median nerve. The shading is the same as in fig. 39. (After Head.)

(*b*) In lesion at the wrist joint, the motor symptoms are limited to palsy of the opponens and abductor actions of the thumb and of the two outer lumbrical muscles. The sensory phenomena correspond to those of lesion at or above the elbow joint.

Minor injuries of the median nerve are apt to be overlooked owing to the absence of motor deformity, the small protopathic loss and the retention of deep sensibility (Sherren).

The **prognosis** and **treatment** are based upon the general considerations already laid down on pp. 100 and 101.

THE ULNAR NERVE

This nerve arises from the inner cord of the brachial plexus, and supplies the following muscles: flexor carpi ulnaris, ulnar half of the flexor profundus digitorum, interossei, two inner lumbricals, adductor and half the flexor brevis pollicis and the muscles of the hypothenar eminence.

Etiology. Ulnar paralysis is not uncommon, and is due to injuries and wounds about the elbow, in the forearm, and at the wrist. It may occur as an occupation paralysis in glass-workers and cigarette-makers, and it is found in rheumatic and gouty subjects in consequence of a local neuritis.

Symptoms. These may be described according as the lesion is situated: —

(*a*) In the neighbourhood of the elbow joint.

(*b*) At the wrist joint below the origin of the dorsal cutaneous branch.

FIG. 41.— Showing the appearance of the hand after injury to the ulnar nerve.

(*a*) Lesion at the elbow joint. If the lesion is above the origin of the muscular branches to the forearm, palsy of the flexor carpi ulnaris and the ulnar half of the flexor profundus digitorum is observed in addition to paralysis of the hand. This is due to palsy of the interossei, the two inner lumbricals, and the adductor of the thumb. The appearance of the hand is characteristic. The little and ring fingers are hyper-extended at the metacarpo-phalangeal joints; all the fingers, but especially the little and ring fingers, are flexed at the inter-phalangeal joints. Abductor and adductor action of the fingers is lost, adduction of the thumb is also in abeyance, although a false adduction is possible by aid of the flexor longus pollicis and the extensors of the thumb (Sherren). The interosseus spaces are well defined owing to wasting of the interossei muscles. The hand may also be tilted somewhat to the radial side. (Fig. 41.)

The *sensory* symptoms consist of epicritic loss over the little and ulnar half of the ring finger and a corresponding area over the palm and back of the hand, as far as the wrist. The epicritic loss is in excess of the protopathic, which is variable. Loss of deep sensibility corresponds to the area of protopathic anæsthesia. (Figs. 42 and 43.)

(*b*) Lesion at the wrist below the origin of the dorsal cutaneous branch. The motor symptoms are seen in palsy of all the intrinsic muscles of the hand supplied by the ulnar nerve. The area of epicritic loss corresponds to that seen

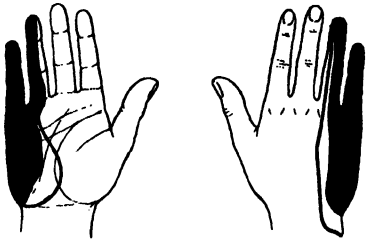


FIG. 42.—Loss of sensation following a lesion of the ulnar nerve. The total area is contained within the continuous black line. The shading is the same as in fig. 39. (After Head.)

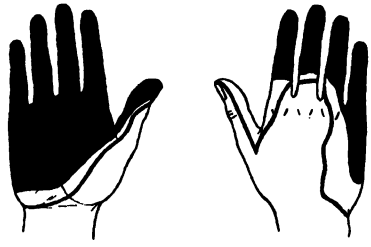


FIG. 43.—Showing the loss of sensation following lesion of the ulnar and median nerves. (After Head.)

after lesion of the whole nerve. The area of protopathic loss varies, but is usually less extensive than that seen after lesion of the nerve higher up, and deep sensibility is retained.

The **prognosis** and the **treatment** are referred to in detail on pp. 100 and 101.

CHAPTER III

PARALYSES OF THE LOWER LIMB

ROOT LESIONS—PARALYSIS OF THE CAUDA EQUINA

The cauda equina is formed by the nerve roots of the lumbo-sacral segments of the cord. As in the adult the spinal cord ends at the level of the first lumbar vertebra, the nerve roots have to pass for a considerable distance within the neural canal before they reach their points of exit at the corresponding intervertebral foramina. As each pair of foramina is reached, the corresponding nerve roots separate from the cauda equina and pass out of the neural canal. Thus the upper lumbar roots are situated peripherally and run a relatively short course within the canal, while the

sacral roots are placed more mesially and have a longer course.

The conus terminalis, or extreme lower end of the spinal cord, is defined anatomically as that portion of the cord which lies below the level of the second sacral segment. As the conus is surrounded by the nerve roots from the second lumbar segment downwards, it follows that lesions of the upper part of the cauda equina may also involve the conus terminalis. It does not contain any pyramidal fibres below the level of the fourth sacral segment, but contains the centres for erection of the penis, and the anal and vesical sphincters, although, according to Müller,[1] the reflex centres for micturition and defæcation are found in the pelvic sympathetic ganglia.

Etiology. The nerve roots forming the cauda equina may be implicated by traumatic lesions of the spine, by an extension of malignant or tuberculous disease from the bones of the lumbar and sacral regions, by meningitis—especially the gummatous variety—and by new growths, mainly sarcomata, neurofibromata, endotheliomata, and angeiomata.

Symptoms. The symptoms of lesion of the cauda equina vary in their mode of onset and course according to the character and situation of the lesion.

In traumatic lesions the onset of paralysis may be immediate owing to direct injury to the roots, or may follow shortly after from secondary effects, such as hemorrhage or suppuration. In these cases the symptoms are referred to the motor, sensory, and sphincter functions.

Where the lesion is due to chronic meningitis or new growth, the symptoms are of gradual onset, at first limited to certain nerve-root areas and often affecting the motor, sensory, or sphincter functions separately. The situation of the lesion determines the extent and the area of the paralysis. If situated at the lower end, sphincter paralysis and sensory changes in the perinæal region are alone observed. If situated higher up and peripherally, motor, sensory, or motor and sensory paralyses in the distribution of the affected root areas will be found without any sphincter weakness. Should the lesion be situated high up and central in position, severe paralysis, motor and sensory, will be present in the distribution of the sacral and lower lumbar roots, with complete sphincter

[1] Müller, *Deutsch. Zeitsch. f. Nervenheilk.* 1901

paralysis. The motor symptoms are: atrophic flaccid paralysis with abolition of the deep reflexes and the reaction of degeneration in the muscles supplied by the affected roots. The sensory symptoms are: subjective numbness, tingling and pain referred to the sensory areas supplied by the affected roots. The sensory loss develops later and is of the peripheral type—a point of the utmost importance in the differential diagnosis of cauda equina from intramedullary lesions. In

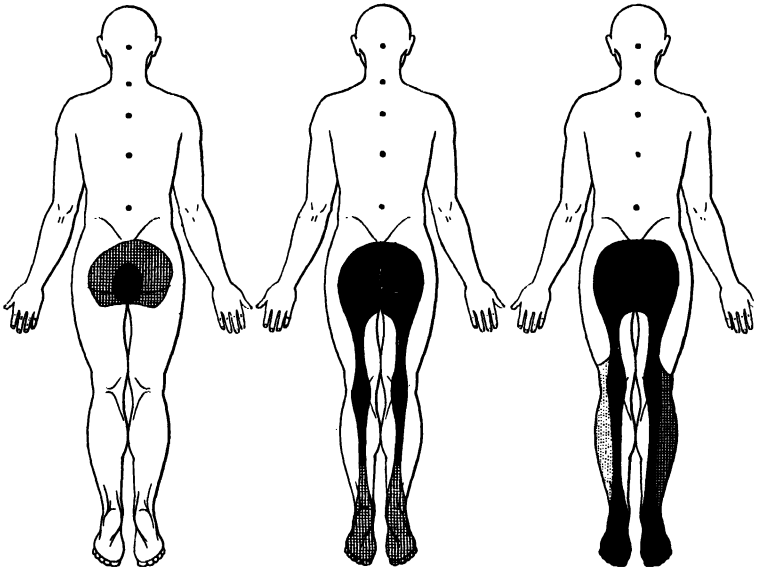


FIG. 44.—The three figures represent, from left to right, the successive stages of involvement of the roots of the cauda equina in a case of sarcoma. The black areas are those of complete sensory loss, the shaded those of incomplete loss. Compare with fig. 91.

root lesions the protopathic and epicritic areas coincide, the former sometimes overlapping the latter. The sphincter symptoms are slight impairment of vesical control and constipation if the sacral roots on one side only are affected; if the lesion is bilateral, complete loss of both the vesical and anal sphincters is present, with patulous anus and loss of sensation. The sensibility of the genitalia is abolished and erection is impossible.

Two distinct types are met with; the first, where the lesion

is mesial and low down, and the second where it is peripheral and towards the upper part of the cauda.

1. *Symptoms of a lesion situated mesially and low down in the cauda equina.*

The earliest symptoms are referred to the lowest sacral roots, objective sensory loss being detected around the anus, upon the genital organs and over the buttocks. This is at first limited to, or is preponderatingly on one side, but eventually extends so as to affect both sides. The earliest bladder and rectal symptoms consist mainly of a loss of voluntary control, whereby a sudden evacuation of either the bladder or the rectum may occur. A loss of sexual power and of erection are coexistent conditions. When the lesion is definitely bilateral, more pronounced vesical and rectal symptoms develop. These consist of a dribbling of urine and incontinence of fæces from paralysis of the sphincters, the patient being unaware of the passage of the excreta.

FIG. 45.—Photograph showing the escape of the 5th, 4th, 3rd, and 2nd sacral areas in a case of lesion of the 12th dorsal to the 1st sacral roots on the right side.

The extension of the lesion is noted by a progressive implication of the third, second, and first sacral, and the fifth and fourth lumbar roots, with corresponding sensory changes over these root areas; motor paralysis with atrophy of muscles and loss of the reflexes—first the plantar, then the ankle jerk, and later the knee jerk. (Figs. 44 and 45.)

In this type therefore the vesical and genital symptoms are early, the motor late in appearing.

2. *The symptoms of a lesion situated peripherally and high up in the cauda equina.*

This type is usually seen in association with meningeal growths and tumours.

Pain is commonly an early symptom and is referred to the third, fourth, and fifth lumbar or the upper sacral roots. Pain remains unilateral for a considerable time; but eventually objective sensory loss is detected and is soon followed by motor paralysis, while the bladder, rectal and generative functions remain intact. Thus, in one case of gummatous meningitis the earliest symptoms were unilateral loss of sensation over the third, fourth, and fifth lumbar areas with weakness and atrophy of the corresponding muscles, loss of the knee jerk with preservation of the ankle jerk and flexor plantar response, and intact bladder and rectal functions. As the disease extended, the first, second, and third sacral areas became involved with loss of the ankle jerk and plantar reflex, and symptoms of involvement of the third and fourth lumbar roots upon the opposite side. Eventually the whole of the cauda equina on both sides became affected, sphincter trouble, bedsores, and death eventually supervening.

For the motor and sensory localisation of the several root lesions, the reader is referred to the Table on p. 317 and figs. 90 and 91.

The **diagnosis** of lesions of the cauda equina has to be made from several conditions.

1. *Lesions of the conus medullaris* may be either primary, or occur in association with lesion of the cauda equina. The symptoms which characterise a lesion limited to the conus are, loss of control over the bladder and rectum—dribbling of urine, and either constipation or involuntary evacuation of the rectum; loss of sensation (total or dissociated) over the ano-perinæal region and genitalia—testicular tenderness being preserved; loss of voluntary or reflex sexual action; and anæsthesia over the areas on the buttocks supplied by the third and fourth sacral roots. There is no motor disability, and the deep and superficial reflexes are normal. The onset and course of the symptoms are more rapid than in lesions of the cauda and are more often bilateral. The dissociation of sensation is often striking, and may serve to distinguish a

lesion of the conus from one situated mesially and low down in the cauda.

If the lesion in the conus extends upwards so as to involve the first and second sacral segments, loss of sensation over the soles of the feet and over a strip of skin up the centre of the posterior surface of the lower limbs is found. This is associated with paralysis of the glutei, triceps, semi-membranosus, tibialis posticus and calf muscles, and the intrinsic muscles of the feet. The plantar reflexes and ankle jerks are impaired or abolished.

2. *A lesion of the spinal cord at the level of the second and third lumbar segments* may resemble one of the cauda equina. The differentiation is difficult only if the lesion is total, when there would be found complete atrophic flaccid paralysis of the lower limbs, complete loss of all forms of sensation, abolition of the reflexes and total sphincter paralysis. This association of symptoms could only be attributed to the cauda equina as a result of a traumatic lesion. In the absence of this history it would have to be ascribed to a total transverse lesion of the cord.

3. *Malignant and tuberculous disease of the sacrum* may give rise to symptoms of lesions of the cauda equina. These diseases are of slow and gradual onset, with ill-defined though sometimes sharp and shooting pains in the sacral region. Pain is usually present, often more intense in the recumbent posture.

The nerve symptoms depend upon the implication of the nerve roots, which may be individually picked out by the progressive extension of the malady. The local objective signs are slight in contrast to the severity of the subjective symptoms and the obvious cachectic condition of the patient. Local prominence and tenderness on pressure over the sacrum should be looked for, and in all cases a careful rectal and sigmoidoscopic examination should be made. The Röntgen rays may also be used with advantage in solving the problem of the diagnosis, which is often for a time obscure.

Lesions of the Lumbo-Sacral Plexus

The lumbo-sacral plexus is a complex arrangement of nerves, and may be divided into three sub-systems: the lumbar,

the sacral or sciatic, and the pudendal plexuses. The lumbar plexus is formed by the anterior branches of the nerves issuing from the first to the fourth lumbar segments, the sacral or sciatic plexus by those from the fifth lumbar to the

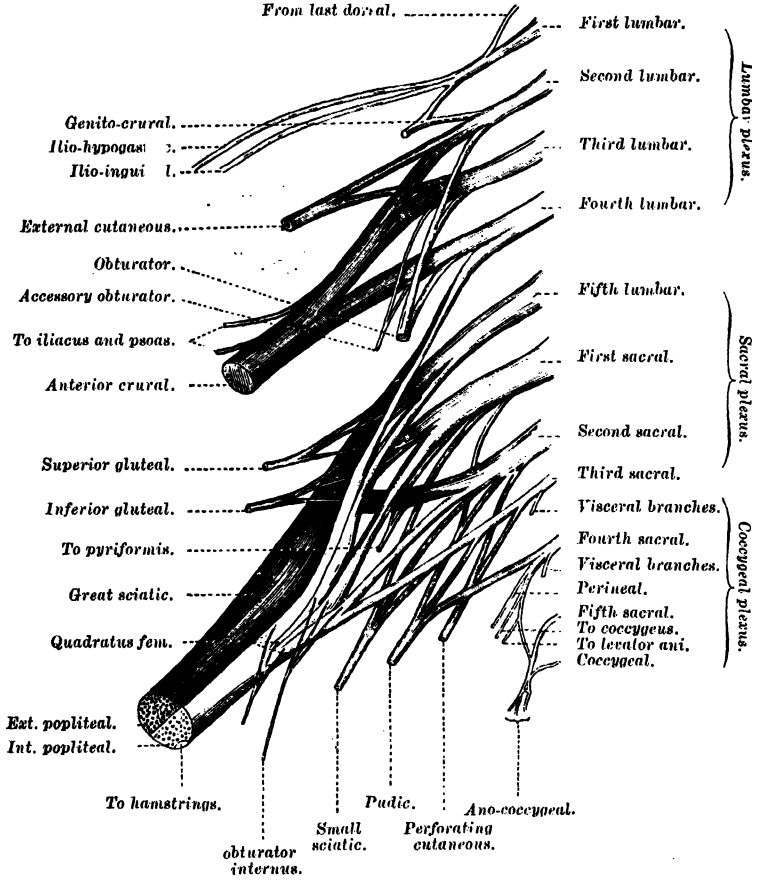


FIG. 46.—Diagram of a common form of lumbo-sacral plexus. (From Morris's 'Anatomy.')

third sacral, and the pudendal plexus by those from the fourth and fifth sacral and coccygeal segments. (Fig. 46.)

The *causes* of paralysis of the nerves of the lower limb are found in connexion with disease of the bones of the

lumbar and sacral vertebræ, and of the pelvic organs. These nerves are also liable to injury from the use of the forceps during delivery.

The great sciatic nerve is more often wounded in gunshot injury than any other nerve of the body. It may also be paralysed in the manipulations of reducing congenital dislocation of the hip.

Various toxic conditions may also lead to paralysis of the nerves of the lower limb. Their influence is seen in the alcoholic and arsenical forms of peripheral neuritis. Puerperal neuritis and neuritis following upon septic conditions within the pelvis have also been observed. Paralysis of the external popliteal nerve is an early symptom of diabetic neuritis.

It is a well-known fact that the peroneal, or external popliteal nerve is more liable to paralysis than the internal branch of the great sciatic.

The lumbar plexus

The first lumbar nerve gives origin to the ilio-hypogastric and ilio-inguinal nerves, and partly to the genito-crural. These nerves are mainly sensory, but give motor filaments to the recti, the obliqui abdominales, and the cremaster muscles.

The chief motor branches of the lumbar plexus arise from the second, third, and fourth lumbar nerves. They are the anterior crural, obturator, and external cutaneous nerves.

The anterior crural nerve

The anterior crural nerve supplies the quadriceps extensor, the pectineus in part, and the sartorius muscles. Common causes of paralysis of this nerve are tumours of the pelvis and psoas abscess; a primary neuritis may sometimes affect it. Its paralysis is characterised by inability to extend the knee joint, and by abolition of the knee jerk. The quadriceps muscle is wasted.

There is loss of epicritic and protopathic sensation over an area situated on the inner aspect of the leg extending from the inner and upper border of the plantar arch, over the internal malleolus to more than halfway up the leg. Its

upper border becomes less distinct and merges into a large area over the front and inner aspect of the thigh, in which there is no complete loss of sensibility (Sherren). (Fig. 47.)

The obturator nerve

The obturator nerve supplies the adductors of the thigh, the obturator externus, the pectineus (partly), and the gracilis muscles. Hence its paralysis impairs the approximation of the thighs, outward rotation of the hip joint, and the movement of crossing the legs. There is wasting on the inner aspect of the thigh. It may be injured in prolonged instrumental delivery.

There is no definite sensory loss.

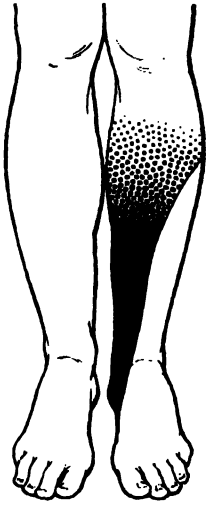


Fig. 47.—The area of sensory loss from a lesion of the anterior crural and its internal saphenous branch. (After Head.)

The external cutaneous nerve

The external cutaneous is a sensory nerve, and supplies the skin over the outer aspect of the thigh with epicritic and protopathic sensation.

A condition—*meralgia paræsthetica*—characterised by paræsthesia, and accompanied by more or less objective disturbance of the sensibilities of touch, pain, and temperature, has been described over the distribution of the nerve on the outer side of the thigh. There may be tenderness on pressure over the nerve at the point where it issues from the deep fascia. In a case of this affection E. Bramwell[1] found no sign of neuritis in a portion of the nerve which had been excised.

The Sacral Plexus

The sacral or sciatic plexus is formed by the nerves from the fifth lumbar to the third sacral, including a branch from the fourth lumbar nerve. It gives origin to one large

[1] Bramwell (Edwin), *Edin. Med. Journal*, 1903.

composite nerve trunk—the great sciatic nerve. This is formed by two separate and distinct nerves usually contained in one sheath—the internal popliteal or tibial nerve, formed by the anterior branches of the plexus, and the external popliteal or peroneal nerve, formed by the posterior branches.

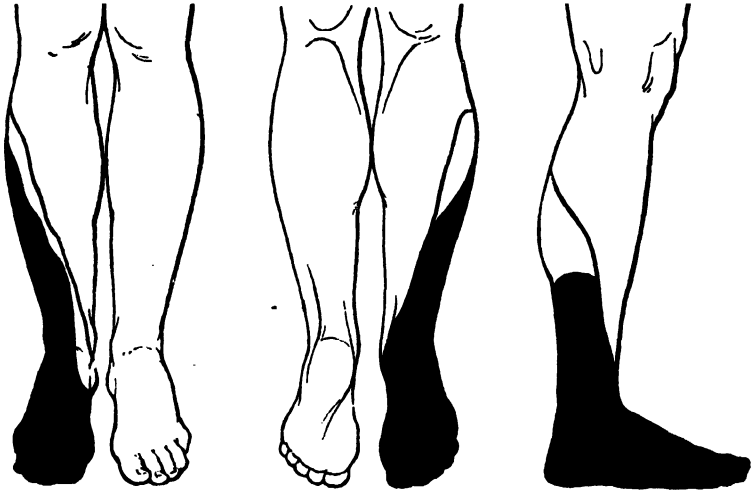


FIG. 48.—Front, back, and side views of the leg showing the distribution of the loss of sensibility following injury to the great sciatic nerve. The black area represents the zone of protopathic and epicritic loss, the continuous line the extent of the epicritic overlap. (After Head.)

In addition to the great sciatic nerve, several subsidiary nerves are described as arising from the sciatic plexus: (*a*) the nerve to the tensor fasciæ femoris from the fourth lumbar; (*b*) the superior gluteal nerve to the gluteus medius and minimus, from the fourth and fifth lumbar and first sacral; (*c*) the inferior gluteal nerve to the gluteus maximus, from the fifth lumbar and first and second sacral; (*d*) branches from the first three sacral nerves which pass downwards to join the pudendal plexus and form the small sciatic nerve.

The great sciatic nerve

Paralysis of all the muscles supplied by both divisions of the nerve is theoretically possible, but rarely occurs. More commonly there is paralysis of one or other of its branches,

in whole or in part, and almost invariably the external popliteal branch is that which suffers most.

If the lesion is high up in the thigh the motor symptoms consist of paralysis of all the muscles below the knee, the flexors of the knee, and the flexor portion of the adductor magnus. The sensory loss is of wide distribution and consists of epicritic and protopathic anæsthesia below the knee, except for a strip on the inner aspect of the leg, which corresponds to the supply of the internal saphenous nerve. (Fig. 48.)

The **nerve** to the **hamstrings** arises from all the roots forming the tibial, or internal popliteal division of the great sciatic (L 4 and 5, S 1, 2 and 3). Although passing along with the sciatic in the upper part of its course, it is given off high up in the thigh, and thus escapes gunshot and other wounds of the sciatic in this region. The absence of paralysis of the hamstring muscles (biceps, semi-membranosus and semi-tendinosus), with palsy of the tibial nerve, is therefore significant of a lesion of the sciatic in the lower half of the thigh, while a coexistent palsy of the hamstrings, the calf muscles, and the flexors of the toes, points to a lesion either of the sciatic plexus, the cauda equina, or the spinal segments.

The internal popliteal nerve

Lesion of the internal popliteal, or tibial, nerve is followed by paralysis of the muscles of the calf of the leg, the tibialis posticus, and the long flexors of the toes and hallux. Through its internal, or plantar branch—which corresponds to the median nerve in the hand—it supplies the flexor brevis digitorum, the small muscles of the big toe (except the adductor hallucis), and the two inner lumbricals. Through the external plantar branch—which is analogous to the ulnar nerve—it supplies the muscles of the little toe, the two outer lumbricals, the adductor hallucis, and all the interossei.

In paralysis of the nerve the foot and toes cannot be pointed, nor the toes flexed; and inversion of the foot in the extended position is impossible. The foot assumes the position

of talipes calcaneo-valgus. The toes are hyper-extended at the proximal, and flexed at the distal joints.

In severe lesions both epicritic and protopathic sensibilities are lost over the sole of the foot, and along its outer border, as far up as the outer ankle bone. (Fig. 49.)

In less severe cases, epicritic sensibility alone is impaired or lost. In one case of this character the area of epicritic

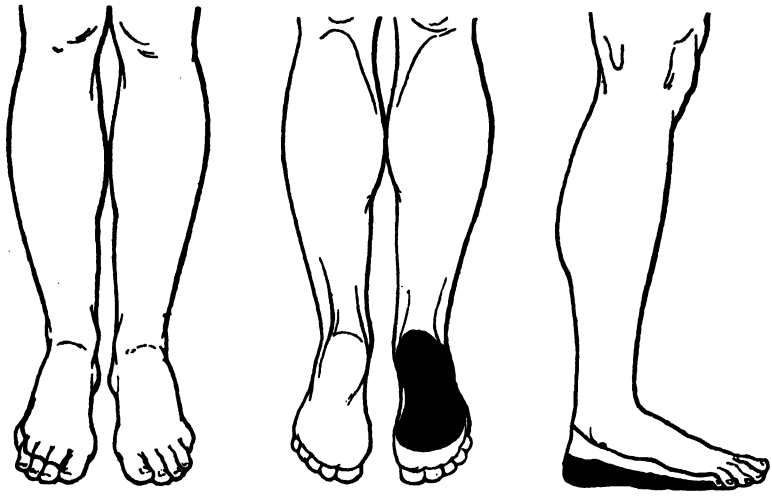


FIG. 49.—Areas of protopathic and epicritic loss from lesion of the posterior tibial nerve. The shading is the same as for fig. 39. (After Head.)

loss covered the sole of the foot and toes, the outer border and part of the dorsum of the foot as far as the external malleolus, and the dorsal aspect of the distal phalanges of the toes.

Deep sensibility is unaffected.

The external popliteal nerve

The external popliteal or peroneal nerve supplies the tibialis anticus, extensor longus digitorum, extensor proprius hallucis, the peroneal muscles, and the extensor brevis digitorum.

In paralysis of the nerve, the foot cannot be dorsiflexed or everted, nor can the toes be extended. The characteristic appearance of drop foot is seen with a tendency towards

talipes equino-varus, and later flexor contracture of the toes. In lesions high up there is loss of epicritic and protopathic sensibility over the outer aspect of the leg from below the knee and extending on the dorsum of the foot to the metatarso-phalangeal joints of the toes. In lesions below the level of origin of the lateral cutaneous branch, the sensory loss is over the dorsum of the foot, and only extends up the outer aspect

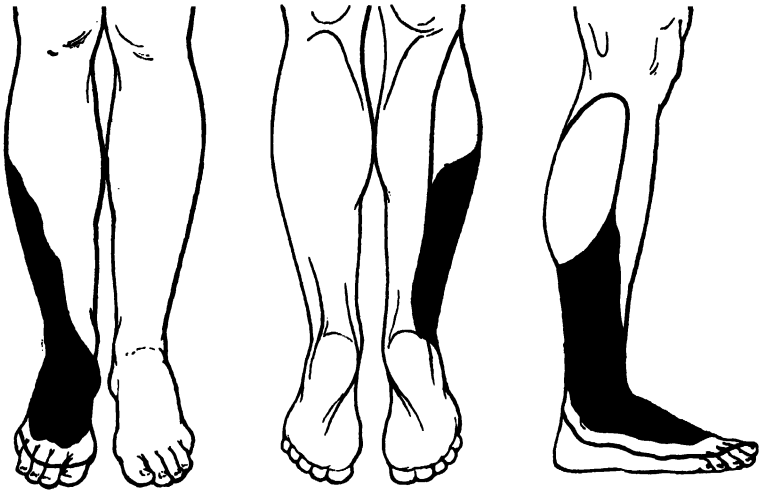


FIG. 50.—Areas of protopathic and epicritic loss after lesion of the external popliteal nerve. The shading is the same as in fig. 39. (After Head.)

of the leg for a few inches above the external malleolus (fig. 50). (Sherren.)

THE PUDENDAL PLEXUS

This plexus is formed partly by the small sciatic nerve, which arises from the first to the third sacral nerves, and partly by the fourth and fifth sacral and the coccygeal nerves. It is distributed to the skin over the back of the thigh and the genital organs. It supplies the perineal muscles, levator ani, external sphincter ani, and erector penis.

Owing to its position in the lower and back part of the pelvis, this plexus is rarely the seat of disease or of traumatism. Paralytic symptoms over its area of distribution are usually due to lesion of the sacral nerve roots or the cauda equina.

Chapter IV

TUMOURS OF NERVES

New growths may involve the roots and the peripheral nerves in any part of their course. They may be single or multiple, benign or malignant. They may be classified as follows:—

1. True neuromata.
2. False neuromata.
   (*a*) Benign: fibromata, myxomata, lipomata, fibro-myxomata, and cystic tumours. Such tumours may be solitary or multiple.
   (*b*) Malignant: sarcomata and carcinomata.
   (*c*) Amputation neuromata.

True neuromata are rare, and are only found in connexion with the sympathetic nervous system—especially in the abdominal plexuses and in the cellular tissues. They may be single or multiple, circumscribed or diffused. They are innocent in character. Their size varies from that of a pea to that of a grape-fruit.

They are often present in children, but may not develop till later, when the sympathetic system is fully formed. The tumours are of a firm or elastic consistence, and are encapsulated. Microscopically they are composed of sympathetic ganglion cells and non-medullated nerve fibres, with an abundant supply of blood-vessels. The nerve cells are round or oval in shape, unipolar with clear or granular protoplasm, and contain a distinct nucleus or nucleolus.

Clinically there are no nervous symptoms, and the tumours are not tender on pressure.

False neuromata. Under this term are classed all the benign and malignant tumours occurring in association with the peripheral nervous system, as well as the bulbous tumours found after injury or division of nerves.

The *benign* tumours are fibromata, myxomata, lipomata, fibro-myxomata, and cystic tumours, resulting from degeneration. They may be single or multiple. The solitary tumours, which have the histological characters of those already mentioned, may occur in any position, either on the roots, trunks,

or subcutaneous filaments of the nerves. The common localities for these tumours are the auditory nerve, where they form one of the varieties of extra-cerebellar growth (p. 267); the fifth cranial nerve, and the posterior nerve roots within the spinal canal, where they form one of the causes of compression paraplegia (p. 348). They may be both intra- and extradural in situation. They are also not uncommon on the nerve trunks, but are more frequently seen upon the nerve filaments, where they form painful subcutaneous tubercles.

The benign growths are found in persons otherwise healthy. They usually come on between the twentieth and fortieth years, and affect women slightly more often than men.

Symptoms. The symptoms of intracranial and intra-neural growths are elsewhere described (pp. 267 and 348).

When situated on a sensory nerve, they give rise to neuralgic pains. When upon a mixed nerve, the pain may be less severe, and is associated with motor weakness. Complete sensory and motor paralyses are practically unknown. The pain may be located at the seat of the tumour, or referred to the course of the nerve. It may be constant with exacerbations, or may be intermittent.

On examination, a swelling of varying consistence may be felt in the course of the nerve. The tumour is painful on pressure, and its recognition is usually easy. In some cases its presence has been observed by the patient for some time before the onset of symptoms. If a deep nerve is the seat of a growth, the diagnosis is more difficult, but the distribution of the pain and motor weakness, taken in association with the finding of a localised tender spot on pressure somewhere in the course of the nerve, is significant of the condition.

Fibromata are of slow growth, usually circumscribed and encapsulated, and composed of fibrous tissue arranged in whorls; the blood-vessels are well developed, and the growth does not invade the nerve fibres, which merely suffer by pressure.

Multiple benign growths are of various types:—

(*a*) *Multiple neuro-fibromata or general fibromatosis.* In this condition the nerves present an irregular, diffuse, thickened appearance, and may be the seat of multiple tumours similar to those described under the solitary forms. The

essential character of this condition is an increase of the fibrous tissue of the endoneurium with general thickening of the epineurium. In some cases the nerve fibres are degenerated. The posterior nerve roots and ganglia may be affected as well as the peripheral nerves.

Within the neural canal, the cauda equina and cervical roots are most frequently involved. Any of the cranial nerves but especially the fifth, seventh, eighth, and tenth—may be involved.

Clinically, in striking contrast to the almost invariable tenderness of the single tumours of nerves, general neurofibromatosis is characterised by an absence of tenderness on pressure; but during the course of the disease, one or more of the tumours may give rise to pain and local tenderness.

(*b*) *Plexiform neuromata.* In this case the change is confined to one or more contiguous nerves or plexuses. Pathologically, this variety is similar to that just described. Its most common situations are the forehead and temple, the posterior part of the neck, and the extremities.

(*c*) *Cutaneous neurofibromata*—Molluscum fibrosum, or von Recklinghausen's disease. The distribution of this affection is generalised over the whole body, with the exception of the palms and the soles. The tumours are mainly of soft consistence and subcutaneous.

Malignant tumours are sarcomatous and carcinomatous. The sarcomata are often primary, or may develop from a simple fibroma. They are spindle or round-celled, and may be single or multiple. They tend to invade the nerve and destroy the nerve fibres.

The symptoms at the onset are similar to those found in benign growths, but progress more rapidly, and the ultimate paralytic effects are more severe. The growth tends to spread along the nerves, and when situated in the intraneural or intracranial cavities, involves the dura mater and adjacent nerves.

They are common upon the cauda equina and the cranial nerves, but are less frequent upon the peripheral nerves of the limbs.

The carcinomata are rare.

Amputation neuromata are not tumours in the strict

sense, but are rather part of the normal regenerative process, and as such contain newly formed nerve fibres.

Treatment. In cases of single benign tumours of nerves, removal of the growth is satisfactorily and successfully carried out by ordinary surgical methods; but in general neurofibromatosis, when pain is a complicating symptom, it is preferable to resect a portion of the nerve higher up, to divide the corresponding posterior roots, or to amputate the affected limb.

Syphilis of the Peripheral Nerve

Syphilitic affections of the nerves may occur in combination with intracranial syphilis, but are much more commonly found in association with multiple gummata of the bones and muscles. The changes may be: (1) involvement of the nerve by an extra-neural gumma, (2) gummata in connexion with the sheath of a nerve, (3) a thickening of the nerve sheath and of the peri- and endoneurium, and (4) occasionally and in association with thickening of the nerve sheath obstruction of the vasa nervorum, and parenchymatous degeneration of the myeline sheath from arrest of the blood supply.

Chapter V

MULTIPLE NEURITIS

(Syn.: Peripheral Neuritis, Polyneuritis)

Multiple neuritis is a disease affecting, more or less simultaneously and symmetrically, all or many of the peripheral nerves.

Pathology. The pathological changes found in multiple neuritis are of two kinds: (*a*) interstitial neuritis, characterised by inflammation and proliferation of the nerve sheath and fibrous tissues of the peri- and endoneurium; (*b*) parenchymatous neuritis shown by degeneration and disintegration of the medullary sheaths and destruction of the axis cylinders.

These changes are most intense towards the termination

of the nerves, and may be followed by secondary alterations in the cells of the anterior horns and in the muscles.

In certain cases the primary changes are limited to the fibrous structures, the parenchymatous changes being secondary and slight. In others, the lesion is confined to the anterior horn cells and the nerve fibres. In most cases, however, both types are combined. These differences would seem to depend upon the selective action of the toxic agent, but in both varieties the peripheral portions of the nerves suffer more intensely than the proximal.

Varieties. The following etiological classification embraces all the well-recognised forms of multiple neuritis:

1. Cases due to poisons derived from outside the body—alcohol, the coal-tar products, lead, arsenic, mercury, copper, and phosphorus.

2. Cases due to poisons acquired or developed within the body as the result of infective disorders—diphtheria, influenza, malaria, typhoid fever, scarlatina, puerperal fever, gonorrhœa, septicæmia, and beriberi.

3. Cases associated with general disorders—diabetes, gout, rheumatism, anæmia, malnutrition, tubercle, and carcinoma.

4. Cases due to the local action of organisms—leprosy and syphilis.

The commonest form of multiple neuritis in this country is that due to alcohol. It may therefore be taken as the type of the disease, and will be described in some detail.

ALCOHOLIC NEURITIS

Etiology. Alcoholic paralysis occurs more frequently in women than in men, a fact which may be accounted for by the lesser resistance of the female sex to the effects of alcohol, and by the greater incidence of secret and persistent drinking amongst women. It is a disease of adult life.

It is a well-recognised fact that the disease is found not so much in those who drink openly as in those who drink in secret. It is also more common in soakers than in those who give way to periodic drinking bouts, and affects spirit more than beer drinkers. The exciting causes are exposure to cold, privation, and malnutrition.

Symptomatology. Alcoholic neuritis manifests itself clinically in three types—the sensory, the motor, and the ataxic. They may all be combined in the same case, but one type usually predominates in each individual instance.

The sensory type. The earliest subjective symptoms are numbness or deadness affecting first the feet and later on the hands. This is rapidly followed by the occurrence of pain on movements of the limbs and by painful cramps, especially in

FIG. 51.—Showing the characteristic drop feet of alcoholic neuritis.

the legs. Owing to these troubles the patient avoids muscular stress and strain, and takes to lying in bed or in an easy chair, so as to maintain the muscles in a state of relaxation. Trophic changes soon show themselves in the skin of the extremities, especially of the hands and feet, which are usually moist and of a sodden appearance. Some loss of power is also observed in the muscles of the limbs: at first due to an unwillingness to move the muscles, owing to the pain on movement rather than to any actual paralysis. As time goes on the symptoms increase, and contractures take place in the muscles. In the lower limbs, the hip and knee joints are flexed and the foot dropped at the ankle. In the upper limbs, the fingers and the wrist are flexed, owing to the predominant weakness of the extensors. Eventually the limbs become more or less fixed in these positions.

The muscles show general wasting, though not to any

marked extent, and the skin becomes thin, dry, transparent, and glossy. The hands and feet, and more especially the fingers and toes, are thinned and tapering. Eventually the patient becomes bedridden and powerless, but rarely develops bedsores. Often a well-marked tremor is seen in the hands, face, and lips.

With the progress of the disease the mental state shows increasing general enfeeblement. The patient becomes garrulous and emotional, crying or laughing alternately, and almost invariably exhibiting a piteous craving for sympathy.

Mental symptoms. It is noticed that persons who had previously shown a clear and open temperament become secretive and cunning, lose their finer moral feelings, and exhibit an indifference to what is right and wrong. This temperamental change may be accompanied by an abnormal sensitiveness to questions referring to the use or abuse of alcohol.

A further obvious impairment of the mental faculties is characterised by loss of memory for recent events, mental inertia, and blunting of mental acuteness and perception. Little by little this becomes associated with irritability, loss of self-control, and emotional instability. In addition a most characteristic feature, 'disorientation' or loss of appreciation of time and place, is noticed. Eventually such patients become dirty and careless in their habits, not from any weakness of sphincter control, but from loss of self-respect and want of attention to personal appearances. In some cases the onset of the disease is preceded by an attack of delirium tremens.

The *cranial nerves* are seldom affected, but the conjugate movements of the eyes are easily fatigued and not well sustained. Sometimes nystagmoid jerkings are observed.

It is rare to find peripheral facial palsy, although it occasionally occurs. The tongue is tremulous.

Motor system. Motor weakness is earliest seen in the extremities of the limbs, especially in the actions of dorsiflexion of the feet and extension of the wrist and fingers. Wasting of the muscles is not usually marked, but electrical stimulation may reveal a partial or complete reaction of degeneration.

Sensory system. The patient complains of deadness, numbness, tingling, and burning in the hands and feet and cramp-like pains in the muscles.

There is loss or impairment of all forms of cutaneous sensibility, especially over the distal portions of the limbs with a shading off towards the proximal parts.

Superficial tenderness is occasionally present; but in all cases there is a great muscular hyperæsthesia and even moderate pressure on the muscles may evoke great agony—a point of differential diagnosis from tabes dorsalis.

The sense of passive position and of movement of the limbs is impaired.

Reflexes. The deep reflexes are diminished and finally lost. The superficial are increased at first, but later may become diminished or absent.

The *sphincters* are not affected; but in the later stages, owing to the mental condition, the calls of nature are not attended to.

Trophic. The skin of the extremities is at first moist and sodden, but later shows dryness and glossiness, sometimes shedding fine epithelial scales, which appear like a thin tissue over the underlying skin. In this way the typical tapering, thin, dry, glossy, and semi-translucent hand and fingers are produced. An œdematous condition of the extremities is sometimes found in cases with cardiac and renal complications. The nails become ribbed and brittle, and the hair fine and thin. Trophic joint changes with adhesions may also develop and cause permanent deformity, even in cases which recover.

The motor type. In this variety of alcoholic neuritis the earliest symptoms are weakness of the legs and the readiness with which fatigue is induced. The weakness is at first seen in the distal portions of the extremities, especially in the dorsiflexors of the feet and the extensors of the wrist and fingers. The toes catch the ground in walking, and steppage gait, drop foot, and drop wrist, are characteristic. The degree of weakness may be out of all proportion to the discernible muscular atrophy, although in some cases an early and rapid muscular wasting occurs, the muscles becoming flabby, hyperæsthetic, and losing their myotatic irritability. In many the wasting, which eventually ensues, does not appear until a late stage.

In this type subjective paræsthesia in the hands and feet is almost always present, as well as muscular hyperæsthesia;

but cutaneous sensibility may only be relatively impaired. The deep reflexes are lost early. Trophic changes in the skin are late symptoms. The mental condition is as already described.

The ataxic type. In this type inco-ordination of movement is a striking feature, and may come on with the earliest symptoms of motor weakness. It is most marked in the legs, and is always associated with muscular tenderness and sometimes with muscular hypotonicity. Objective cutaneous sensory changes are minimal in degree and extent, but loss of sense of position of the limbs is relatively more pronounced.

The deep reflexes are lost early.

This form of peripheral neuritis is probably due to a proportionately greater affection of the fibres subserving deep rather than epicritic and protopathic sensibility.

This type is best seen in the subacute and chronic cases of the disease.

Complications. Insomnia, often of a profound and troublesome type; nausea, retching, sickness, and loss of appetite are early and persistent features. Irregularity of the heart's action, often associated with dilatation and fatty changes, is also found.

Bronchitis in older subjects, and pulmonary tuberculosis in younger patients, constitute grave and serious complications.

Albuminuria is frequent; but the most serious complication is the liability towards pleural, pericardial, or peritoneal effusions. Febrile disturbances are liable to produce delirium and hyperpyrexia, from which recovery is rare.

Prognosis. If the case is seen early and the cause can be entirely removed, recovery in from two, three, or four months may take place under appropriate treatment. In more severe cases, recovery may also be obtained as a result of persistent treatment up to three years. In cases with much mental impairment, recovery is never complete, as the inertia and obstinacy of the patient militate against all attempts at improvement. Second and third attacks are less satisfactory, and recovery, even if eventually complete, may be delayed for months or years.

Treatment. The first essential of treatment is the removal of the cause. Simple as this may seem, its accomplishment is impossible unless the patient is removed to an institution,

or placed under the direct and continuous supervision of trustworthy attendants.

Secondly, the patient is placed in bed and the pain eased, natural sleep induced, and the disordered digestion corrected by suitable remedies and diet.

Hypodermic injections of strychnine (3–5 minims of the liquor) may be given twice or thrice daily, combined with general tonic treatment. As the chief hindrance to recovery is the weakness of will-power of the patient to persevere with treatment, firmness, patience, and encouragement on the part of the physician are essential.

Local treatment. One of the earliest and most important points in the treatment of these cases is to prevent the development of troublesome contractures of the hands and feet. This is effected by freeing the legs from the weight and pressure of the bed-clothes by the employment of a cradle, and by seeing that the limbs are maintained and supported in a correct position by sand-bags or other means.

If contractures have not developed, their occurrence is prevented by aid of gentle passive movements, which should be employed as soon as possible. If already present, they are overcome by the adoption of mechanical appliances—such as 'Gowers's boots,' which are designed to counteract the over-action of the calf muscles; and, if necessary, of the flexors of the knees by the aid of elastic straps attached to the feet.

Where these are insufficient, adhesions and contractures require to be broken down under anæsthesia, and extension afterwards applied.

As soon as the acute stage has subsided, galvanism or galvano-faradisation should be applied to the limbs in combination with gentle massage, in order to maintain and improve the condition of the muscular, sensory, and trophic systems. Hot-air baths and warm local applications are of service both in the relief of pain and in assisting the reparative process in the nerves.

ARSENICAL NEURITIS

This form of neuritis occurs in those who are, in various ways, subject to poisoning by arsenic or its preparations. Of these may be mentioned the chronic poisoning which occurs

in certain industries such as the manufacture of wall papers containing arsenic in the pigments, in sulphuric acid factories, and in the manufacture of artificial flowers. A form of arsenical neuritis was found amongst the drinkers of beer brewed from glucose, which had been prepared with contaminated sulphuric acid, in Manchester in 1899 1900. It has also been seen in children suffering from chorea who have been treated for long periods with Fowler's solution.

Arsenic affects the peripheral nerves by producing a parenchymatous neuritis without inflammation of the interstitial tissues. The axis cylinders are as a rule preserved. Arsenic, however, like lead, has an effect upon the nervous system as a whole, giving rise to degenerative changes in the cells of the anterior horns of the cord and of the cortex cerebri.

The **symptoms** are comparable to those described under alcoholic neuritis. The earliest symptoms are paræsthesia and pains in the limbs, to which paralysis and ataxia are superadded. There is usually marked loss of motor power before wasting ensues. The reaction of degeneration is found in the muscles. The palsy is commonest in the legs, is bilateral and symmetrical, and affects the extensor muscles in excess of the flexors. The result is drop foot and drop wrist often of a marked and persistent kind.

Deformities may result in talipes equino-varus and flexor contracture at the wrist.

In some cases there may be rapid wasting with loss of motor power coming on simultaneously, but in these cases spinal cord changes probably coexist.

The sensory symptoms are excessive hyperæsthesia of the muscles of the arms and legs, and objective anæsthesia of the peripheral tpyc.

The tendon reflexes are abolished.

Of importance in the diagnosis of arsenical neuritis are the coexistent skin changes, which take the form of pigmentary discoloration around the neck and on the abdomen. The skin is dark brown and mottled, but the mucous membranes are never discolored as in Addison's disease.

Prognosis. The recovery is much slower than in the alcoholic form, and even after recovery from the paralysis, paræsthesia and numbness may persist.

The **treatment** is conducted on the lines laid down for alcoholic neuritis.

LEAD NEURITIS

Lead neuritis occurs in workers in lead—painters, compositors, and plumbers—and also as a result of drinking water contaminated with lead.

Lead also exerts extensive general effects upon the nervous system: first by its direct action upon the central nervous apparatus (brain and spinal cord), and secondly, indirectly, through its action upon the blood-vessels, heart, and kidneys. In consequence of such lesions it may give rise to numerous disorders affecting the nervous system other than peripheral neuritis —such as cerebral hemorrhage and thrombosis.

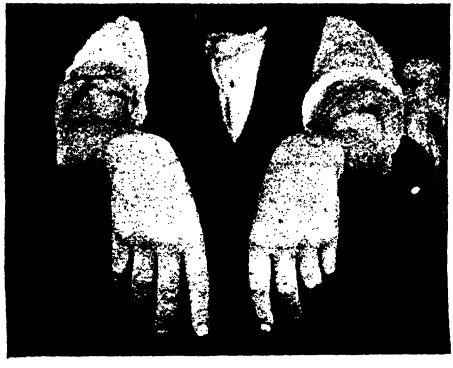


FIG. 52.—Showing double drop wrist in a case of lead neuritis.

A history of one or more attacks of lead poisoning—such as colic, and sometimes of a previous attack of neuritis—is generally obtained. The patients are often anæmic, and in almost every case a blue line may be observed along the margin of the gums.

The **symptoms** are confined to the motor system, sensory symptoms being very rare. A fine tremor of the hands is an early and prominent feature. The paralysis affects the arms earlier and more extensively than the legs: in some cases being limited to the arms. The muscles most paralysed are the extensors of the wrist, thumb, and fingers. (Fig. 52.)

The distinction between a musculo-spiral paralysis and the multiple form of peripheral palsy is the retention of the power of the supinator longus muscle, and sometimes of the extensor metacarpi pollicis in the latter type.

The legs are only rarely affected, in which event the peronei and anterior tibial muscles are those primarily paralysed.

In these cases wasting and muscular atrophy are not marked, although the reaction of degeneration is present; sensory changes are strikingly absent.

There is another type of case in which the shoulder muscles are mainly affected: the deltoid being earliest involved, and later the biceps, brachialis anticus, triceps, and supinator longus.

On the other hand, a type has been described in which general muscular wasting—often irregularly distributed throughout the body, but sometimes confined to the small muscles of the hands—is associated with an increase of the deep reflexes and an extensor response. These cases, which resemble the amyotrophic type of palsy, are not really neuritic, but are due to an associated degeneration of the pyramidal tract and anterior horn cells of the spinal cord.

Prognosis. The disease runs a long course of several months, recovery taking place first in the least affected muscles; relapses are not uncommon on returning to work.

The **treatment** is carried out on the general lines laid down for alcoholic neuritis. The elimination of lead from the system is favoured by the administration internally of iodide of potassium, while occasional free purgation is an important aid.

DIABETIC NEURITIS

It has long been recognised that peripheral neuritis may be associated with the presence of sugar in the urine. Its occurrence is probably due to defective metabolism and auto-intoxication.

Symptoms. Diabetic neuritis differs from the alcoholic form in that subjective paraesthesia are slight, though pain may be severe. Almost invariably the legs are more affected than the arms, and the external popliteal nerve is especially prone to suffer in the early stages. Drop foot and paralysis are only seen in the severer cases, but weakness of the legs and ataxia may be present.

The ankle jerks are abolished, and the knee jerks may be

retained, diminished, or lost. Muscular hyperæsthesia and slight sensory changes are present. The predominant sensory change is loss of the sense of vibration (Williamson).[1]

Trophic sores, either in the form of gangrene or perforating ulcer, may be observed in the feet, but are distinguished from the tabetic ulcers by the fact that they are painful.

We have observed several cases in which there was for a long period unilateral isolated affection of the external popliteal nerve.

The **differential diagnosis** of diabetic neuritis from tabes dorsalis is based upon the absence of pupillary changes, the presence of muscular hyperæsthesia, the early loss of vibration sensibility, the presence of slight motor weakness, and by the detection of sugar in the urine.

The **treatment** of this condition is that of diabetes mellitus (dietetic and medicinal) combined with local applications of electricity and massage.

POST-DIPHTHERITIC NEURITIS

Parenchymatous neuritis, with occasional interstitial changes, is the characteristic lesion in the nerves in post-diphtheritic paralysis. The changes are best seen in the intramuscular portions of the nerves; but the large cells, both of the anterior horns and the posterior root ganglia, may be affected.

The evidence, so far as ascertained, seems to point to the anterior horn cells of the spinal cord as that part of the nervous system which is first affected. This is followed by degenerative changes in the most peripheral portions of the neurone, and later on in the whole nerve.

There is no relation between the severity of the original disease and the occurrence or extent of paralysis. It is more common, however, after the faucial than after any other form. The paralysis is stated to occur in about 8 to 10 per cent. of all cases of diphtheria.

The early adoption of the serum treatment of diphtheria may materially diminish the liability to paralysis, especially of a severe type. Rolleston[2] states that the onset of post-diphtheritic paralysis varies from 4·9 per cent. when antitoxin

[1] Williamson, *Lancet*, 1905. [2] Rolleston, *Practitioner*, 1909.

is given on the first day, to 31·4 per cent. when its administration is delayed until the fifth day.

Symptoms. By far the most frequent, and usually the earliest, symptom is paralysis of the soft palate, which gives rise to a nasal tone of voice and a tendency for fluids to regurgitate through the nose. When tested, it will be found that patients are unable to blow out the cheeks, or to suck or gargle. On examining the throat, the palate may hang lower than usual, it may or may not be insensitive, but the palatal reflex is always diminished or lost.

In slight cases the paralysis may be confined to this region, and gradual recovery takes place within two or three weeks. In severer cases paralysis of the glottis, characterised by loss of voice and inability to cough, with impairment of sensibility is found. In other cases paralysis of the pharynx permits food to pass into the glottis.

Cycloplegia, or paralysis of accommodation with preservation of the light-reflex of the iris, does not occur before the fourth or fifth week. Other cranial nerves are rarely paralysed, except the sixth; and ill-sustained conjugate movements of the globes may also be observed.

Paralysis of the limbs may coincide with the palatal and ocular paralyses; but usually appears about the fifth or sixth week. In some cases, especially nasal diphtheria, the palate may escape, but the extremities suffer earliest.

The legs are first affected, and later on the arms, intercostal, and trunk muscles. When atrophy occurs it is usually rapid, and the muscles show degenerative electrical changes. Palsy of the diaphragm and of the heart muscle are the most serious symptoms, and in severe cases come on early in the course of the disease.

The sensory symptoms are slight, and consist of numbness and paræsthesia; but muscular hyperæsthesia is neither common nor well marked. Objective sensory disturbances of the peripheral type may be found.

The deep reflexes are diminished or lost. The knee jerk is in abeyance from the commencement, and may remain absent from three to eight months, after all other signs of the disease have disappeared.

The sphincters are rarely affected.

The **prognosis** is good, provided that the respiratory

muscles are not affected, and that the heart's action remains normal. The palatal paralysis resolves in from two to three weeks, and the palsy of the limbs in about three or four months, though the knee jerks may remain in abeyance for eight months or longer.

The **treatment** is that of multiple neuritis. Complete rest in bed is essential, ample nourishment is necessary, and stimulants may be required. Care should be taken to prevent food entering the larynx; if there is severe pharyngeal paralysis, it may be necessary to feed through the nose. Hypodermic injections of strychnine are valuable.

There is no satisfactory evidence to show that injections of antitoxin have any remedial effect upon the symptoms of paralysis once they have arisen.

ACUTE TOXIC POLYNEURITIS

Cases resembling Landry's paralysis, following influenza, have been variously interpreted. In several such personally observed, in which lumbar puncture yielded no evidence of organismal infection of the central nervous system, the motor and sensory symptoms pointed to a multiple neuritis.

The clinical picture presented by these cases shows, in the first place, a slight degree of numbness in the hands and feet without any muscular hyperæsthesia or objective sensory loss. This is rapidly succeeded by motor weakness in the arms and legs of wide distribution, affecting both the proximal and distal parts, but predominating in the extensor groups. At this stage the deep reflexes rapidly diminish, and are soon lost. Within two or three days muscular wasting of slight degree but wide extent, with electrical degenerative alterations, appears. Synchronously with this, complaint is made of pains and muscular tenderness, and loss or diminution to all forms of sensation in the peripheral portions of the limbs is detected. The motor weakness may also affect the muscles of the trunk, thorax, and in some cases the face; and a case seen with such paralyses before the onset of the sensory symptoms is clinically indistinguishable from one of Landry's paralysis. The sensory changes, however, become so definite that the diagnosis is made sufficiently clear. Recovery is usually rapid and complete, the deep

reflexes remaining in abeyance for some time after the motor power has returned. It would seem that in these cases the toxic action fell first upon the motor cells in the spinal cord, and later gave rise to parenchymatous neuritis. (See Table, p. 448.)

Barnes[1] and Williamson[2] have described under the term 'Toxic Degeneration of the Lower Neurones' a condition which arises after acute febrile affections. It is characterised in the early stages by slight sensory symptoms, and is attended by great atrophy of the small muscles of the hands. Recovery is slow as regards paralysis and atrophy, and in one case two relapses occurred.

This condition would appear to us to resemble that just described under acute toxic polyneuritis, but to differ from it in the fact that the course of the malady is of a subacute or chronic character, and that the changes fall particularly upon the small muscles of the hands.

In one case submitted to post-mortem examination, the morbid appearances consisted of degeneration of the peripheral nerves of the arms and atrophy of the anterior horn cells.

Various forms of *local neuritis* are also observed after acute febrile attacks. The most common varieties occur in connexion with the brachial, sciatic, and occipital nerves.

Meralgia paræsthetica, or pain and tenderness along the external cutaneous nerve, is also found in this association. Probably of the same character are the 'tender toes,' so common in typhoid fever.

LEPROUS NEURITIS

This is a rare disease in this country, but is from time to time seen in persons who have resided in localities in which leprosy is endemic.

Pathology. In the early stages of the disease, some enlargement of the nerves may be detected, but when the malady is more advanced, or the neuritis of old standing, atrophy of the affected nerve trunks is the rule. The characteristic change in the nerves is an interstitial neuritis, due to the presence of the leprosy bacillus in the meshes of the peri- and epineurium. The infection of the nerves by the

[1] Barnes, *Brain*, 1902. [2] Williamson, *Brain*, 1903.

bacillus is the outstanding pathological feature of this form of neuritis. Small foci of bacilli are detected in the interstitial tissues, more especially in relation to the neurilemma cells. Secondary parenchymatous degeneration of the myeline sheaths is caused by their pressure upon the nerve fibres, as well as by the overgrowth of the connective tissues of the nerve trunks. The bacilli are found in most organs and tissues of the body, but are not, as a rule, detected in the central nervous system.

Symptoms. The symptoms of leprous neuritis are slow and progressive in character. In a case observed by ourselves, fifteen years elapsed between the first sign of paralysis and death from an intercurrent pulmonary affection. The leading clinical features of the disease are progressive atrophic paralysis, anæsthesia of the distal portions of the limbs or of irregular and patchy distribution, and trophic changes in the extremities.

These symptoms may not be confined to the limbs, but may be referred to the nerves of the trunk and face.

The *motor symptoms* consist of a slowly progressive atrophic paralysis of irregular distribution, though often affecting muscles supplied by a particular nerve. Paralysis of any one muscle is slow and incomplete until an advanced stage of the disease. This is probably accounted for by the escape of some nerve fibres. The reaction of degeneration is present.

The *sensory symptoms* are subjective and objective. The subjective are often slight, pain is rare, but numbness and tingling are met with in most cases. The objective changes are variable, not only as regards their distribution, but also in the quality of the sensory loss. In general terms it may be stated that the sensory loss is found over the distal portions of the limbs, but local patches of more or less complete loss may be found in association with more advanced lesions. Epicritic loss may alone be present, or both epicritic and protopathic, in which case trophic sores are common. Loss of deep sensibility may also be present.

The state of the *reflexes* depends upon the position of the nerve lesions.

The *skin changes* consist chiefly of discoloration of a bronze appearance, occurring in patches. Localised thickenings, or prominences of the skin of the face, ears, and other

parts, occur from the local deposition of bacilli in the subcutaneous tissues. The nerve trunks may be felt on palpation when hypertrophied in the early stages.

The **diagnosis** has to be made mainly from spinal gliosis and syringomyelia. The association of atrophic paralysis—sensory changes consisting of the characteristic dissociation of thermal and painful from tactile sensibility—and trophic alterations in the extremities, are common to both disorders.

The detection of the leprosy bacillus in the subcutaneous tissues places the diagnosis beyond dispute.

The **course** of the disease is towards a fatal termination from intercurrent disorder, and **treatment** is of little avail, except in the management of symptoms or complications.

BERIBERI

This is a disease endemic in some Eastern countries, but from time to time taking on an epidemic character. Sporadic cases are occasionally seen in this country amongst sailors from the East. It is due to the presence of a micro-organism, which has been found in many of the organs and tissues of infected persons. It would appear to effect an entrance into the body mainly through the alimentary canal, probably from the consumption of rice. Its habitat has been shown to be the duodenum, where it grows and multiplies, and whence a toxic substance is introduced into the circulation. The development of the disease is favoured by states of impaired nutrition, bad hygienic surroundings, and exposure to cold and damp. It affects males more often than females, and young adults in preference to older people.

Symptoms. Gastro-intestinal symptoms—such as loss of appetite, a feeling of epigastric oppression, nausea, and vomiting, accompanied by malaise, proneness to fatigue, and palpitation—usually precede the onset of the nervous symptoms. These are, in their main features, similar to those which have been described under alcoholic neuritis. Numbness, paræsthesiä, and muscular hyperæsthesia usher in the onset of motor weakness in the hands and feet. The tendon jerks are abolished, cutaneous sensibility is impaired, and the electrical excitability of the affected muscles is impaired or lost.

In severer cases, rapid atrophic paralysis with the reaction of degeneration develops, and the palsy extends from the limbs to the muscles of the trunk. Complete paralysis supervenes with great pain and muscular tenderness, and death may occur from exhaustion or intercurrent disease.

A special feature of beriberi is the early involvement of the cardio-vascular system. The heart's action is rapid and irregular, the arterial tension falls, and œdema of the extremities, sometimes with effusion into the serous cavities, is a characteristic feature of most cases.

Course. The average duration of the common type varies from one to several months. The disease is not usually fatal except at the height of an epidemic. Death may occur from exhaustion, heart failure, or intercurrent disease.

Treatment is based on the principles laid down for other forms of peripheral neuritis. The tendency towards cardiac asthenia requires the administration of stimulants and heart tonics.

PROGRESSIVE HYPERTROPHIC INTERTESTIAL NEURITIS

This is a rare disease, originally described by Déjérine and Sottas. It is characterised pathologically by thickening of the nerve roots, enlargement of the spinal ganglia, and increase in volume of the peripheral nerves. The thickening of the nerves is due to an overgrowth of the fibrous tissue of the nerve bundles and nerve sheaths, with resulting atrophy and disappearance of the nerve fibres. The spinal cord may present secondary degenerative changes, especially in the posterior columns. The muscles also show changes in their structure, consisting of disappearance of the normal striation, and the replacement of the muscular fibres by fat and fibrous tissue.

The clinical phenomena consist of lightning pains, ataxia with muscular atrophy, marked sensory changes, and hypertrophy of those nerve trunks which can be felt under the skin, Nystagmus, reflex pupillary immobility and kypho-scoliosis are also observed.

The leading clinical features of the disease, therefore,

include generalised amyotrophy, kypho-scoliosis, and tangible hypertrophy of the nerve trunks. The tendon reflexes are lost, and the sphincters are unaffected.

The disease somewhat resembles tabes dorsalis, from which, however, it may be distinguished, clinically, by the absence of sphincter impairment and the presence of definite hypertrophy of the peripheral nerves, and pathologically by the characteristic alterations in the nerve fibres.

Miscellaneous forms of neuritis

Oppenheim and others have described a form of multiple neuritis occurring in old age, associated with arterio-sclerosis. In these cases the symptoms of neuritis are slight and of slow onset, and the prognosis is not unfavourable. Multiple neuritis has also been stated to occur as a result of poisoning by carbon monoxide, sulphide of carbon, and the coal-tar products—such as sulphonal, trional, antipyrin, and the anilines.

Other forms of toxic neuritis, in addition to those already described, are found in connexion with septicæmia, the puerperium, gonorrhœa, malaria, and tuberculosis.

Local neuritis is not uncommon in association with various conditions—such as rheumatism, gout, tubercle, syphilis, and carcinoma.

Tobacco. In chronic tobacco-poisoning, various symptoms may be present—vertigo, tachycardia, nausea, pain after food, insomnia, neuralgia and general tremulousness. More characteristic, although less common, are interference with the heart's action and amblyopia from retro-bulbar neuritis. The symptoms of the latter are impairment of vision and central scotoma for green, which in severe cases may increase so as to impair all colour sense.

Chapter VI

NEURALGIA

Neuralgia is a clinical term signifying pain in a nerve. The term has been applied loosely to all forms of nerve pain, irrespective of the presence or absence of obvious lesion of the

nerve elements. While fully recognising the impossibility of determining, in many cases, whether nerve pain is due to an organic lesion, or not, it is advisable to differentiate tentatively between three types : —

(*a*) Neuralgia unassociated with either present or previous organic disease.

(*b*) Neuralgia associated with organic disease—gout, rheumatism, tabes dorsalis, and neuritis.

(*c*) Neuralgia following upon organic disease—such as malaria, influenza, herpes zoster, and neuritis.

Etiology. Many conditions predispose to neuralgia. It may come on at any age, but childhood and old age are less prone to it than middle life. Women would appear to be more liable than men. It is more common in persons of nervous temperament, and in the weak, the anæmic, and the debilitated. Gouty and rheumatic subjects are peculiarly susceptible.

The exciting causes are: the infective disorders—more especially malaria and influenza; poisons introduced from outside the body—such as lead and alcohol; poisons formed within the body—as in gout and diabetes; exhausting diseases, and exposure to cold and damp.

The influence of mental and physical strain, of overwork, anxiety, and distress is often a potent factor in its causation.

Pains of a neuralgia-like character may be found in association with organic disease of the central and peripheral nervous system, in traumatic lesions of the nerves, in combination with osteo-arthritis, and in herpes zoster.

Pain may be referred to various regions of the body—at some distance from the actual seat of disease in an organ or viscus. These are not genuine neuralgias, but are known as 'referred pains.'

Symptoms. Neuralgia as a symptom occurs either as pain limited to one nerve or its branches, or as an element in general nervous debility. In most cases of neuralgia, certain painful points on pressure may be discovered. These are found where cutaneous branches penetrate bony canals or fibrous structures. These are usually the seats of the greatest pain, although pain may also affect the nerve trunk and its branches.

The character of the pain varies. In true neuralgia it is

paroxysmal. Sometimes it is dull, boring, or burning. At other times it is sharp and lancinating, darting through the limb, face, or body. The duration of the attacks varies from a few seconds to several minutes, and their frequency from two or three times daily to several in an hour. In some forms of neuralgia the pain may be very severe, and accompanied by cutaneous hyperæsthesia, so that the patient is unable to bear the pressure of the bed-clothes; and in the trigeminal form, the slightest breath of cold air may start a paroxysm of pain.

The pain may be accompanied by a feeling of numbness or deadness in the affected limb.

Vaso-motor symptoms are not uncommon. These may take the form of patches of erythema, irregularly scattered over the neuralgic area. Angio-neurotic œdema has been seen in association with severe attacks, and subcutaneous lumps or nodules of a transient character have also been observed.

The most characteristic feature of neuralgia is its tendency to relapse. In the malarial and post-influenzal varieties the relapse may occur at a definite and fixed time, and pass off as the day advances, to return at the corresponding hour next day. The malarial variety of neuralgia is most prone to affect the supra-orbital and sciatic nerves: post-influenzal neuralgia especially the supra-orbital branch of the trigeminus.

Diagnosis. Neuralgia requires to be distinguished mainly from neuritis. In the latter condition the pain is constant rather than paroxysmal, and the nerve is tender usually throughout the greater part of its extent; in severer forms of neuritis paralysis, wasting, and anæsthesia are present.

A form of pain, not unlike that which is characteristic of neuralgia, may be found as a purely psychical condition, increased by mental and emotional causes, and relieved by suggestion and distraction.

Neuralgic pains are found as symptoms of many organic diseases of the nervous system—such as tabes dorsalis, tumours pressing upon the spinal nerve roots, and chronic meningeal lesions. It is therefore important to eliminate such diseases before the diagnosis of neuralgia is made.

Prognosis. The prognosis is that of the different forms of neuralgia to be presently described.

Treatment. In the first place, attention should be paid

to the cause of the disease, and treatment directed to the underlying predisposition. All exciting causes should, as far as possible, be removed. Tonics may be administered, either in the form of quinine, iron, arsenic, and strychnine; or as the glycero-phosphates—Easton's syrup, or cod-liver oil. In special forms of neuralgia, such as the malarial and influenzal, no remedy surpasses quinine, both as a prophylactic and as an analgesic—5 grs. of quinine taken half an hour before the onset of the attack of pain will often prevent its occurrence. Salicylate of soda, salicine, aspirin, phenacetin, antipyrin, and the other analgesics, may all be used from time to time in the treatment of this affection.

Cases due to syphilis and lead, or of obviously gouty or rheumatic cause, should be treated according to general principles.

Massage, hydropathy, the application of the galvanic current and vibration may also be of use in particular cases, or at some period in the course of the disease. Nerve stretching, neurectomy, excision of the posterior root ganglia, or division of the posterior nerve roots may be necessary in otherwise intractable cases.

TRIGEMINAL NEURALGIA (TIC DOULOUREUX)

This is a painful disease of the fifth nerve, unassociated with a definite or well-established morbid anatomy, although in most cases, in which the Gasserian ganglion has been subsequently examined, an interstitial fibrous overgrowth has been found. The ganglion cells are, as a rule, of normal appearance. Thickening of the nerve sheath and endarteritis have also been observed.

The disease is one of adult and late adult life, beginning between the ages of forty and fifty, and continuing for many years. It occurs about equally in the sexes.

Symptoms. It is characterised by paroxysms of intense pain, lasting a second or two, and continued for a few hours or days, over prolonged periods. The distribution of the pain is over one or more of the branches of the fifth nerve. It may come on sometimes without any cause: at other times on attempting to speak, smile, or laugh; when eating, while cleansing the teeth, or occasionally from a breath of cold air upon the

cheek. The pain may affect one or more spots; sometimes it is the nose or the eye, or it may be the jaw, or the side of the tongue, or the roof of the mouth. From the starting-place it may radiate over the branch mainly affected. The starting-points are those where a branch of the nerve becomes subcutaneous. These spots are tender to the slightest pressure in some cases, but in others no such tenderness is detected.

The paroxysms of pain are usually accompanied by flushing of the face on the affected side; there is often a shiny or greasy appearance of the skin; sometimes the cheek is puffy or œdematous; there may be a profuse secretion of tears, photophobia, and increase of the salivary secretion. Tremors or twitchings of the facial muscles may accompany the paroxysms.

The facial appearance of these patients, after the disease has persisted for a time, is often characteristic. There is an expression of stolidity from a fear of emotional display—as the slightest facial movement may induce a painful paroxysm. The mouth is foul and the breath offensive—as attempts to cleanse the teeth give rise to pain. In well-marked cases the jaws are edentulous—as the teeth, carious and healthy alike, have been by degrees extracted in the vain hope of curing the malady.

Diagnosis. The well-established disease offers no difficulty in diagnosis, but in the early stages and in all cases of facial neuralgia, the possibility of carious teeth, of malignant disease, or of suppuration in the sinuses (frontal, maxillary, ethmoidal) should be borne in mind as not improbable causes of the pain.

Prognosis and course. The disease may run a prolonged course over many years, with no tendency to spontaneous cure. Although the paroxysms may recur at frequent intervals, long periods of freedom from pain, amounting to several months or years, are not uncommon. The tendency in the severe cases is for the relapses to occur with increasing frequency.

The **treatment** consists of both medical and surgical methods. The only certain cure of the disease lies in excision of the Gasserian ganglion. This operation has now been proved in some hundreds of cases to be entirely satisfactory, recurrence of the pain rarely, if ever, taking place.

Medicinal remedies by the mouth are of little use except as palliatives. The most efficacious are quinine, in two to five grain doses, thrice daily; tincture of gelsemium or aconite pushed to the production of toxic symptoms, and butyl-chloral, ten to fifteen grains, repeated about every four hours. Analgesics—such as antipyrin, phenacetin, phenalgin, exalgin, and aspirin—may be of temporary use, but cannot be regarded as in any sense trustworthy remedial agents. Colchicum has given relief in gouty cases of a plethoric nature.

Injections of substances into the nerve have been resorted to lately, with considerable benefit in many of the recorded cases. This method has been advocated as being less dangerous than excision of the Gasserian ganglion, while affording a more permanent relief than can be obtained by drugs. Two solutions have been used successfully: (1) 5-10 minims of 80-per-cent. alcohol; (2) 5 minims of a 2-per-cent. solution of osmic acid. These may be injected into the branches of the nerve as they issue from their respective foramina.

Subcutaneous injections of morphia and cocaine are not to be recommended, and should only be given under critical and exceptional circumstances.

The local application of galvanism (positive pole over the tender points) is sometimes attended by highly satisfactory results, but in other instances the pain may be increased.

SCIATIC NEURALGIA—SCIATICA

Pain in the distribution of the great sciatic nerve and its branches is a common ailment, more especially in men of adult and middle age. In contrast to brachial neuralgia, it is more frequent upon the left side.

Some cases would appear to be instances of neuralgia, but the majority are due to an inflammatory affection of the sheath of the nerve—a perineuritis. In all old-standing or inveterate cases, especially if accompanied by much lameness, deformity, or wasting of the limb, a careful examination of the hip joint should be made, as sciatic pain is a common accompaniment of osteo-arthritis of the hip joint, just as brachial neuralgia is frequently associated with a similar affection of the shoulder joint.

Etiology. The disease is often hereditary, and is predisposed to by gout and rheumatism. The great exciting cause is exposure to cold and damp, especially in those undergoing prolonged physical strain. Bilateral sciatic pain is suggestive of disease within the spinal canal, of malignant or tuberculous disease of the sacrum and pelvis, and of osteo-arthritis of the hip joints.

Symptoms. Pain of a constant gnawing character, in the position of the trunk of the nerve or its branches, with tenderness on pressure along the nerve or in certain places, is the chief characteristic of sciatica. The tender points are: (*a*) over the sacro-sciatic notch; (*b*) behind the great trochanter; (*c*) in the middle of the thigh posteriorly; (*d*) behind the head of the fibula, and on the peroneal aspect of the leg; and (*e*) on the outer side of the ankle or foot.

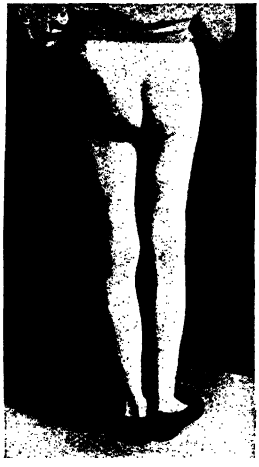


FIG. 53.—Photograph of a case of old-standing sciatica of the right leg, showing slight general muscular wasting.

At the onset of an attack, the pain may be so severe as to confine the patient to bed; but in the later stages it becomes intermittent, being readily induced by fatigue, exercise, or any sudden strain or effort of the leg.

The pain is increased by movements of the limb, by bending the body at the hips, or by flexing the hip with the leg extended at the knee, and in walking. Hence a characteristic attitude of the leg is assumed, the hip and knee joint being slightly flexed, and the heel raised from the ground. Owing to the sense of comfort brought about in this way, contracture of the leg—sometimes of a very troublesome character—is induced.

The pain induced by pressure on the nerve in sitting favours a posture in which the patient rests upon the tuber ischii of the sound side.

As a rule muscular atrophy is absent, although in old-standing cases a general wasting of the whole limb may

be observed. Unless some degree of neuritis is present, no sensory changes are detected. When present they consist of impairment of the epicritic sensibility over the sole and outer side of the dorsum of the foot, and the peroneal aspect of the leg. The knee jerks are brisk. In the neuralgic cases the Achilles jerks are normal; in the neuritic cases, on the other hand, the Achilles jerk on the affected side is abolished, and may remain in abeyance even long after the pain has disappeared.

Obvious muscular wasting, with impairment of and qualitative changes in the electrical irritability of the muscles and decided sensory loss, point to a sciatic neuritis.

Diagnosis. Pain along the sciatic nerve may be due to several conditions other than sciatic neuralgia. These are:—

1. Sciatic neuritis.
2. Disease of the hip joint.
3. Lesions of the cauda equina.
4. Malignant disease of the pelvic organs.

Sciatic neuritis is characterised by muscular wasting, sensory impairment, abolition of the ankle jerk, nerve tenderness, and altered electrical excitability of the muscles.

In *disease of the hip joint* the use of the Röntgen rays is most valuable in revealing changes in the head of the femur or the acetabulum. The two affections of this joint most commonly observed are tuberculous disease and osteo-arthritis. In both the movements of the joint are restricted, and the limb may be slightly shortened and rotated inwards. Pain on pressure along the nerve is rare. Rheumatoid changes in other joints may also be observed.

In *lesions of the cauda equina*, whether arising from primary intraneural disease or from caries of the sacrum, muscular wasting with electrical alterations, segmental sensory disturbances and sphincter weakness, are usually sufficiently obvious to make the diagnosis clear (p. 120).

The **prognosis** of the malady varies, as sciatic pain may persist over many months. One of its features, as in all cases of neuralgia, is a tendency to relapse, apparently without any discoverable reason. We have seen sudden turning in bed bring on a relapse more severe than the original affection. Post-herpetic sciatica is a severe variety of the malady, but pursues a more continuous course towards

recovery. A cure may be guaranteed in all cases of true sciatica.

Treatment. Sciatica is a very troublesome malady, and often taxes the resources of the physician. In the early stages rest in bed is essential, when hot applications and mild counter-irritants may be applied to the leg. Radiant heat applications and 'mud baths' are also of benefit, when administered daily for a time. Internally, iodide of potassium, salicylate of soda, salicine, and aspirin may be prescribed with benefit. Injections of morphia may be necessary.

After the acute stage has passed away, massage, passive movements of the limb, especially if contracture is present, and sometimes galvanism, are of great service. The thermo-cautery or blistering, applied over the tender point or along the line of the nerve, has often been of use in relieving pain and discomfort.

Sulphur appears to have in many cases almost a specific action, and may be prescribed either internally, as sulphur water, or in the form of sulphur baths.

Injections of chloroform, osmic acid, and alcohol into the nerve have been advocated.

In old-standing or inveterate cases, following upon an inflammation of the nerve sheath, the nerve may be exposed and the sheath freed, or nerve-stretching may be attempted after its exposure by surgical methods. The majority of cases resolve without any surgical assistance.

OTHER FORMS OF NEURALGIA

Cervico-occipital neuralgia is a condition characterised by pain at the back of the head and neck, and extending upwards over the scalp towards the vertex. It is accompanied by tenderness along the great occipital nerve and the posterior branches of the cervical plexus. The scalp is often peculiarly tender and sensitive to brushing the hair, and mental depression is a not uncommon accompaniment. The pain is usually bilateral, and often of a constant dull aching type with paroxysmal exacerbations. Pain is increased on movement of the head.

Brachial neuralgia (brachialgia) is a common form of neuralgia. Complaint is made of intense pain, chiefly in the

shoulder, but also extending down the arm to the tips of the fingers, usually of the fore and middle fingers. It is always increased by movements of the arm. Most of the nerves are tender on pressure. In less acute cases the pain may be limited to the distribution of the circumflex nerve, to the bend of the elbow, or to the nerves about the wrist and palm of the hand.

The presence of a brachial neuralgia, whether affecting the whole limb or only the hand and wrist, may lead to inability to write. The existence of this disability gives rise to the impression that the patient has writer's or other form of professional neurosis. It is more frequently seen in the right arm.

In all cases of persistent brachial neuralgia the shoulder joint should be carefully examined, as osteo-arthritic changes are not uncommonly found in association with it.

Intercostal and abdominal neuralgia. If pain is in the left mammary region, the patient is troubled with the idea of heart disease; while in the abdomen it may lead to the impression that appendicitis or organic visceral disease is the cause of the pain. It is increased by the respiratory movements. If the upper dorsal nerves of the left side are affected, pain resembling that of angina pectoris may be felt along the ulnar border of the arm and hand.

It is usually accompanied by marked tenderness on pressure along the intercostal spaces; but especially to one side of the vertebral spines, in the mid-axillary region, and near the costo-sternal articulations.

It is a common antecedent and sequel of herpes zoster.

The lower abdominal nerves (ilio-inguinal and ilio-hypogastric) are also the seat of occasional neuralgia. The usual tender points are present. It may extend as a dull, aching, boring pain into the testicular region and groin.

Anterior crural neuralgia. Pain in this nerve may occur alone, or as part of a generalised neuralgic affection of the whole limb. It would seem to be especially prone to occur in men from the fatigues of long marches, mountain climbing, or other muscular overaction of the lower limbs. It is sometimes an early symptom of disease of the bones of the dorso-lumbar region of the spine. The nerve is tender to pressure within Scarpa's triangle, and standing and walking are impaired.

Mastodynia is the term given to a painful condition of the breast in women. It occurs during pregnancy, lactation, and in association with disease of the female pelvic organs. It may lead to an obsession or fear of cancer of the breast.

The pain may be severe, and is usually constant with paroxysmal exacerbations. The breast may be red and swollen, and the nipple sensitive and tender. It is relieved by the removal of the associated cause.

Coccygodynia. Neuralgia of the coccygeal nerves is rare. Pain in the coccyx is, however, common. It is a local symptom of a general disorder—such as hysteria or neurasthenia—and is frequently due to a fall upon or injury to the lower part of the spine.

There is marked tenderness on pressure over the coccyx, and pain is also complained of in other parts of the spine, which may be tender. The treatment is that for hysteria. Although the coccyx has been excised, pain may continue after its removal.

Chapter VII

HERPES ZOSTER

Herpes zoster is an inflammatory, often hemorrhagic, condition affecting the posterior root ganglia, accompanied by pain, sensory disturbances, and the presence of a characteristic vesicular eruption in the area of distribution of the affected nerve or nerves. It may be either a primary condition, or symptomatic of interference with the functions of the posterior ganglia from carious or malignant disease of the bones of the spine.

Pathology. The observations of Head and Campbell[1] have shown that the changes in this condition consist of an exudation of small round cells and extravasation of blood within the *ganglia upon the posterior roots.* In the centre of the hemorrhage or inflammatory focus, the ganglion cells and fibres are completely destroyed, while at the periphery the

[1] Head and Campbell, *Brain*, 1900.

cells are swollen, structureless, and stain abnormally. Inflammatory changes are also seen in the sheath of the ganglion.

In slight cases these changes may clear up without leaving any obvious scar; but in the severe forms, regressive changes are observed in the extravasated blood, and the affected focus becomes replaced by fibrous scar tissue.

The changes found in the *posterior nerve roots* correspond to the severity of the ganglionic lesion, and consist of an acute degeneration followed by a greater or less amount of sclerosis.

In the *peripheral nerves* close to the ganglia, acute degeneration of the fibres is seen, with breaking up of the myeline sheaths, especially in the posterior primary divisions of the nerves. In the severe cases marked sclerosis of the nerve may result. In a few cases hemorrhage and inflammation may actually occur in the peripheral nerve. The acute degenerative products are found to be rapidly absorbed and the secondary sclerosis quickly ensues. No changes are found in the anterior nerve roots and motor fibres.

In the *spinal cord*, degenerated fibres corresponding to the affected ganglia, may be found in the posterior root zone.

Similar changes are found in the secondary and symptomatic zosters, arising from implication of the posterior root ganglia in malignant, tuberculous, and traumatic affections of the spine. Herpes is also stated to occur in association with tabes and paretic dementia (Head).

The *skin changes* are also of an inflammatory character and consist of an eruption of vesicles, which usually contain a clear serous fluid, and whose floor is formed by inflamed papillæ. Degenerated nerves have also been found passing towards the areas of affected skin.

An inflammation of the lymphatic glands is also seen in these cases, the axillary glands being affected when the lesion is above the seventh dorsal nerve and the inguinal when it is below this level.

Symptoms. The malady usually arises without any obvious peripheral or central cause. The patient feels ill, suffers considerable malaise and pain, and the temperature is elevated. These symptoms may last for three or four days, after which, in most cases, the characteristic vesicular

eruption appears. In many cases, however, the eruption is preceded for some days by severe local pain and sometimes hyperæsthesia over the distribution of the affected nerve.

The commonest situations are the cervical region, particularly the third and fourth cervical nerves, and from the third dorsal to the second lumbar nerves, especially the third, fourth, and fifth dorsal. The trigeminal and facial nerves (Gasserian and geniculate ganglia) may also be affected.

Fig. 54.—Photograph of a case of herpes zoster.

The eruption is distinctive in character. It consists of groups of vesicles, arising from an erythematous base, occurring in crops and containing fluid. This may be clear, or in severe cases bloodstained; and in others it may be purulent. It appears along the distribution of the nerve root, although not necessarily affecting the whole root area. It spreads with varying rapidity, but appears first and is most severe over the posterior primary, the lateral and anterior branches of the anterior primary divisions. The vesicles may break down and form an ulcer. From the fifth to the tenth day after their appearance, the vesicles begin to dry up and form scabs. In the milder cases no trace may be left, but in the more severe forms white scars take the place of the scabs and persist. (Fig. 54.)

During the period of the eruption the pain may be of a severe

and burning character, and worse at night. In a few cases, especially in old people, it may persist for an indefinite time.

While the rash is present painful sensibility and the severer degrees of heat and cold are abolished in a small portion of the affected area; but in the peripheral parts hyperalgesia may be found. Tactile sensibility is unaffected, and the whole area may even be hyperæsthetic. After the disappearance of the rash there is little or no interference with sensation, except where scarring is present, when some general impairment of all forms of sensation may be found.

A second attack rarely occurs, and a bilateral distribution of the eruption is rare.

The second, third, and fourth cervical nerve root areas are frequently affected together; and occasionally two or more contiguous roots in other parts of the body, although, as a rule, the eruption is confined to one root area.

The **diagnosis** does not present any difficulty.

The **prognosis** as regards recovery from the attack, is good. In a few cases, especially the senile forms, neuralgic pain of a severe character may persist.

The **treatment** consists chiefly in the application locally to the eruption of soothing and sedative ointments and powders—such as oxide of zinc, boracic ointment with cocaine and starch powder. The eruption may be advantageously covered with collodion to protect it from external influences. Internally antipyrin, phenacetin, or aspirin may be given for the relief of pain, which if severe, however, requires morphia.

The application of galvanism along the nerve may be found of use in some cases of persistent post-herpetic pain.

Part VI

DISEASES OF THE BRAIN

Chapter I

THE INTRACRANIAL BLOOD SUPPLY [1]

The brain is supplied by two arteries: the basilar, formed by the union of the vertebral arteries, and the internal carotid artery. The basilar artery gives origin to the posterior cerebral artery, which sends off a small posterior communicating branch to the internal carotid. The internal carotid artery divides into the middle and anterior cerebral arteries. The two anterior cerebral arteries are joined together by the anterior communicating artery, in this way completing the circle of Willis. All these blood-vessels in their course give off numerous smaller arterioles, which pass into the brain and supply its several parts.

Two points in connexion with the vascular supply require mention. First, no anastomoses exist between the arteries supplying respectively the cortex and the deeper structures. There is therefore a hard-and-fast line of demarcation between the superficial and the deep areas. Secondly, the three cortical systems intercommunicate with each other at the confines of their respective areas; but the individual branches of the cortical arteries are end-arteries, and do not anastomose with their contiguous branches.

The simplest method of description is to take each primary artery and refer to its distribution: first upon the cortex of the brain, and secondly, in the subcortical structures. The written account should be read and studied in conjunction with the figures. (Figs. 55–58.)

[1] The account given here is based upon the paper of Dr. Beevor.—*Brain*, 1907.

1. Anterior cerebral artery

(*a*) *Superficial distribution.* This embraces the mesial surface from the frontal pole as far as, or about one inch short of, the internal parieto-occipital fissure, including the whole of the marginal gyrus, the gyrus fornicatus, the corpus callosum, and the orbital surface of the frontal lobe. On the external surface it extends as far back as the middle of the parietal lobe, and as far downwards as the level of the superior frontal sulcus.

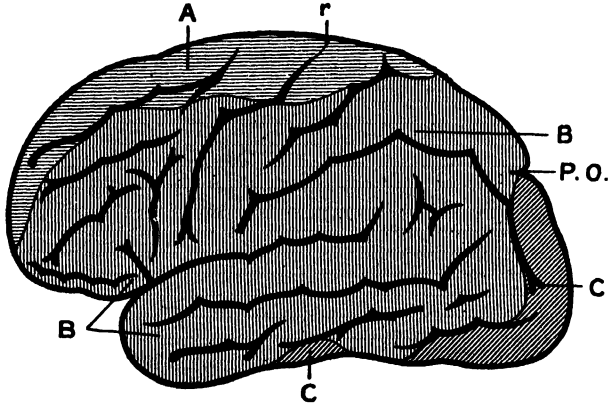


FIG. 55.—The vascular supply of the convexity of the brain. *A* (horizontal lines), anterior cerebral artery. *B* (vertical lines), middle cerebral artery. *C* (oblique lines), posterior cerebral artery. *P.O.*, external parietal-occipital fissure. *r*, fissure of Rolando.

(*b*) *Deep distribution.* This includes the inferior half of the head of the caudate nucleus and anterior limb of the internal capsule, the antero-inferior part of the external and sometimes the second segment of the lenticular nucleus, the floor of the third ventricle, and portions of the anterior commissure and anterior pillar of the fornix.

This artery also supplies the mesial half of the centrum ovale of the frontal lobe, as far back as the caudate nucleus.

2. Middle cerebral artery

(*a*) *Superficial distribution.* The upper limit of the middle cerebral supply upon the convexity is bounded in front by the superior frontal sulcus, and behind by the intraparietal fissure and anterior occipital sulcus.

Its posterior limit varies with that of the anterior border of the posterior cerebral supply, but in most cases it reaches as far as, or just short of, the occipital pole. Its lower limit lies along the middle of the inferior temporal convolution, or the second temporal sulcus. It also supplies the outer half of the orbital surface of the frontal lobe.

(*b*) The *deep distribution* includes the middle and external

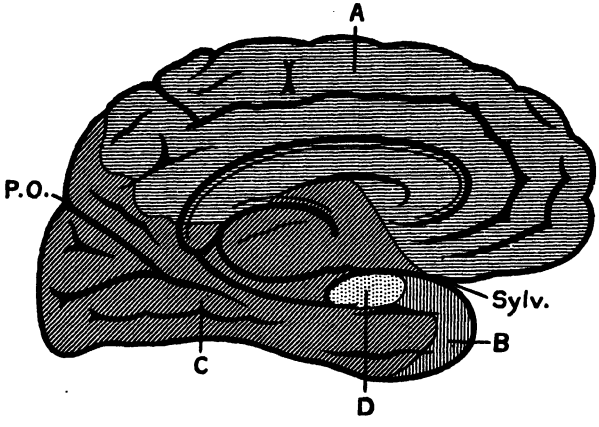


FIG. 56.—Vascular supply of the mesial surface of the hemisphere. The lettering is the same as in fig. 55. *D* (dotted area), anterior choroidal artery.

segments of the lenticular nucleus, the posterior limb of the internal capsule, the remainder of the caudate nucleus, and the whole of the centrum ovale corresponding to the extent of the superficial, or cortical supply. This comprises the outer third of the frontal segment, the external capsule and insula, the Rolandic and parietal segments and the outer and upper half of the occipital segment of the central white matter, and the optic radiations.

3. Posterior cerebral artery

(*a*) The *superficial distribution* extends over the whole of the ventro-mesial surface of the temporo-occipital lobe as far as the internal parieto-occipital fissure, or even to a short distance in front of it. It does not, however, supply the uncinate convolution, which receives a branch from the anterior choroidal artery. It extends round the occipital pole for a distance of a half-inch on to the external surface, but

superiorly may extend as far forwards as the external parieto-occipital fissure or even farther, and abut against the anterior cerebral distribution.

(*b*) *Deep distribution.* Posterior two-thirds of the pes pedunculi, the red nucleus, the posterior half of the optic thalamus, the pulvinar, and the lower and inner segment of the optic radiations.

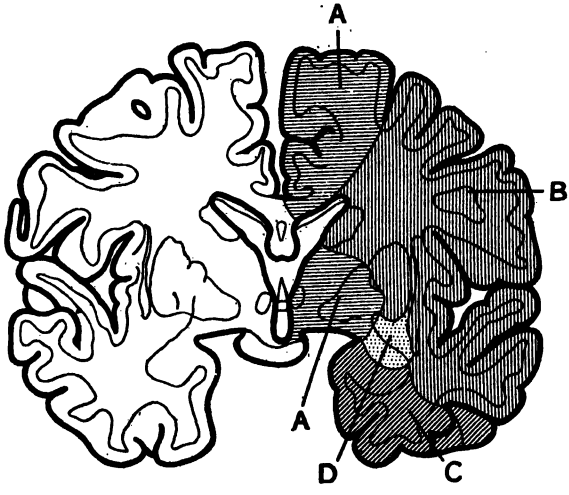


FIG. 57.—Vascular supply of the interior of the hemisphere, at the level of the anterior limb of the internal capsule. The lettering is the same as in figs. 55 and 56.

4. The anterior choroidal artery

(*a*) *Superficial distribution.* This is limited to the uncinate lobule.

(*b*) *Deep distribution.* The anterior part of the optic thalamus, the greater portion of the fornix, the corpora-quadrigemina and the internal and external geniculate bodies; the posterior two-thirds of the posterior limb of the internal capsule, the internal segment of the lenticular nucleus, the retrolenticular fibres of the internal capsule and the origin of the optic radiations.

5. The cerebellar arteries

The cerebellum is supplied by three arteries derived from the vertebral and basilar arteries. The inferior cerebellar

branch of the vertebral artery winds round the medulla oblongata, and supplies the under surface of the cerebellum. The middle cerebellar artery is given off from the basilar, and is distributed to the under surface of the cerebellum. The superior cerebellar artery—a branch of the basilar—winds round the crus cerebri, and is distributed over the superior surface of the cerebellum.

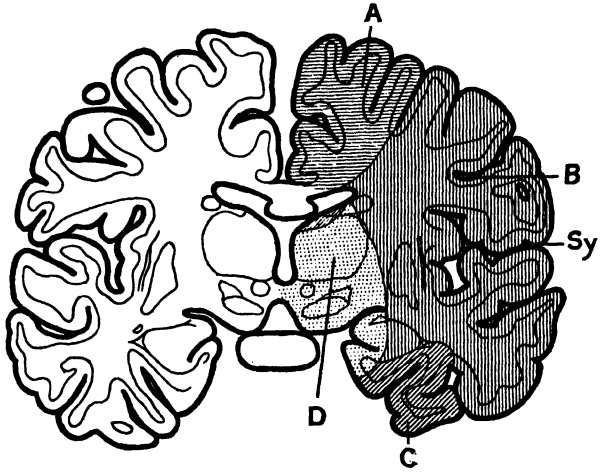


FIG. 58.—The vascular supply of the interior of the hemisphere at the level of the optic thalamus and posterior limb of the internal capsule. The lettering is the same as in the preceding figure.

6. The bulbar arteries

The pons and medulla are richly supplied with vessels derived from the basilar artery, which penetrate its substance both mesially and laterally. Larger branches of the vertebral and basilar arteries also are conveyed into the bulb along the roots of the cranial nerves. These arteries are all end-arteries, and find their way through the bulb to the grey matter of the floor of the fourth ventricle.

7. The venous return

The venous blood from the brain returns by a number of sinuses, situated mainly in the dura mater, to the internal jugular vein. A large number of veins from the convexity

enter the superior longitudinal sinus, which passes backwards to the torcular Herophili. The blood from the interior of the brain, the ventricles, and callosal region returns by the inferior longitudinal sinus, the veins of Galen, and the straight sinus to the torcular. The superior longitudinal and straight sinuses fuse in this locality with the large lateral sinus, which is derived from the cavernous and inferior petrosal sinuses, into which the cerebellar veins also open.

It should be remembered that the intracranial sinuses are also in direct communication with veins from the face and scalp, from the nose and orbit, from the mastoid bone, and from the diploë of the cranial bones.

Chapter II

GENERAL SYMPTOMS OF BRAIN DISEASE

Before entering upon a description of the diseases of the brain, it is necessary to refer briefly to the symptoms of cerebral disease in general, as these depend more upon the seat than upon the nature of the lesion.

The symptoms of cerebral disease are shown by an interference with the functions of the brain in three ways: (*a*) by excess of function (motor convulsion and sensory paræsthesia); (*b*) by diminution or abolition of function (motor and sensory paralysis); and (*c*) by a combination of impairment and excess of function.

In order to comprehend the factors underlying the production of the symptoms of brain disease, it should be borne in mind that the cerebral cortex is the highest centre of the nervous system, that in it are received and perceived the several forms of sensibility, general and special, that it is the centre for voluntary movement, and that it controls to some extent the lower centres. Its integrity is essential for the development and evolution of all mental processes. It is, therefore, obvious that all kinds of brain disorder may, irrespective of the local disturbance due to their situation, lead to interference with the functions of the brain as a whole.

The highest centres of the brain occupy a relatively large area in the grey matter of the cerebral cortex, and are localised to certain well recognised regions. Experimental, histological, pathological, and clinical investigations have permitted the mapping out on the surface of the brain, the position of the centres for motion, sensation, vision, hearing, taste, and smell, as well as more specialised centres for speech and writing. Some of these centres are in juxtaposition, and others are some distance apart.

Cortical lesions. As the centres of voluntary motion and of general and special sensation are situated in the cerebral cortex, cortical lesions will cause disturbance of function, characterised by symptoms of an irritative character, so long as the cortical elements are not destroyed. Cortical lesions are, therefore, characterised by symptoms of irritation—focal epilepsy—either motor or sensory, according to the situation of the lesion; and are accompanied or followed by paralytic symptoms of a temporary or permanent character (hemiplegia, hemianæsthesia, hemianopsia, &c.).

Subcortical lesions. In passing from the cerebral cortex to the brain stem and spinal cord, the tracts conveying impressions to and from the cortex are in closer anatomical association. In the internal capsule, the motor, sensory, and visual fibres are arranged in this order from before backwards (pp. 3, 4). A lesion in this locality may, therefore, give rise to motor, sensory, and visual symptoms of a paralytic character, as nerve tracts, and not nerve centres, are involved. The paralytic symptoms will, however, be wide and extensive, although the loss of power in any one part is not so great as in complete destruction of its cortical centre.

The motor and sensory tracts pass through the *crura cerebri* in their passage to and from the internal capsule and pons. Symptoms of lesion of the crus, therefore, may be hemiplegic paralysis and hemianæsthesia of the contralateral side, with homolateral paralysis of the third cranial nerve.

Unilateral lesions of the *pons*, if high up, may cause hemiplegia with or without hemianæsthesia. If the lesion be situated below the decussation of the fibres to the facial nucleus, the face may not be paralysed.

HEMIPLEGIA

In hemiplegia paralysis is present on the side of the body contralateral to the cerebral lesion. The paralysis is one of movement and not of muscles. The actions of contralateral muscles, which are only used in association with corresponding homolateral muscles, are less impaired than the actions of contralateral muscles acting solely on the contralateral side. This explains the relatively slight impairment of the intercostal and masseter muscles on the contralateral side.

This is probably due to the bilateral representation of bilateral movements in the cerebral cortex and to a connexion between the bulbar and spinal centres of the two sides.

From this it might be supposed that bilateral movements would be impaired on both sides of the body, and such is found to be the case.

A further point of interest is that certain movements, such as respiration, are normally automatic and innervated chiefly from lower centres in the bulb. In addition, voluntary respiratory movements may occur—as in taking a deep inspiration; such movement being innervated from the cerebral cortex. As Hughlings Jackson pointed out, in a patient suffering from recent hemiplegia, during ordinary respiration the paralysed side of the chest expands more than the non-paralysed: whereas if the patient be asked to take a deep breath, the non-paralysed side expands more than the paralysed.

Paralysis of the action of certain muscles may occur upon the same side as the lesion (homolateral), when such action is used to effect a lateral movement towards the contralateral hemiplegic side. Notable examples of this are:—

1. The impairment of rotation of the head to the contralateral side. This movement is chiefly performed by the sterno-mastoid muscle on the homolateral side acting along with other less important muscles on the contralateral side. That the depressor action of the homolateral sterno-mastoid muscle is not paralysed may be demonstrated by making the patient depress his head against resistance. It will be found that the head deviates towards the contralateral side, owing to the weakness of the contralateral sterno-mastoid muscle.

2. The impairment of conjugate lateral movement of the

eyes to the contralateral side. This movement is effected by the external rectus muscle of the contralateral side acting in conjunction with the internal rectus muscle of the homolateral side. In hemiplegia there is no paralysis of the convergent action of either internal rectus muscle.

In a typical case of hemiplegia, the arm, leg, and the lower part of the face are paralysed. The tongue, when protruded, deviates to the paralysed side, and the voluntary movements of the bilateral trunk muscles are also impaired. Thus, when the patient tries to sit up, the umbilicus is drawn towards the non-paralysed side.

The paralysis of the face may be complete for voluntary, emotional, or associated movements. In cases where the lesion is in the neighbourhood of the optic thalamus, emotional movements only may be affected, whereas lesions in other situations may show paralysis of voluntary and associated movements with little or no impairment of emotional movement.

The loss of power is usually greater in the arm than in the leg, and greater in the extremity of the limb than towards the trunk. As a general rule the leg recovers before the arm. Cases in which the leg is more severely paralysed are due to lesions of the motor fibres to the leg; the commonest site of such lesion is the posterior part of the internal capsule. As the sensory and visual fibres are in close relation to the fibres for the leg in this region, hemianæsthesia and hemianopsia are common accompaniments (p. 4, fig. 4).

Associated movement sometimes occurs in the paralysed limb. If a patient with hemiplegia—who is unable to move, for example, the left hand—be made to close the right hand tightly some movement of the paralysed left hand may be observed. Much less frequent, but more striking, is the fact that occasionally a patient—one of whose arms is completely paralysed for voluntary movement—may, when yawning, raise his paralysed hand to his mouth.

Rigidity. Gowers has described three types of rigidity: (*a*) Initial rigidity, which may be present at the onset of hemiplegia and only lasts for an hour or two. (*b*) Early rigidity, which commences within a few days of the onset, and lasts for one to four or five weeks: this rigidity is

FIG. 59.

FIG. 60.

FIG. 61.

FIGS. 59–61 show the face at rest, smiling, and laughing, in a case of right-sided hemiplegia; the lesion being in the neighbourhood of the optic thalamus.

slight in degree and variable. (*c*) Late rigidity or spasticity, which comes on several weeks after the stroke. Although it is present in all the muscles, the limbs tend to become fixed in characteristic positions owing to the greater power of certain groups of muscles. Thus the arm is adducted at the shoulder and flexed at the elbow; the forearm is in pronation, the fingers are clenched, and the thumb flexed and opposed into the palm. The thigh is adducted at the hip, the knee is rigid, the heel slightly drawn up, the foot inverted and the toes pointed.

Later on, structural changes in the muscles and joints may give rise to fixation of the limbs. This is distinct from spasticity, and cannot be overcome by passive movement even under an anæsthetic.

Muscular tone. During the acute stage of hemiplegia, before the onset of early rigidity, there may be a stage of temporary flaccidity. Except during this stage, the muscular tone is increased and with it the corresponding tendon reflexes.

Vaso-motor and trophic changes. For some time after a stroke the temperature of the paralysed side is raised from one to two degrees above the nonparalysed. The paralysed side may not present any vaso-motor changes: on the other hand it may be red, swollen and perspire freely; or it may be cold and blue—the latter appearances being seen in the late stages.

Trophic changes may be absent, or may be slight and consist of a general wasting with dryness of the skin: on the other hand, in such cases as infantile hemiplegia and extensive cortical lesions, marked atrophy of the limbs may be observed.

Disorders of movement. These may be convulsive, tonic or clonic, regular or irregular, or consist of slow mobile spasm, depending on the seat and nature of the lesion. The different varieties are described on pp. 216 and 221.

Sensory symptoms. As with motor paralysis sensory disturbances may be hemiplegic, monoplegic, or even more limited. All forms of sensation may be affected. In cortical lesions the sensory loss may exist without motor palsy and be of limited distribution. In lesions involving the sensory tracts in the lower portions of the brain, motor and visual paralyses may coexist.

Reflexes. On the paralysed side the deep reflexes are exaggerated, the superficial abdominal reflexes are impaired or abolished, and the plantar response is extensor in type.

General symptoms, such as headache, vertigo, and mental impairment are frequent, and are referred to later.

Cranial nerve paralyses, as symptoms of brain disease, have been dealt with in Part IV.

FOCAL EPILEPSY

This term has been applied to cases in which seizures of an epileptic character result from the direct, immediate, or late effect of a lesion of the brain, the membranes, or the skull. This application of the term includes all cases in which fits occur in consequence of a cerebral lesion. In the majority of cases the attacks are of local character and limited extent—Jacksonian epilepsy; in the remainder, epileptic fits in every way resembling those of the idiopathic disease are set up: but it is a common clinical experience to find either a combination of the two forms or a transition type of seizure. Cases showing either of these varieties require careful study—attention being given to the original character of the seizures and to the physical signs. Focal fits happening late in the history of a case may be false localising signs, while generalised epilepsy occurring early may, on the other hand, obscure the local nature of a lesion.

Jacksonian epilepsy may occur in consequence of any organic lesion of the brain, or its membranes. If the lesion is situated so as to press upon or involve the cortex, fits are almost invariable, and are one of the earliest signs. Should the lesion be seated deeply convulsions are usually late in onset, and, although often Jacksonian in type, are more frequently generalised.

It is worthy of notice that in cases of cerebral syphilis, or multiple lesions of the brain, fits with local commencement are common; but there is no constancy in the seat of commencement, the 'discharge' sometimes taking place from one cortical area and sometimes from another.

It may be mentioned that in cases with increase of the

general intracranial pressure—such as hydrocephalus and cerebral tumour—although generalised epileptic seizures are the rule, yet attacks with local commencement may supervene.

Cortical lesions. The fits in focal or Jacksonian epilepsy have certain definite and constant features.

1. They always commence in the same manner in the same part of the body: the initial symptoms of each attack are therefore always the same.

2. The part of the body affected corresponds to the cortical area from which the 'discharge' issues.

3. The march of the fit is in a more or less definite order, in consequence of the overflow of the 'discharge' from the primary focus to adjacent cortical centres.

4. Consciousness is not lost except when the fit becomes general, or arises from lesion in the highest psychical centres.

The nature of the fit varies according to the situation of the irritative lesion. If in the frontal region, the initial symptoms are psychical; if in the motor, convulsive; if in the sensory, paræsthetic; if in one of the specialised centres, visual, auditory, olfactory or gustatory, respectively.

1. *Epilepsy arising from lesion of the motor area.* The cortical discharge results in convulsive movements starting in a particular area of the body. These movements are brought about by clonic contractions of the executive muscle or group of muscles. The contractions commence slowly, but become more rapid, tending towards tonic spasm and passing off gradually by a succession of wider but less frequent clonic jerks.

If the discharge is severe the clonic may pass into a tonic stage, which passes off with slow clonic jerkings occurring at less frequent intervals.

After the fit a condition of exhaustion paralysis results. This is seen in weakness of certain movements, more especially those which were involved most completely in the irritative stage.

The exhaustion phenomena are usually transitory; but a progressive and persistent paresis may eventually ensue as a result of permanent injury to the discharging cortical area.

Focal epilepsy, followed by progressive weakness of the parts affected by the discharge, is therefore characteristic of a progressive cortical lesion—at first small and irritative, later

large and destructive. In certain cases, a sudden onset of paralysis occurs in the 'fit area,' due in most instances to the occurrence of hemorrhage into the primary focus of irritation.

Jacksonian epilepsy may occur in cases in which paralysis already exists, and it is striking that in such cases the fits usually commence by convulsive movements in the paralysed muscles. A cerebral lesion may directly induce the fit, or may by reducing the stability of the cortical centres render them more liable to discharge; and the greater the number of fits, the less stable is the condition of the cerebral cortex.

2. *Epilepsy arising from lesion of the sensory area.* Usually the fit is ushered in by a feeling of numbness—'pins-and-needles,' or 'drawing' of the skin—in a particular area; more rarely by a subjective sensation of movement in some part of the body.

These sensations spread and are usually, though not invariably, accompanied or followed by a motor seizure in the corresponding areas. After the fit, a loss or diminution of common sensation of a transient character is detected over the affected area. Permanent sensory loss is brought about in a manner similar to motor paralysis.

3. *Epilepsy arising from lesion of the special sense areas.* Irritative seizures are rarely highly specialised. In occipital lesions, subjective visual sensations are observed in the corresponding visual fields, and occasionally these are followed by transient blurring or loss of sight. As a rule no permanent impairment of function results from the fits, although such may result directly from the lesion.

Subjective sensations of smell, taste, and hearing are usually bilateral and are not followed by complete loss of function.

4. *Epilepsy arising from lesion of the psychical centres.* In these cases the highest consciousness is impaired or abolished. The patient 'loses himself,' as in 'petit mal' attacks. In other cases automatism is present.

In most cases of Jacksonian epilepsy, there is a tendency for the fit to extend to other regions, so that one side of the body may be involved in the convulsion. When the cortical discharge takes place rapidly, consciousness is often lost, and the fit tends to become generalised, implicating both sides of the body.

APHASIA, OR LOSS OF SPEECH

Language, using the term in its widest sense, may be taken to be the means by which communication and exchange of ideas takes place between individuals. It includes gestures, signs, expression, speech, and writing. Articulate speech is the form of language characteristic solely of man, and is the result of the higher evolution of the human brain.

Before attempting to describe the various disorders of speech, occurring as the result of disease, a general account of the processes of acquisition and evolution of speech in the child is desirable.

It may be mentioned, by way of preface, that the specialised centres subserving speech are developed, in the majority of persons, in the left cerebral hemisphere. This special function of the left side of the brain appears to have been acquired along with right-handedness in the process of evolution.

The child at first commences to recognise different objects by their size, shape, colour, and touch, and learns to distinguish them by the differences which they exhibit. Later on, he associates various attributes with each object. In course of time he hears names applied to the objects, and from their constant repetition and from the observation of what is associated with their use, he begins to understand the meaning of the words before he is able to intelligently reproduce their sound. It may be that at times the child accidentally reproduces a correct word, and it is ultimately, by the perception of the correctness of the sound and by many attempts at its repetition, that at last he not only recognises, but is able to reproduce the word. A similar process attends the acquisition of writing, where signs are recognised and intelligently reproduced.

It is obvious that two fundamental factors are essential for the production of speech: first, perception upon the sensory side, and secondly, expression upon the motor side.

The fundamental sensory functions most intimately connected with speech are audition and vision; but, as will be seen from a further study of this subject, the accurate expression of the motor side of speech depends—at least in its earlier stages—upon the perception of the muscular articulatory movements entailed in their performance. Thus, a child

is unable voluntarily to reproduce a word until it can correlate satisfactorily the sense of movement produced by the correct articulation with the appreciation of its sound. It is by the use of the sense of movement of the lips and tongue, in articulation in association with visual impressions, that the congenitally deaf, or deaf-mutes, learn to speak.

The *motor*, or expressive side of speech, consists of the primary centres in the precentral gyrus for the lips, tongue, vocal cords, their connexions with the bulb, the bulbar nuclei, the motor nerves, and the muscles subserving articulation.

Specialised or higher motor centres, of later development, in which movements associated with speech and writing are solely represented, are situated apart from the motor centres in the intermediate or psychomotor region of the frontal lobe.

The position of these centres is probably in the posterior ends of the second and third frontal gyri—the intermediate precentral area described and figured by Campbell.

The centre, lying within the third frontal gyrus, is known as Broca's area, and is more especially associated with spoken language; while the centre in the second frontal gyrus subserves the expression of written language—the writing centre (fig. 2, p. 3).

The *sensory* or receptive side of speech consists of two parts—the auditory and the visual.

1. *The auditory centre.* Sound is conveyed from the organ of Corti by the auditory nerve and central auditory tract partly to the temporal lobe of the opposite side, and in part to that of the same side, both ears being bilaterally represented in the cortex. The auditory centre is situated in the posterior portion of the first temporal gyrus and in the gyri of Heschl. Of this area, a certain portion—the audito-psychical or the auditory word-centre—is located in the posterior part of the first temporal gyrus (fig. 11, p. 64).

2. *The visual centre.* The primary visual centres are situated in the cortex of the occipital lobe around the calcarine fissure. A higher visual or visuo-psychical centre is described by Campbell as situated upon the external surface of the occipital lobe. A cortical centre for the recognition of written language or visual word-centre is situated in the angular, and partly in the supra-marginal gyri.

The centre for the sense of muscular movement of the

lips, tongue, and vocal organs is situated in the post-central gyrus, as elsewhere described (p. 18).

It is a well-recognised fact that the centres subserving the functions of speech lie on either side of the Sylvian fissure. Around its posterior end and corresponding to the posterior segments of the fissure, both above and below, are the visual and auditory receptive centres; while anteriorly forming partly the upper border of the fissure and partly its base are Broca's area and the island of Reil. Situated within the fissure and supplying the gyri on both sides is the Sylvian artery: thrombotic lesions of which are not uncommon—a fact which explains the frequent association of paralysis of the receptive centres.

Defects of speech arising from cortical or subcortical lesions may be subdivided into the sensory and motor aphasias. These two forms of aphasia may occur separately or in association. Interference with the sensory side or perception of spoken or written language, materially impairs the motor or expressive side, and with it there is usually observed some degree of mental impairment. In his doctrine of aphasia recently propounded, Marie[1] has stated that lesion of the sensory speech-centres and their subjacent white matter (the zone of Wernicke) is the lesion essential to aphasia, and if loss of motor speech is also present, it is accounted for by a subcortical lesion giving rise to anarthria, or paralysis of articulation.

It is, however, necessary to refer to the symptoms which result from lesion of one or other of the cortical centres of speech and their subcortical connexions.

Word deafness. Destruction of the auditory word-centre is followed by word deafness, or inability to understand spoken language.

Although motor speech is not impossible, the effect of destruction of the auditory word-centre is to produce a jargon, which is not, however, recognised as such by the patient.

In minor degrees of interference with the centre, there exists an inability to recall words, or a tendency to use the wrong words (paraphasia). There is also a loss of power of naming objects, although the use or purpose of the object is known, and can be expressed in a roundabout fashion.

[1] Marie, *La semaine médicale*, 1906.

Persons so affected may be able to read aloud, owing to a direct connexion between the visual word-centre and Broca's area.

The inability to name objects, already mentioned, may be the first sign of interference with the functions of the auditory word-centre from lesion of the temporal lobe. It has been found to be the earliest symptom of abscess of this lobe, secondary to suppurative middle ear disease, and it was also observed in a case of subdural abscess, in which no lesion was present within the temporal lobe.

The auditory word-centre has numerous communications with the other speech centres, of which the following may be mentioned with the symptoms resulting from their destruction.

1. With the higher visual centre in the angular gyrus—the audito-visual commissure. Destruction of this tract is followed by an inability to pick out words or figures from print or writing when asked to do so; although a person affected in this way would be able to read aloud.

2. With the lower visual centres in the occipital lobe, by the inferior longitudinal fasciculus: inability to pick out objects named.

3. With the motor centres in the precentral gyrus; inability to repeat spoken words or phrases.

4. With the higher motor centre of the intermediate precentral or Broca's area: inability to reply to spoken language—in other words, conversation would be impossible.

5. With the higher motor centre of the intermediate precentral zone or graphic area: inability to write from dictation.

Word blindness. Destruction of the visual word-centre results in word blindness, or an inability to understand written or printed language. This has received the name of *Alexia*. There is usually also some interference with writing —wrong words being used or written in a wrong way (paragraphia). Although the power of recognising letters may be lost, the recognition of figures may be retained. Inability to read aloud, to write from dictation, or to copy or transfer print into writing are also present.

The visual word-centre is commissurally connected with other centres concerned in speech. The following are the main connexions with their physiological uses.

1. With the auditory word-centre—visuo-auditory commissure. This commissure is of use for the purposes of reading aloud, except in strong 'visuals' in whom, as already mentioned, a direct connexion exists between the higher visual and the higher speech-centre in Broca's area.

2. With the precentral and intermediate precentral centres for writing: the former permits of copying, and the latter of transcribing printed into written language.

Subcortical aphasia. This is the term applied to the effects consequent upon a lesion not of the cortical centres, as above described, but of their subcortical connexions and association tracts. An extensive lesion of this character brings about the most complete form of sensory aphasia.

If the lesion is subcortical to the auditory centre, the symptoms are: inability to understand spoken language, to name objects, to pick out objects or words named, or even to speak, with a possible exception in the case of a strong 'visual.'

If the lesion is subcortical to the visual word-centre, the patient cannot understand written or printed language, and he can neither read aloud, write nor copy. Although he could probably name objects, he would be unable to write the name or to recognise it when written. This lesion is associated with homonymous hemianopsia to the opposite side.

Motor aphasia. Motor aphasia consists of inability to express language in speech or writing, with retention of the power of understanding what is said or written. The executive centres for speech are situated in the intermediate precentral zone: that for articulatory speech in Broca's area, and that for writing in the posterior end of the second frontal gyrus. Destruction of the former may result in a loss of power of movement for articulatory speech without coexistent motor paralysis of the muscles used in speech; of the latter in a loss of the power of writing spontaneously.

In this connexion reference may again be made to the recent observations of Marie, who has advanced the view that an isolated lesion of Broca's convolution may be unassociated with motor aphasia, and that when this symptom exists, the lesion is more extensive, and extends backwards towards the sensory perceptive centres.

As clinical evidence, however, of the existence of these centres it may be pointed out that, in certain cases of tumour

of the left frontal lobe, fits have been observed in which a complete temporary loss of speech without loss of consciousness occurred. Written and spoken commands were understood, and there was no paralysis of voluntary movements of the face, tongue, or palate. During the seizure, the patient was able to signal for assistance and, later, to record accurately the sensations experienced during the attack. A further example, pointing to the existence of a specialised speech-centre, was that of a child, who for about six months had been able to speak. After a generalised epileptic fit, permanent loss of motor speech ensued without anarthria, motor palsy, or sensory aphasia. That this was not due to lesion of the lower motor centres was proved by the ability to move the lips and tongue, and by imitation to produce words.

Commonly, the lesion giving rise to aphasia involves not only Broca's convolution, but its connexions with the motor centres in the precentral gyrus and its subcortical fibres. In these cases aphasia with motor paralysis is the result. In subcortical lesions of the left frontal lobe and adjacent parts an inability to speak is accompanied by a right-sided hemiplegia. In these cases there is no true aphasia, but anarthria or paralysis of articulation.

The following is the method of examination of a case of aphasia according to Bastian's scheme:—

1. Ascertain whether the patient is right- or left-handed.
2. What is the state of his education as regards reading and writing?
3. Ascertain the condition of hearing and of vision.
4. Does he understand language—whether spoken, written, or printed?
5. Has the patient spontaneous speech—articulatory, or graphic? If so, what is its nature? Does he use wrong words?
6. Can he repeat words spoken to him?
   Can he read what he has written?
7. Can he copy?
   Can he transfer print into writing?
   Can he write numerals?
8. Can he read aloud?
   Can he write from dictation?
9. Can he name objects, letters, or numerals?

APRAXIA

The term apraxia should be restricted to that condition in which a patient, without obvious motor, sensory, or mental impairment, is unable to apply to its proper use an object which he can name and whose uses and characteristics he is able to describe. It is a psycho-motor defect.

The term has been loosely applied to cases in which impairment of intelligence and obvious mental defect have been present; but pure cases have been described in lesions involving the frontal lobes and anterior part of the corpus callosum.

Chapter III

VASCULAR LESIONS

Arterio-sclerosis is a condition in which there is weakening of the muscular and elastic tissues of the arteries with compensatory fibrous thickening of the intima. These changes may be general or local, and render the blood-vessel rigid, calcareous, and tortuous. The lumen of the artery is narrowed.

Etiology. Arterial degeneration is an invariable sign of senile decay, and is therefore found as a natural phenomenon of advancing years, but in certain families a tendency to early vascular degeneration is observed. Under fifty years of age it is more frequent in males than in females, but after this age the sexes are equally affected. Certain diseases—such as gout, rheumatism, and arthritis—favour its development. It is a secondary effect of renal disease. The toxic causes of vascular degeneration are mainly alcohol and lead poisoning. Meat eaters are more liable to it than those who refrain from this food. It is especially prone to occur in those whose occupation requires continuous strain or exertion.

Syphilis is one of the chief causes of arterial degeneration. It may affect the vessels either generally or locally, the commonest form being a localised thickening of the inner coat—syphilitic endarteritis. Degenerative changes may

also occur in the muscular fibres and in the elastic lamina, which becomes split up and separated by a growth of new cells. In more acute cases, periarteritis with leucocyte infiltration is present, and in the larger vessels the vasa vasorum may become obliterated, leading to further degeneration. There is little tendency towards calcification of the arteries.

Cerebral lesions of vascular origin are produced by (1) a cutting off or diminution of the blood supply, and (2) the rupture of an artery.

The ultimate effect of vascular disease upon the cerebral tissues depends upon two factors: first, the degree of the vascular changes, and secondly, the state of the cardio-vascular system.

In every case of vascular disease there is—at least in theory—a state of arterial blood pressure at which the circulation may be maintained without the occurrence of either thrombosis or hemorrhage.

In general terms, the greater degrees of vascular degeneration, if associated with a low blood pressure, favour thrombosis, or blocking of an artery; and if the blood pressure is high, rupture of an artery or hemorrhage.

Vascular lesions occurring in infancy and childhood differ from those of later life both in histological features and clinical characteristics. They are therefore described separately in the chapter on the Cerebral Paralyses of Infancy and Childhood (p. 211).

In adults the cutting off of the blood supply may be either partial or complete.

PARTIAL OCCLUSION OR EVASCULARISATION

The pathological condition underlying this is widespread vascular degeneration. The arteries show extensive arteriosclerosis, and on examining the brain even the arterioles are seen to stand out prominently. There is neither softening nor hemorrhage, but occasionally the perivascular tissue is faintly tinged. Round the vessels some rarefaction of the cerebral tissues or a definite increase of the neuroglia is observed.

The nerve elements, cells as well as fibres, show degenerative changes. The degeneration of the nerve fibres is relatively greater than that of the cells, and is specially

conspicuous in the region of the basal ganglia. These changes are probably brought about by a failure of nutrition acting directly upon the nerve fibres, and indirectly through their cells of origin. The cell changes consist of chromatolysis and atrophy (Buzzard and Barnes [1]).

Symptoms. There is a gradual and progressive mental impairment, with loss of emotional control. As time goes on the facial expression becomes more or less fixed, resembling that of paralysis agitans. General spasticity slowly develops in association with muscular weakness of general distribution and slight degree. The deep reflexes are exaggerated, but only in the later stages do definite signs of pyramidal degeneration show themselves in extensor responses and ankle clonus.

The **differential diagnosis** has to be made from paralysis agitans without tremor, and pseudo-bulbar palsy. In the early stages the facial expression, and the slight degree of spasticity combined with muscular weakness, present a superficial resemblance to paralysis agitans; but in paralysis agitans, mental impairment and emotional instability are not found. In the later stages the extensor plantar responses put the diagnosis of paralysis agitans out of court.

It differs from pseudo-bulbar palsy by the absence of a history of slight 'strokes,' by its progressive character, its symmetrical distribution, the relatively slight degree of muscular weakness proportionate to the spasticity, and by the postponement of organic changes in the reflexes until the late stages.

Complete occlusion. This may be due to: (1) *Thrombosis*, which consists of the formation of a clot *in situ* obstructing the blood stream, and finally occluding the artery. (2) *Embolism*, or the blocking of a vessel by a clot carried from a distance.

CEREBRAL THROMBOSIS.

This may arise from: *(a)* Vascular degeneration, either syphilitic or atheromatous. Syphilis is the common cause in younger people and between the ages of thirty and forty

[1] F. Buzzard and Barnes, *Review of Neurology*, 1906.

years, and atheromatous degeneration from the age of fifty-five onwards. (*b*) Enfeebled circulation with low blood pressure in senile cases, and in cases of cardiac failure. (*c*) Abnormal states of the blood favourable to clotting, such as anæmia and puerperal conditions. (*d*) Combinations of the above causes. (*e*) Pressure on blood-vessels from without, obstructing the flow of blood—as in tumours, gummata, &c.

In a series of 25 cases of thrombotic softening submitted to post-mortem examination, 20 showed well-marked arterial disease; of these, 9 had granular kidneys, 8 were syphilitic, and 3 were alcoholic. In 5 the blood-vessels were stated to be normal; in 2 of these thrombosis was associated with acute rheumatism, and in 1 with the puerperium.

Situation of cerebral softening. Some regions corresponding to areas of arterial distribution are more frequently affected by softening than others. A perusal of the cases submitted to post-mortem examination showed that the basal ganglia, more especially the lenticular nucleus, with the closely related internal capsule, were the structures most frequently involved. Next in frequency came the caudate nucleus, optic thalamus, and adjacent parts of the centrum ovale and corona radiata. The frontal or anterior segment of the centrum ovale was more commonly involved than the occipital or posterior segment. In marked contradistinction to what was seen in hemorrhage, the temporo-sphenoidal lobe was frequently the seat of thrombotic softening. Next in order of frequency were the pons, cerebellum, midbrain, and the corpus callosum.

The extent of the softened area depends upon the size and distribution of the affected vessel, and the area of collateral circulation. When softening occurs in non-vascular parts of the brain, there is simple necrosis of the white matter with formation of fatty débris; if in a vascular area, it is red in colour owing to the regurgitation of blood into the softened zone. Yellow softening is merely a later stage of red softening, in which changes have taken place in the blood pigment. Where the vascular occlusion has been only partial, or after the establishment of collateral circulation, a slow degeneration of the myeline sheaths occurs with secondary proliferation of the neuroglia. This has been spoken of as grey softening, but is really sclerosis.

Symptoms of thrombosis. General symptoms due to the underlying arterio-sclerosis may be present for some months before the stroke. These are alterations in temperament and mental condition, failure of memory, irritability, lethargy, and drowsiness, often accompanied by slight general spasticity and articulatory difficulty. Focal symptoms—such as numbness or transitory paresis—are usually referred to the region of the body subsequently affected by the stroke, and are dependent upon minute thrombotic occlusions.

Degenerative vascular changes and hemorrhages in the fundus oculi frequently precede the onset of the cerebral thrombosis by some months.

The *premonitory* symptoms may usher in the stroke by a few hours, and consist of headache, giddiness, mental dullness, and an increasing sleepiness and lethargy.

The onset of thrombosis may be (*a*) gradual, (*b*) sudden without impairment of consciousness, or (*c*) sudden with loss of consciousness.

(*a*) The onset may be gradual, unattended by any loss of consciousness, and consist merely of an increase of the paretic or paræsthetic premonitory symptoms, already mentioned.

(*b*) In a large number of cases the onset of paralytic symptoms is more or less sudden and incomplete with preservation of consciousness, but followed some hours later by a further increase of paralysis associated with loss of consciousness. Instances of this type of thrombosis are seen in those cases in which during the evening a patient complains of unilateral numbness and paresis, but subsequently loses consciousness, and is found hemiplegic in the morning.

(*c*) There are other cases, in which the onset is sudden with initial loss of consciousness, and attended sometimes by unilateral or bilateral convulsions.

The loss of consciousness in cases of thrombosis is not so sudden or constant a feature as in cerebral hemorrhage. In thrombosis, also, there is usually definite evidence, or a history of previous paretic or hemiplegic symptoms, and coma is rarely so profound even when prolonged. The exception to this is found in those cases in which large vessels, such as the middle cerebral artery, become blocked, when delirium and restlessness precede the onset of coma.

The *focal* symptoms are those arising from occlusion of particular arteries.

(*a*) *Internal carotid artery.* The symptoms are usually profound and extensive, causing hemiplegia, coma, and death.

(*b*) *Middle cerebral artery.* If the artery is blocked at its source on the left side, motor and sensory aphasia, with complete right-sided hemiplegia and hemianæsthesia, ensue. If, however, thrombosis is limited to the first branch, there are motor aphasia and paralysis of the face and tongue, with weakness usually of the arm; and occlusion of the second branch produces the brachio-facial type of hemiplegia. If the Sylvian branch is blocked beyond the fissure of Rolando, slight hemiplegia with a well-defined word-blindness and hemianopsia are present, and sometimes word-deafness.

(*c*) *Posterior cerebral artery.* Occlusion of this artery is followed by sensory symptoms, chiefly hemianopsia and hemianæsthesia, and sometimes by tremor of the limbs on the opposite side.

(*d*) *Basilar artery.* The symptoms of occlusion of this vessel are bilateral paralysis of the limbs and articulatory paralysis. If the lesion is towards the termination of the artery, there is a third nerve paralysis with crossed hemiplegia; if the occlusion is in the middle portion of the artery, the symptoms are facial paralysis, palsy of the fifth nerve, and of the lateral conjugate movements of the eyes.

(*e*) Although the area supplied by the *anterior cerebral artery* is frequently the seat of softening, symptoms resulting from its occlusion are not distinctive, but are probably psychical in character, and of the nature of progressive dementia.

Prognosis and course. In syphilitic cases the prognosis, both as regards the stroke and the subsequent duration of life, is favourable; in our series life was prolonged from two to ten years. In atheromatous cases the duration of life is less likely to exceed two years, but may be prolonged three or even five years. In both series the existence of renal disease or hypertrophy of the heart renders the prognosis much less favourable, the subsequent duration of life being rarely more than one year. Recovery from the immediate effects of the stroke is good, except in cases where consciousness is

lost for more than forty-eight hours, although prolonged unconsciousness is not so unfavourable as in hemorrhage.

If no return of motor power occurs within two or three weeks of the onset of paralysis, the outlook as regards recovery from the paralysis is unfavourable, as softening in the area of the thrombosed artery ensues with permanent paralysis, rigidity, and contracture.

Treatment. (*a*) *Immediate treatment.* In the syphilitic cases stimulation is indicated if the blood pressure is not high. If there is a previous history of thrombotic attacks, stimulants ought to be prescribed with care, and their administration guided by the state of the blood pressure, as in such cases the vessels at the seat of old thromboses are liable to rupture from sudden increase of arterial tension.

As soon as the diagnosis is made, a course of anti-syphilitic treatment by mercury and the iodides should be commenced.

In the atheromatous cases, if the blood pressure is low, and there is no history of previous thrombosis, stimulants may be given.

If there is associated renal disease and cardiac hypertrophy, stimulants should be avoided entirely, or only prescribed in the form of mild diffusible drugs. In these cases the injudicious giving of stimulants, such as alcohol, strychnine, and digitalis, has resulted in fatal cerebral hemorrhage. The indiscriminate use of purgatives is to be avoided in most cases.

(*b*) *After treatment.* Attention should be devoted to the condition of the bladder, the mouth, lips, and tongue; and to the skin, as bed-sores may form. The diet should be light and nutritious. As regards the paralysis, passive movements should be prescribed several times daily after the first week. The tendency to rigidity and flexor contractures is overcome by passive movements and massage, combined with electrical applications to the extensor groups of muscles. The patient should also be encouraged to move the limbs, and when possible to re-educate the hand in the finer movements of writing, &c. Adhesions at the joints may require to be broken down under anæsthesia, and inflammatory changes are prevented or minimised by the local application of hot-air or radiant-heat baths.

CEREBRAL EMBOLISM

In embolism the obstruction of the blood stream is produced by a clot or foreign body carried from a distant part. Such a clot must of necessity be derived from some portion of the circulatory apparatus between the lungs and its place of lodgement.

Emboli are usually derived from the heart, in consequence either of a cardiac clot or an endocardial lesion. Occasionally a portion of a cerebral thrombus is detached and lodged in a distal blood-vessel. Septic conditions may also give rise to emboli, especially in the small arteries, and lead to acute multiple softenings, which may be purulent.

In embolism an acute œdema of the cerebral tissue around the infarct takes place, but the later changes are identical with those described under thrombosis.

It usually occurs in young people, either during an attack of acute endocarditis, or may supervene at any time afterwards from the detachment of a fragment of fibrous material.

Symptoms. The onset is sudden, and is attended by loss of consciousness, which is rarely profound, more or less transient, and depends in most cases upon the suddenness of the shock. Convulsions frequently occur. Paralysis may or may not be present, depending upon the situation of the blocked artery. Usually a small artery is affected, so that paralysis need not be extensive. On the other hand, a large artery may be blocked, with extensive paralysis; this, however, is a rarer condition. The extent and severity of the paralysis may increase slightly within twelve hours after the onset of the symptoms, owing to the local œdematous conditions around the embolic area. Focal symptoms depend upon the vessel occluded, as described under thrombosis (p. 191).

The temperature depends mainly upon the associated causal condition, but may be raised for a short time after the stroke; the respirations are not materially altered. Examination of the heart demonstrates the presence of valvular disease or an active endocarditis. The blood-vessels are normal, but evidence of infarcts in other organs—such as the spleen and the kidneys—may be present.

The **prognosis** depends upon the causing condition. If

occurring during the course of an acute endocarditis, ulcerative valvular disease, or pyæmia, the prognosis is unfavourable. In the absence of acute maladies, such as the above, the outlook is favourable, as recovery may take place, especially in those cases in which little or no paralysis is present.

Treatment. The general principles are the same as for thrombosis, more particularly as regards the administration of stimulants. Rest in bed, the avoidance of all unnecessary movements, and attention to the bladder and bowels, are important. The general treatment is also to a large extent that of the causing condition.

CEREBRAL HEMORRHAGE

Two conditions are accessory to the production of cerebral hemorrhage: (*a*) a structural weakening of the blood-vessel, and (*b*) a blood pressure sufficiently high to rupture the weakened vessel. The latter condition is the determining factor, and, as far as our knowledge goes, there is no state of continued high blood pressure without definite pathological changes occurring in the arterial vascular system. But however essential a high blood pressure may be in the production of cerebral hemorrhage, rupture of a normal artery, whose walls are well supported, is wellnigh impossible. Thus it comes about that in the study of cases of cerebral hemorrhage, evidence of arterial degeneration is never lacking.

The weakening of the artery is due to structural changes in its walls, leading to loss of elasticity. Where such a condition exists, a temporary increase in the blood pressure may of itself suffice to cause a rupture. This increase is, as a rule, abrupt and profound, and may be due to sudden emotional states or excessive physical strain. Extensive vascular disease, with a corresponding and compensatory hypertrophy of the heart, rarely, if ever, results in cerebral hemorrhage without the presence of the exciting causes just mentioned.

The flow of the blood stream through the narrowed and roughened arterial lumen is retarded, and may eventually be arrested. Such a condition is most liable to occur in the smaller arterioles or end-arteries, and leads to malnutrition and softening of the surrounding brain tissue. To the

original weakness of the vascular system is added the additional weakness of the supporting tissues—a combination which favours the occurrence of rupture and hemorrhagic effusion. In such cases the blood pressure, which is alone not sufficient to cause rupture of a well-supported though degenerated artery, may readily induce it in one surrounded by badly nourished or softened tissue. For example, a patient with atheromatous blood-vessels and hypertrophy of the heart is laid aside by illness. In consequence the blood pressure falls, and indefinite symptoms of minute vascular thromboses—such as paræsthesia, paresis, or mental alterations—present themselves. As the general condition improves the thrombotic symptoms pass away, but the blood pressure returns to its original state, and symptoms of cerebral hemorrhage may suddenly supervene. In such a case no further exciting cause of arterial rupture may be detected.

We find that out of 22 cases of cerebral hemorrhage submitted to post-mortem examination, either atheromatous or syphilitic disease of the cerebral blood-vessels was present in all; 9 (or 40 per cent.) were cases of primary hemorrhage, and all had granular kidneys and hypertrophy of the heart; 13 (or 59 per cent.) had a history of previous thrombotic occlusions. Of those, five had syphilitic arterial disease without cardiac hypertrophy or renal disease, and the remainder were cases of atheromatous degeneration.

Site of hemorrhage. Hemorrhage may occur in any part of the brain, but there are certain situations where it is more common. These are: the internal capsule, the lenticular nucleus, and the optic thalamus. The arteries which rupture in this position are the lenticulo-optic and lenticulo-striate branches of the middle cerebral artery.

Next in frequency comes the centrum ovale—the seat of hemorrhage being more common in the frontal segment, from rupture of the anterior cerebral artery, than in the occipital segment. When hemorrhage occurs into either the basal ganglia or the adjacent centrum ovale, it is prone to rupture into the lateral ventricle. Hemorrhage into the ventricle, therefore, is not uncommon.

Hemorrhage into the temporo-sphenoidal lobe, cerebellum, corpus callosum, and midbrain is rare.

Meningeal hemorrhage (non-traumatic) is found in association with acute rheumatism, endocarditis, and the puerperium, the arteries being healthy.

Effects of hemorrhage. In most cases of cerebral hemorrhage blood is effused locally into the brain substance, the subdural space, or the ventricles, where it forms a clot, which acts like a foreign body or tumour within the brain. The experiments of Leonard Hill[1] have shown that such a clot may cause translocation of the brain as a whole, while producing a local compression of that part of the brain into which the hemorrhage has taken place.

Hill's observations point to the symptoms of cerebral hemorrhage being those of compression produced by cerebral anæmia. The first effect of a hemorrhage is to obliterate the capillaries and veins in its immediate neighbourhood, and to raise the intracranial pressure there to the level of the arterial pressure. In the adjacent parts, where the blood-vessels are not completely obliterated, the pressure will be less high, and in the most distant parts of the brain—such as the cerebellum and bulb—the intracranial pressure may be normal. The major symptoms of apoplexy will therefore only arise when the bulbar capillaries are obliterated. For this reason a comparatively small subtentorial hemorrhage will prove fatal, while a much larger effusion in the cerebral chamber is necessary to cause death.

Hill has pointed out that the cause of death in cerebral compression is bulbar anæmia, with (1) primary failure of respiration and secondary failure of the heart and vaso-motor mechanism; or (2) primary failure of the vaso-motor centre with secondary failure of the respiration and the heart.

Should the patient survive the immediate effects of the hemorrhage, there will be in the cranium a localised foreign body, destroying the functions of a certain portion of the brain. Surrounding this is a compressed zone in which the circulation has ceased, and outside this a less compressed area in which the circulation continues with difficulty. Any increase of the arterial pressure will, under these conditions, increase the compression, and so lead to anæmia of other vascular areas. This point is of great practical importance

[1] Leonard Hill, *The Cerebral Circulation*, 1896.

in the treatment of cerebral hemorrhage; as lowering of the arterial pressure by means of venesection or local depletion would seem to be the procedure indicated on physiological grounds.

Etiology. Cerebral hemorrhage is more common in men than in women, their occupation and habits predisposing them to vascular degeneration. We found it most frequent between the ages of forty-seven and fifty-five. In those with a syphilitic history the age at onset was a few years earlier, probably owing to the presence of previous thrombotic softenings. In the older cases the underlying atheroma had prepared the way by antecedent softening.

The predisposing causes are: cerebral softening, atheroma, renal disease, and cardiac hypertrophy; syphilitic endarteritis; alcohol, lead, and gout; and blood states—such as purpura, pernicious anæmia, and leucocythemia.

The exciting factors are: physical efforts—such as straining at stool, coughing, vomiting, or hurrying; and emotional states—such as rage, fright, anger, shock, and anxiety.

Symptoms. (*a*) *Premonitory symptoms.* A sense of fullness and throbbing in the head, vertigo, headache, and epistaxis are occasionally met with as premonitory symptoms of impending hemorrhage. These ought to be distinguished from the slight mental impairment, paresis, spasticity of the legs, and dysarthria, which are common antecedent symptoms of cerebral thrombosis and hemorrhage, and are due to the underlying arterio-sclerosis.

(*b*) The *mode of onset* differs according to the situation of the rupture, and to the severity and rapidity of the effusion; but as a general rule consciousness is lost at once if the hemorrhage is sudden and extensive. Convulsions, often one-sided, may usher in an attack when the lesion is meningeal or cortical; but larger hemorrhages within the brain may also be attended by generalised convulsion.

(*c*) The *general symptoms* vary according to the size and character of the effusion. If the effusion is small and circumscribed, the patient is suddenly seized with a feeling of faintness or weakness, the speech becomes slurred, and paresis or numbness may develop on one side of the body. He is forced to sit down, or may fall, the hemiplegic weakness

increases in intensity, dullness and drowsiness supervene, consciousness is lost, and coma ensues.

If the effusion is larger and more rapid in onset, the patient suddenly falls down unconscious, and may remain so for one or more days. With the recovery of consciousness the reflex actions of the pupil and of swallowing return, and unilateral flaccid paralysis is detected.

On the other hand, the onset of the symptoms may be of a slow and *ingravescent* type. In these cases the commencement of the seizure is prolonged, paralysis gradually develops, and consciousness, at first impaired, rapidly becomes lost. During the early stages the paretic may be distinguished from the non-paralysed side by the exaggeration of the deep and the absence of the abdominal reflexes, and the presence of an extensor plantar response. The pupil on the hemiplegic side is usually the larger. The temperature is normal or subnormal, the pulse slow, firm, and incompressible, and the respirations slightly increased. The coma eventually deepens, the limbs on both sides become flaccid, the deep reflexes disappear, and finally the pupillary light reflex is lost. A rising temperature and a rapid, irregular pulse, associated with slow, cyclic, or Cheyne-Stokes respiration and wide, dilated pupils, becoming pin-point in size, complete the clinical picture.

When hemorrhage ruptures into the ventricle, the loss of consciousness is sudden and complete, and may be accompanied by convulsion and vomiting. The limbs are flaccid, the breathing stertorous, the face flushed, and the cheeks are puffed out at each expiration. Coma is profound, and the reflexes are lost—the light-reflex of the pupil as well as the deep and superficial reflexes. The breathing becomes slow and irregular—6 to 14 per minute; often more or less cyclic, three or four respirations coming together, and followed by a pause, or of the Cheyne-Stokes type. The pulse, at first full and slow, rapidly becomes feeble and irregular—120 to 150 per minute. The pupils, widely dilated at the outset, become contracted. The temperature shows an initial depression, but rapidly rises to 104°–107° F., and death soon ensues without return to consciousness.

Symptoms of compression. With the stroke the patient falls down unconscious, and passes into a state of coma. The

muscles are relaxed and flaccid, all reflex action is abolished, and urine and fæces may be passed involuntarily. The breathing is laboured and stertorous, sometimes cyclic or Cheyne-Stokes in character. The pulse as a rule is slow and incompressible. During this stage it may be impossible to tell which is the paralysed side.

If the coma is less deep the pupils react to light, and the reflex act of swallowing may be accomplished. The head and eyes are turned to the side of the cerebral lesion (paralytic deviation), reflex action returns on the non-paralysed side, and on raising the paralysed arm and leg they will be found to be flaccid and powerless, while those on the non-paralysed side do not show the same degree of flaccidity. If the patient remains in a state of coma bed-sores may form, and death occur. In most cases where some recovery of consciousness occurs within the first twelve hours, there is a short period, lasting for one or two days, in which the temperature rises, the general condition appears to be worse, consciousness is again blunted, and the limbs on the paralysed side are noticed to be rigid. This is 'early rigidity,' and is a transitory symptom.

During the next few days the general condition improves, so that the extent, degree, and quality of the paralysis become defined. In cerebral hemorrhage the initial paralysis is in excess of the permanent, owing to pressure of the effused blood upon adjacent nerve tracts, and indirectly on other portions of the brain. These pressure effects are reduced with the absorption of the clot.

Pontine hemorrhage. Initial loss of consciousness is sudden and profound, should the effusion be large. Vomiting and convulsions, usually bilateral, are sometimes present. There is complete flaccid paralysis with abolition of the reflexes. The pupils are contracted to the size of pin-points. Respiration is often markedly irregular, and may cease suddenly —sometimes before the pulse stops. The temperature, at first depressed, rises rapidly to hyperpyrexia, and death may occur within a few hours, or even within a few minutes, of the onset of the first symptoms.

Multiple hemorrhages of small size may also occur in the pons. Their onset is unattended by loss of consciousness; the palsy of the limbs may be on one or other, or both sides;

the articulation is defective and nasal in character, and swallowing may be impaired. The picture presented in these cases is a form of bulbar palsy, forming one of the types of 'pseudo-bulbar' paralysis.

In addition to these symptoms, paralysis or paresis of several cranial nerves may be observed. The nerves commonly involved are the fifth, sixth, seventh, ninth, tenth, and twelfth. Paralysis of the higher cranial nerves may be found upon one side, with crossed motor and sensory paralyses of the body and limbs, and in some cases paralysis of conjugate lateral movement of the eyes may be noticed.

Cerebellar hemorrhage. Sudden and extensive hemorrhage into the cerebellum is accompanied by unconsciousness, but usually unattended by hemiplegic weakness unless the pons is also affected. The vomiting is specially characteristic, and often persistent. Skew deviation of the eyeballs, and a tendency to rotate to the side of the lesion, may be present. Cerebellar hemorrhage may rupture into the fourth ventricle and cause death within a few hours. In these cases sugar is present in the urine without the acetone reaction.

Small hemorrhages limited to the cerebellum are unaccompanied by loss of consciousness. The patient is seized with vertigo and vomiting, which are aggravated with every movement of the head. Skew deviation of the eyes may be a transient symptom. If the lesion is limited to one lateral lobe, a staggering gait with a tendency to fall to the side of the lesion and coarse nystagmoid jerkings of the eyes on conjugate movement towards the affected side are observed. Ataxia of the limbs on the side of the lesion is well marked.

If the hemorrhage takes place into the middle lobe, opisthotonos may occur; but in the later stages the gait is unsteady, with a tendency for the patient to fall forwards or backwards. Coarse nystagmus is also present on looking to either side, and ataxia—especially of the trunk and lower limbs.

Meningeal hemorrhage. This variety of intracranial hemorrhage may be due either to traumatic or non-traumatic causes. Of the latter, acute rheumatism, endocarditis, and the puerperium are the most common. Vascular degeneration is a rare cause of hemorrhage in this situation.

The patient usually complains of sudden and severe pain in the head, calls out, and falls down convulsed. Sometimes the convulsion is limited to one side without loss of consciousness; but if the effusion is extensive consciousness is lost, and coma is induced, which may persist, deepen, and rapidly prove fatal with respiratory and cardiac failure and hyperpyrexia.

If the patient survives the persistence of coma for more than twenty-four hours, the chances of recovery increase, as the hemorrhage may be arrested by the pressure of the clot upon the ruptured vessel.

In other cases the initial hemorrhage may be circumscribed, so that the symptoms consist only of slight hemiparesis of the limbs opposite the lesion. An effort of exertion, or alcoholic stimulant taken at this time, may favour the recurrence of hemorrhage with rapid loss of consciousness.

In many cases delirium, mental confusion, and dementia are also present.

Hemorrhage into the basal meninges proves rapidly fatal. The pupils become pin-point, coma deepens, the temperature rises to hyperpyrexia, the pulse is rapid and feeble, and death results from respiratory failure.

In traumatic cases, if the hemorrhage is extradural and operation for its removal adopted early, the outlook is good; put if the hemorrhage is primarily basal, death occurs in a few hours.

In a few cases where the effusion is limited by adhesions between the dura mater and the skull, the prognosis is more favourable, especially if operation is undertaken for the removal of the clot.

The outlook in cases of intradural hemorrhage is most unfavourable. In basal cases and in those arising from rupture of an aneurism the fatal termination is rapid. The early recognition of ingravescent hemorrhage, whether arterial or venous, and the adoption of surgical measures, may save life.

Prognosis. A prognosis has to be given upon three points:—

1. *The immediate prognosis as regards life.* The following are unfavourable features in a case of apoplexy: (*a*) Deep coma prolonged over twenty-four hours; (*b*) cyclic or

Cheyne-Stokes respiration persisting for more than six or eight hours; (*c*) severe initial depression of temperature, or a rapid pyrexial rise after an initial depression; (*d*) a bilateral distribution of the paralysis; (*e*) the presence of complications—such as pulmonary congestion, severe renal or cardiac disease, or the occurrence of bed-sores.

2. *Future prospect of life.* This is always less favourable than in cases of thrombosis. If there has been a history of antecedent thrombotic attacks, especially when associated with renal disease and hypertrophy of the heart, the duration of life rarely exceeds one year, and may be shorter. If the stroke has been due to a violent strain or emotion without previous symptoms of cerebral vascular disease, careful regulation of the patient's habits, and especially of the blood pressure, renders the prognosis less grave, and life may be prolonged for two or two and a half years.

3. *Recovery from paralysis.* As a general rule, those portions of the body in which movement returns within one month of the stroke will recover, provided that the occurrence of adhesions and joint changes is prevented; but when no movement has returned within three months of the stroke the paralysis is likely to be permanent.

Treatment. The treatment of cerebral hemorrhage is directed towards saving the patient's life, which is threatened by cerebral compression and anæmia of the bulbar centres. The symptoms of compression are, as already explained, due to increased intracranial pressure from the presence of the hemorrhagic clot, acting as a foreign body within the skull.

The physician has two objects before him: first, the arrest of the hemorrhage, and secondly, the relief of compression.

The methods of arresting hemorrhage are: complete rest, allaying the action of the heart, and lowering the blood pressure, either by means of mild counter-irritation and local depletion, purgation, venesection, or ligature of the internal carotid artery.

Compression may be relieved by lumbar puncture, trephining, or lowering the blood pressure. Lumbar puncture and trephining are attended by certain risks—namely, a possible tendency to bring about rupture or hemorrhage

into the ventricles, or through the convexity of the brain, by assisting its passage along the lines of least resistance.

Rest. Complete rest in bed is essential—the patient being laid flat, with the head and shoulders slightly raised. Everything likely to constrict the neck should be removed. If the patient is conscious, no muscular effort should be allowed, nor under any circumstances should he be moved more than is necessary.

Active treatment. The physician should be guided by the state of the blood pressure [1] and the degree and severity of the symptoms of compression, as to what he should do in a case of cerebral hemorrhage.

(*a*) If the arterial pressure is not high, but the symptoms of compression severe, local depletion—e.g., the application of mustard to the feet or abdomen, or mild purgation—is sufficient. A moderate quantity of cerebro-spinal fluid may also be withdrawn by lumbar puncture.

(*b*) If the arterial pressure is high without much compression, a sharp purge or venesection is indicated.

(*c*) If the arterial pressure is high and cerebral compression profound, the blood pressure should be lowered by a sharp purge or venesection, and the pressure symptoms relieved by lumbar puncture or trephining—this latter being especially useful when the hemorrhage is subtentorial in position (pons or cerebellum). If the hemorrhage is in the cerebrum, ligature of the internal carotid artery on the side of the lesion is preferable, as by this means the hemorrhage will cease and further compression be prevented, while the general arterial pressure (including that of the basilar artery) will not be lowered, nor the blood supply of the bulbar centres diminished.

In ingravescent hemorrhage, purgation or venesection are indicated. If compression symptoms remain profound after the hemorrhage has ceased, trephining and removal of the clot may be carried out.

In meningeal hemorrhage, trephining, removal of the clot, and ligature of the ruptured artery are the only methods of treatment.

[1] An arterial pressure of over 180 m'm. of mercury may be regarded as high blood pressure; but it should be remembered that any sudden increase of intracranial pressure causes a temporary rise of the blood pressure.

DIFFERENTIAL DIAGNOSIS

Between Cerebral Thrombosis, Cerebral Embolism, and Cerebral Hemorrhage

	Thrombosis.	Embolism.	Hemorrhage into Brain.	Ventricular Hemorrhage.	Meningeal Hemorrhage.
Previous history	Syphilis Atheroma Previous slight attacks	Rheumatic fever Endocarditis Previous evidence of emboli in spleen, kidney, and brain	May or may not be any history of premonitory symptoms	History of sudden emotion or physical strain	Trauma Rheumatism Puerperium Aneurism
Mode of onset	Usually gradual during rest or after fatigue	Sudden	Sudden; sometimes ingravescent	Sudden	Ingravescent or sudden
Consciousness	Often not lost, especially in syphilitic cases Loss is gradual, or may be sudden or of short duration, and not profound, except when large vessel is blocked	Loss of consciousness, which may be of short duration	Lost: coma deep and sudden, except in the ingravescent form	Coma profound	Loss of consciousness, of gradual or sudden onset, ending in coma
Convulsions	Occasionally	Frequent	Rare	None	Frequent and may be unilateral
Paralysis	Usually present before loss of consciousness, and often partial During coma, distinction between paralysed and non-paralysed limbs usually apparent	Occasionally slight in extent and degree, with temporary increase later	Complete hemiplegia, often with flaccidity and paralysis	Complete bilateral flaccidity	Irritative symptoms followed by paralysis
Temperature	As a rule subnormal	Usually dependent upon the causal malady: often increased after the stroke	Initial depression followed by a rise during 2 or 3 days. In some fatal cases may not rise subsequently	Usually rapid rise to hyperpyrexia	Elevated, may go to hyperpyrexia, or else depressed
Respiration	Not affected	Not affected	Stertorous. Often 'Cheyne-Stokes' in type	Stertorous Cheyne-Stokes	Stertorous

Focal symptoms	Frequently absent, when thrombosis is in a 'silent' area. If present depend on artery thrombosed	Depend, if present, on affected artery	If in a 'silent' area focal symptoms from pressure not uncommon	Masked	Irritative
Condition of patient	Debilitated	Debilitated	Face turgid; pale or pinched Skin moist	Plethoric	Lethargy, restlessness or delirium
Vessels	Usually atheromatous in the old or syphilitic in the young	Apparently healthy	Atheromatous	Atheromatous	Variable, but often normal
Heart and pulse	Weakened action If hypertrophied, compensation often failing Pulse compressible	Endocarditis or valvular lesions	Hypertrophied Pulse full, incompressible; later may become rapid and feeble	Hypertrophy of heart Action strong	Pulse slow, becoming rapid Heart variable
Kidneys	Variable; may or may not be albuminuria	Usually normal May be seat of infarcts	Granular kidneys Albuminuria	Granular kidneys	Often normal
Eyes	May be thrombosis of central artery and œdema 'Macular figure' often present Degeneration of retinal arteries	May be embolism of central artery or hemorrhages	Albuminuric retinitis Vessels degenerated	Albuminuric retinitis	Usually normal, but may show subhyaloid hemorrhage
Lumbar puncture	No blood in cerebro-spinal fluid	No blood	No blood	Blood in cerebro-spinal fluid	Blood in cerebro-spinal fluid
Prognosis and duration	Favourable to life, especially in syphilitic cases (2 to 10 years) In atheroma; not more than 2 years Liability to recurrence in both Recovery from paralysis good in young cases	Unfavourable, except in non-malignant cases	Duration usually under one year Permanent palsy more complete	Fatal under 14 hours	Basal, rapidly fatal. Convexity: variable, but not unfavourable in ingravescent or traumatic cases

HEMIPLEGIA

Hemiplegia, or paralysis of one side of the body, is the result of an apoplectic seizure, whatever be the nature of the underlying lesion.

For descriptive purposes it may be divided into two periods: (1) that observed during the first three or four weeks following 'the stroke,' when it is characterised by more or less complete unilateral paralysis with flaccidity or only incipient rigidity of the limbs; and (2) that which succeeds the above, and determines the condition of chronic, or persistent, hemiplegic weakness, whose features are partial paralysis, spasticity and contracture of the limbs, and sometimes associated posthemiplegic disorders of movement.

1. In favourable cases the hemiplegic weakness may rapidly subside, so that in two or three months little evidence of the previous paralysis may be observed, although defects in the finer movements of the hand may persist for an indefinite period. Even in cases of considerable severity, some return of movement makes its appearance within three or four weeks, so that the patient may be able to rise from bed and walk with assistance.

The method of recovery of the paralysed side usually follows certain definite lines. The common observation is that the face recovers before the leg, and the leg before the arm. The coarser movements at the larger joints recover before the finer movements at the smaller joints, but the more specialised movements of the hand, fingers, and thumb rarely regain the previous power of precise movement.

A detailed examination of the motor functions, made during the first month after the stroke, will show that the lower portion of the face is considerably more paralysed than the upper part. The tongue is protruded towards the paralysed side. Some weakness of the movements of the head and neck may be observed when movements are made against resistance to the paralysed side. The arm may be raised a little at the shoulder joint, but the movement is accompanied usually by considerable pain; flexion and extension at the elbow are feeble; the grasp, if present at all, is very feeble. The leg may be drawn up at the hip and stretched out at the knee, but dorsiflexion of the ankle is feeble if performed at

all. The trunk movements are weakened on the paralysed side.

On the hemiplegic side the tendon jerks are exaggerated, an ankle clonus and extension of the big toe are usually present, and the abdominal reflexes are abolished, or impaired.

Even during this stage some rigidity of the muscles may be detected with a tendency to contracture of the flexor groups, so that passively straightening the fingers, wrist, or elbow may be accompanied by resistance. The mechanical irritability of the muscles is increased.

The distribution of the paralyses and the presence of associated hemianæsthesia, hemianopsia, and aphasia depend upon the position of the lesion.

2. Although the clinical course of a hemiplegia has been divided into two stages, there is no hard-and-fast line of demarcation, the late emerging gradually out of the early. The most prominent of the symptoms of the later stages are spasticity and contracture of the limbs, in which flexion predominates over extension, and pronation over supination. Hence the arm is adducted at the shoulder and flexed at the elbow, the forearm is in a state of pronation, the fingers are clenched, and the thumb flexed and opposed into the palm. The thigh is adducted at the hip joint, the knee is rigid, the heel slightly drawn up, the foot inverted, and the toes pointed. The attitude and gait of a hemiplegic person are in consequence highly characteristic. The paralysed side seems to move as a whole, and to be lower than the normal side. The arm is carried in the position just described; the leg is moved in a rigid fashion, the foot being carried forward in the arc of a circle, the toes and inner edge of the foot scraping the floor.

These patients are said when turning to pivot upon the non-paralysed side, but there does not seem to be any constancy in this phenomenon.

The mental condition also shows prominent changes in many cases. Alterations in temperament and disposition are stated to have occurred since the stroke. The memory is often impaired, and a marked tendency to loss of emotional control is obvious—these patients laughing, but more usually crying, on the slightest provocation. They frequently become apathetic and indifferent to their surroundings, and a previous

natural tendency to effort and activity may be replaced by timidity and want of decision. It is difficult, however, to say how far the post-hemiplegic dementia may be attributed directly to the focal lesion or to the accompanying arterio-sclerosis.

Treatment. Much may be done in the weeks succeeding the stroke to prevent or retard the onset of the rigidities, contractures, and arthritic changes which characterise so many chronic hemiplegics. In order to be of much avail, massage and passive and active movements against resistance require to be commenced early and continued for a long time, usually for several months.

Treatment should therefore be commenced as soon as the immediate effects of the stroke have passed away. Of most service are massage and passive movements; but the faradic currents may be of use when rigidity is slight or absent. The patient should also be encouraged to use his limbs as freely as possible: daily practice with some form of 'exerciser' being of distinct advantage. For the arthritic pain, radiant-heat baths, combined with passive movements, are useful.

INTRACRANIAL ANEURISM

The description given here refers only to aneurism of the larger cerebral arteries.

Etiology. Aneurism is more frequent in males than in females. It occurs especially from the age of ten years onwards to sixty, but is more common in mid-adult life than at either extreme. The most common cause is embolism of a cerebral artery—occurring in association with endocarditis, syphilitic disease of the arteries, and, in rare instances, arterial degeneration and traumatism.

Frequency. The internal carotid arterial system is more often the seat of aneurism than the vertebral. The arteries affected are in order of frequency (according to Gowers), the middle cerebral, basilar, internal carotid, anterior cerebral, posterior communicating, anterior communicating, vertebral, posterior cerebral, and inferior cerebellar. Aneurisms affecting the vessels of the basal ganglia and centrum ovale are rare, except in the miliary form.

Symptoms. The symptoms produced by aneurism depend more upon the situation than upon the size of the swelling.

In the embolic variety rupture is liable to occur early, on account of the softened condition of the vessel walls, and is often the first indication of the morbid condition. The arterial dilatation which occurs in these cases, even when pressing upon nervous structures, does not give rise to pressure symptoms.

In cases of slowly growing aneurism, with firm walls, symptoms of compression or destruction of the surrounding tissues may appear early in the course of the disease.

From the standpoint of symptoms, aneurisms may be divided into: (*a*) those in which no symptoms were observed during life (18 per cent.); (*b*) those in which there were indications of cerebral tumour (16 per cent.); (*c*) those in which symptoms of tumour, or other cerebral lesion, were followed by fatal apoplexy (20 per cent.); and (*d*) those in which the first indication was an apoplectic seizure (46 per cent.).[1]

Out of 555 cases of cerebral aneurism, 372 died of apoplexy. In 339 of these the apoplectic stroke was due to rupture of the aneurism; while in the remainder, the hemorrhage was due to the rupture of a blood-vessel not affected by aneurism.[1]

General symptoms are frequently absent, but, when present, pain in the head is usually associated with buzzing noises. Optic neuritis is rare, but is probably due to the same causes as in intracranial tumour. Noises in the head—such as singing and buzzing—do not occur out of proportion to their frequency in general intracranial diseases.

The hearing of a murmur on auscultation of the skull, which has been referred to in this connexion by many writers, is of no value, as it has only been verified in two out of 555 cases (Beadles). It has also been heard in cases in which no aneurism was present, as in hydrocephalus, Graves's disease, and anæmia. A murmur has also been heard in cases of vascular tumour of the brain, and when a tumour has pressed upon a cerebral artery.

[1] Beadles, *Brain*, 1907.

The mental dullness and irritability which have been stated to be symptoms of cerebral aneurism are in reality due to general arterio-sclerotic changes.

The only sign of any real significance is the intermittent character of the symptoms.

Focal symptoms. Aneurism of the cavernous part of the internal carotid artery is shown by paralysis of the ocular muscles, the third nerve being first affected, in association with homolateral blindness from pressure upon the optic nerve.

In the case of the anterior cerebral artery, loss of sight and of smell may be present from pressure upon the optic and olfactory nerves.

Third nerve paralysis and homonymous hemianopsia to the opposite side may be found in aneurism of the posterior communicating artery.

In the case of the middle cerebral artery, convulsions followed by paralysis of a focal character simulating cerebral tumour have been described.

Aneurism of the basilar artery, in its anterior part, has been associated with hemiplegia from pressure upon the crus cerebri; in its posterior part, with symptoms of pressure upon the pons. According to its lateral and antero-posterior position, pressure symptoms are referred from the fifth cranial nerve downwards. In a few cases of basilar aneurism, movements of the head have given rise to temporary respiratory difficulty from pressure upon the medulla.

In aneurism of the posterior cerebral artery, palsy of the third and sixth nerves with a crossed hemiplegia has been described.

The cerebellar arteries are rarely affected.

The **diagnosis** of cerebral aneurism is practically impossible; but in a case with focal symptoms referred to the position of one of the cerebral arteries as above described, in which the symptoms are intermittent in character, without the accompanying general symptoms of intracranial tumour, and in which either a history of syphilis or of endocarditis is obtained, the presumption is in favour of cerebral aneurism. The X-rays might prove of assistance.

The **treatment** consists of rest in bed, with large doses of potassium iodide internally. If the aneurism can be localised with certainty, the internal carotid artery should be ligatured.

Miliary Aneurisms

These are minute dilatations of the cerebral arterioles. They are extremely small, and very numerous. They are rarely found under forty years of age. They are most frequent in the vessels supplying the basal ganglia, then the cortex cerebri, pons, cerebellum, and centrum ovale.

A weakening of the muscular coat, so that the vessel wall consists only of the intimal and adventitial sheaths, is the pathological change giving rise to these arterial dilatations. Owing to this change, the vessel wall yields to the intra-vascular pressure.

Their rupture is a common cause of cerebral hemorrhage.

Chapter IV

THE CEREBRAL PARALYSES OF INFANCY AND CHILDHOOD

The various forms of cerebral paralyses occurring in infants and children present many features, which are quite distinct from those seen in adults. This is accounted for by the undeveloped state of the infant brain, by the pathological processes which underly their production, and by the different effects which similar lesions produce in the infant and the adult brain. The paralysis may be monoplegic, hemiplegic, paraplegic, and diplegic. The symptoms may be progressive and regressive. The mental condition may be unimpaired or show any degree of enfeeblement to complete idiocy or amentia.

The lesions which give rise to cerebral paralyses in children are the following:—

Vascular lesions. (1) *Thrombosis.* This may be arterial, venous, or combined arterial and venous. It is, as a rule, associated with marasmus, or an enfeebled state of the circulation following an acute illness.

If the arrest of the blood supply occurs during fœtal or early infant life, the pathological picture is distinct from that seen in later life. If the arrest is incomplete, either atrophic sclerosis, or softening with secondary cystic formation, results.

If complete, certain portions of the brain may disappear, leaving well-defined cavities, as if portions of the brain had been punched out with a cheese-scoop. This condition is known as acquired porencephaly, and may only be discovered at autopsy.

Thrombosis occurring in later childhood resembles, both in its pathological and clinical characters, that which takes place in adults.

2. *Hemorrhage.* Cerebral hemorrhage is not common in infancy or childhood. It may occur as the result of trauma, of rupture of a vessel into a softened area, in some blood diseases, and in the form of capillary hemorrhages in whooping cough.

Meningeal hemorrhage is not uncommon from injury at birth, when the brain suffers not so much from internal injury as from pressure. This form of hemorrhage occurs chiefly from the small veins running into the superior longitudinal sinus.

3. *Embolism.* This is rare in fœtal life, and only occurs in young children as the result of endocarditis or general blood infection.

Encephalitis, or non-purulent inflammation of the brain. Encephalitis is probably the commonest cause of cerebral paralysis in infancy. The nature of the lesion is identical with that seen in acute poliomyelitis, and may result in almost complete recovery, or leave permanent damage in the shape of cicatrices or cystic cavities. Not a few cases terminate fatally during the acute stage.

Traumatic encephalitis may be caused by damage to the skull or brain, and may be complicated by meningeal or intracerebral hemorrhage.

Agenesia. As the several systems of the brain develop at different ages, a lesion may cause either destruction of any particular system or arrest of its development. There would also appear to be in some cases a congenital tendency to arrest of development or early death of the neurone (abiotrophy).

CONGENITAL CEREBRAL DIPLEGIA

This condition arises from an inherited taint, which may originate either during intrauterine life or after birth. The

influences at work are either poisons introduced through the mother during intrauterine life, or directly inherited from one or other parent. Owing to these causes the nervous structures may never develop, may not attain maturity, or may degenerate after development. Clinical examples of these are seen in children who never learn to walk, who walk late but never properly, or who, having learned to walk, gradually become unable to do so.

Not infrequently one physiological system of neurones is alone affected, but commonly two or more may suffer. It is therefore obvious that in cases of diplegia the clinical condition varies according to the neuronic systems involved. In some cases the disability is almost exclusively motor, in others visual or sensory, and in others psychical. The disease is rarely restricted to one system, and most cases exhibit a proneness to convulsions and mental deterioration.

Morbid anatomy. The convolutions are firm and atrophied, and the sulci wide; but the general shape of the cortex is preserved according to the state of development at which the regressive changes commenced. If convolutionary development has taken place, the appearances observed are known as the 'walnut brain.' Here the neuroglial tissue is proliferated, and the nerve elements are absent or degenerated. There is no evidence of vascular or inflammatory changes. Primary atrophy or degeneration of one neuronic system is combined with a secondary atrophy of those in physiological association with it.

Symptoms. In most cases the disease is evident at or shortly after birth. In other cases the symptoms may not show themselves until the child reaches an age at which it ought to walk and talk. In familial cases the onset is frequently delayed until after the sixth year. The sexes suffer equally.

The chief clinical characteristic is rigidity or spasticity, associated with paresis or paralysis. This is bilateral, though sometimes one side suffers more than the other. The whole body may be affected, the face relatively less than the limbs, but in other cases the spastic weakness is limited to the lower limbs (Little's disease). Deformities due to spastic contracture are frequent. (Fig. 62.)

The *mental deficiency* varies from mere backwardness to

absolute and complete idiocy. In the slight cases the mental condition is characterised by backwardness, slowness in talking, and impaired intelligence. More unfavourable are excitability, restlessness, destructive tendencies, and dirty habits.

The mental condition bears no relation to the physical infirmities.

Cranial nerves. Bilateral primary optic atrophy from degeneration of the ganglion cells of the retina is seen in a considerable number of cases.

FIG. 62.—Photograph of a case of cerebral diplegia, showing paralysis and spasticity with a tendency to contractures. The left limbs are more affected than the right. The face is also involved.

Inequality of the pupils with feeble reaction to light is not uncommon. Hippus is frequently seen. Isolated palsies of the third and sixth nerves may be present; but impairment of ocular movements — such as spontaneous nystagmus or strabismus—are more frequent. Convergent squint is common, but divergent is rare.

Spastic weakness of the facial muscles may or may not be present. It is sometimes accompanied by a similar condition of the tongue and palate. The child may be unable to protrude the tongue, which is often large, articulation is impaired, and saliva may dribble from the corners of the mouth. Swallowing is slowly performed—a long time being taken over the bottle. If made to cry, great facial overaction occurs with little or no proportionate sound.

Motor system. Rigidity may be generalised, or limited to the lower limbs. When extreme, it is possible to lift the child up in rigid extension. In less severe degrees, the child is unable to sit owing to inability to flex the thighs.

When walking is possible, the gait may be that known as 'scissor gait,' or cross-legged progression, the feet being pointed and inverted, the knees slightly flexed, and the thighs strongly adducted and rotated inwards. Milder degrees of spastic paraplegia are also found. In some cases the patient may not be able to bring his heels to the ground, but walks on the balls of the toes (digitigrade gait). (Fig. 63.)

Paresis exists in all cases where rigidity is marked, but rigidity may be present with out much motor weakness.

Fig. 63. – Shows the cross-legged or 'scissor gait' of congenital cerebral diplegia.

In severe cases characteristic attitudes may be assumed. The head is bent forward and the spinal column may show either a kyphotic curve or lordosis. The upper limbs may or may not be affected, but are never involved to the same extent as the lower. The upper arm is adducted, the forearm flexed and supinated, and the hand flexed at the wrist. The lower limbs are extended and adducted at the hip, flexed at the knee, and the feet are in the position of pes cavus or talipes equino-varus. The muscles may be firm or hard to the touch, and in cases of athetosis hypertrophy may occur. In the latest stages wasting and fibrillary contraction with impaired faradic and myotatic irritability are observed.

The *sensory* system may not be affected.

The *reflexes* are characteristic of spastic paralysis. The

deep are increased, the superficial often unaffected, and the plantars are extensor in type.

The sphincters. Incontinence is common, but is mainly due to the mental deficiency. Precipitancy may occur.

Disorders of movement. All degrees of disordered movement are present—from clumsiness and overaction of a slow spastic type to well marked intention tremor like that seen in disseminated sclerosis. Very characteristic are bilateral athetoid and choreiform movements, which may be found in cases in which rigidity is present with little motor weakness (athetotic diplegia).

Speech defects. In the congenital cases speech may never be acquired. In those cases which commence after the child has learned to speak, motor aphasia—sometimes temporary at other times partial and complete—may be observed.

In nearly all cases speech is slowly acquired. Articulation is slow, hesitating, or stammering, and accompanied by facial overaction and slobbering. It not infrequently assumes a form in which the syllables are pronounced in an unusual fashion—such as lalling.

Although unable to speak, these children appear in some cases to understand what is said to them.

Convulsions are commonly associated with cerebral diplegia, epilepsy occurring in about 10 per cent. of the cases.

The following *types of cerebral diplegia* are found:—

1. Generalised rigidity with bilateral paralysis, and more or less mental impairment.
2. Imbecility or idiocy, with slight or no motor weakness.
3. Paraplegia—Little's disease.

ACQUIRED CEREBRAL PARALYSES

Infantile Hemiplegia and Diplegia

Acquired infantile hemiplegia and diplegia are of the following kinds:—

1. *Birth palsies.* These arise from meningeal hemorrhage and trauma.
2. *Acquired palsies.* These are due to:
   (*a*) Encephalitis—primary, traumatic following acute infective diseases, and suppurative.

(*b*) Vascular lesions—thrombosis (arterial and venous), hemorrhage (meningeal and cerebral), and embolism.

Encephalitis is the pathological lesion in the majority of cases of infantile hemiplegia. Although frequently occurring as a sporadic disorder, it is occasionally met with in epidemic form, and is especially common during the seasonal period in which poliomyelitis also occurs (p. 438).

Morbid anatomy. Encephalitis is an acute inflammatory process in and around the blood-vessels of the brain, characterised by hyperæmia, hemorrhagic exudation, cellular infiltration, and multiple small hemorrhages. It may have a local or a general distribution, and affect some regions more than others. The grey matter, owing to its greater vascularity, suffers more than the white, the changes being best seen in the cortex and basal ganglia. Similar changes also occur in the central grey matter, floor of the fourth ventricle, and the anterior horns of the spinal cord. The terms encephalitis and poliomyelitis (superior, inferior, and anterior), are based, therefore, on anatomical grounds.

When the cerebral cortex is affected, the pia mater shares in the inflammation. The brain substance is red, swollen, and softened, and the distinction between grey and white matter is obliterated. Numerous small hemorrhages are also present. The cerebro-spinal fluid is increased in the ventricles.

Microscopically, the small blood-vessels are found distended and ruptured. A perivascular cell proliferation is also found, and the vessel walls may show fatty degeneration. In severe cases small foci of degenerated myeline and fatty and granular débris are also seen. The nerve cells may be either destroyed, show fatty and hyaline degeneration, or only chromatolysis, the observed changes depending upon the intensity of the inflammation; even in severe cases many nerve cells escape. Either resolution or destruction of portions of the brain results. If the latter, the changes consist of an increase of the neuroglia with the formation of scars or sclerotic patches. Secondary degeneration of nerve fibres is also found.

Its onset is most common during the first three years of life, contrasting in this way with thrombosis, which is more common about the fifth or sixth year.

The sexes are about equally affected. Parental alcoholism and syphilis may be predisposing influences.

Symptoms. The onset of the disease is sudden, with convulsions, fever, vomiting, and sometimes coma. After a few days these general symptoms begin to subside, and it is then noticed, if not before, that the child is paralysed. The paralysis is first of the flaccid type, but within a week signs of spasticity develop. In mild cases the paralysis may be slight and transitory; in severe cases, on the other hand, spasticity and contracture set in early, and may be associated with trophic disturbance and arrest or impairment of growth on the paralysed side.

The paralysis at first has a more or less general distribution. If encephalitis is widespread and severe, permanent bilateral palsy (diplegia) may result. In many cases the palsy is confined to one side (hemiplegia). Occasionally it is more or less limited to one limb, but even in such cases reflex changes indicative of a unilateral lesion are present. In other cases the sensory and psychical regions of the brain may be chiefly implicated, when sensory symptoms and mental changes of a transitory or permanent nature may to a large extent replace the paralytic phenomena.

The basal ganglia and central grey matter of the mid-brain may be involved conjointly with the cortical tissues, or, as a primary condition, characterised by permanent oculo-motor paralysis and disorders of movement.

In infantile hemiplegia, the face is less affected than the arm or leg. The facial paralysis gives rise to some stiffness and want of expression. The emotional movements are more impaired than the voluntary. Not infrequently slow mobile spasm or choreiform movements develop at a later period.

Cranial nerves. Strabismus and ocular palsies sometimes occur at the onset of hemiplegia, in which respect the infantile differs from the adult form. (Fig. 64.)

Hemianopsia may be found in some cases, especially in association with hemianæsthesia.

Motor system. The upper limb shows spasticity and contracture; and trophic disturbances, causing diminished growth with blueness and coldness of the limb, further characterise the condition. The arm is adducted at the shoulder, the elbow flexed at a right angle, the forearm

pronated, the wrist flexed and deviated to the ulnar side, and the thumb and fingers firmly flexed. Paralysis of the distal parts of the limb is more pronounced than the proximal.

The lower limb presents the same general features, though in less degree. When the palsy has been severe, the lower limb may be shortened from trophic changes; but even without these the contractures may give rise to shortening. The thigh is adducted and rotated inwards, the knee slightly flexed, the heel drawn up, the foot inverted, and the toes flexed. In other cases pes cavus, or talipes equinus or equino-varus, is present. (Fig. 64.)

Fig. 64. Shows the appearance and attitude of a case of infantile hemiplegia. There is a divergent strabismus of the left eye in this case.

The gait resembles, generally, that seen in adults. Even in cases where the leg is severely paralysed, the disability as regards walking is more apparent than real. The leg is circumducted at the hip, and may be carried across the middle line at the end of the movement. The patient walks upon the toes, and if much shortening exists, a compensatory tilting of the pelvis results.

Sensation may not be impaired, but in other cases hemi-anæsthesia may be present.

The *reflexes*. The tendon jerks are characteristic of hemiplegia. The superficial reflexes may be normal. The plantars are extensor.

The *sphincters* are unaffected.

Trophic changes are seen in arrested or impaired growth of the limbs, vaso-motor disturbances, contractures, and in some cases malformation of the bones.

Speech defects. If the child has not learned to speak, the acquisition of speech may be slow; but, as a rule, it is eventually acquired. If the lesion occurs when the child is learning to speak, and involves the motor speech centre,

aphasia results; but speech is slowly regained by the re-education of the other side of the brain. Aphasia may be a purely transient symptom, in cases where the speech centres are slightly involved. If the speech centres are involved, various forms of motor and sensory aphasia may result, which may be permanent, if the mental condition is defective or the child is under six years of age.

FIG. 65.—A view of the palm of the hand in a case of athetosis.

Quite apart from aphasia is the articulatory disturbance and over-action, depending upon spasticity and inco-ordination of the muscles subserving articulation.

Disorders of movements. One of the most characteristic features of infantile hemiplegia is the development of various forms of irregular movement in the paralysed limbs. These

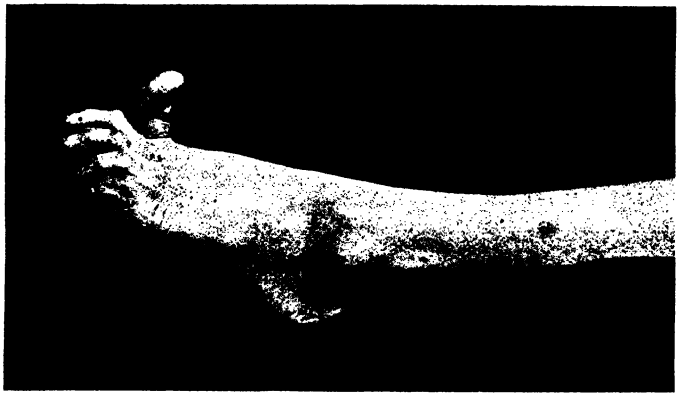


FIG. 66.—Showing athetotic spasm of the great and small toes.

disorders may either occur on voluntary effort or take place spontaneously. (Figs. 65 and 66.)

(*a*) Those occurring on voluntary effort. Owing to stiffness or rigidity movements are carried out slowly and clumsily with

some overaction. Repeated efforts usually diminish the stiffness, with a corresponding improvement in the movement. It is obvious that this disorder of movement can only occur

FIG. 67.

FIG. 68.

FIG. 69.

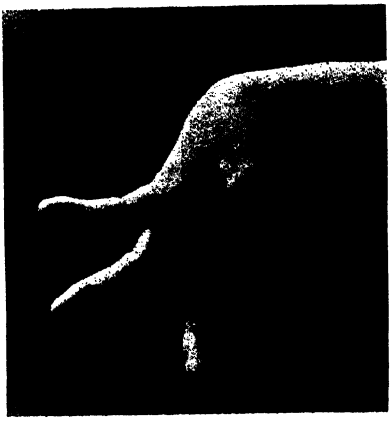


FIG. 70.

FIGS. 67–70.—Four figures showing the attitude assumed by the hands and fingers in a case of athetosis.

in limbs in which a considerable degree of voluntary power exists. Less distinctive, but frequently observed, are coarse or fine tremor, inco-ordination, and rapid jerky movements.

(*b*) The spontaneous movements are chiefly athetosis or mobile spasm. This develops late, and may not supervene

for several years after the onset of the paralysis. Its development signifies that no further recovery from the paralysis will take place.

Athetoid movements are seen in the distal portion of the arm. The wrist remains more or less flexed, the fingers and thumb slowly disengage themselves and become strongly hyperextended, spreading out in a fanlight fashion. The fingers are widely abducted and wander individually or conjointly with a semi-methodical irregularity. (Figs. 67 70.)

Although athetosis is involuntary, it is occasionally set up on attempts at voluntary movements.

Choreiform disorders occur in the form of irregular movements, and, unlike athetosis, often involve the proximal parts of the limbs.

Convulsive seizures. At the onset of the disease, generalised convulsions are invariably present.

Seventy-eight per cent. of the cases of infantile hemiplegia become epileptic. The seizures may either directly supervene on the original convulsive attack, or several years may elapse between the onset of the hemiplegia and the first epileptic seizure. The attacks have the features of the epileptic seizures elsewhere described, the convulsions having a tendency to chiefly implicate the paralysed limbs. There is frequently a local aura, beginning in the paralysed hand or arm. Other auras may point to irritation of a sensory cortical area, with subsequent generalised convulsion. Attacks of minor and of psychical epilepsy may also occur.

PORENCEPHALY

Two varieties of this form of cystic formation in the cerebrum are observed: one developmental, the other secondary, to cutting off of the blood supply—pseudo- or acquired porencephaly. The developmental nature of the first variety is proven by its not infrequent association with other similar conditions—such as cavity formation within the spinal cord (syringomyelia) and spina bifida. The cavity is more or less funnel-shaped, and always communicates with the ventricle and sometimes with the subarachnoid space.

The acquired variety may be found in any part of the

brain, but is more common over the Rolandic and parietal regions. All degrees of porencephaly are seen—from a mere dimple on the surface to well-marked cystic formation.

Clinically, the developmental form may be suspected when other signs of malformation (meningocoele) are associated with mental deficiency or hemiplegic weakness. In the acquired form, the symptoms depend on the situation of the lesion. The existence of porencephaly may be suspected; but in most cases it remains undetected until demonstrated at the autopsy, perhaps many years afterwards.

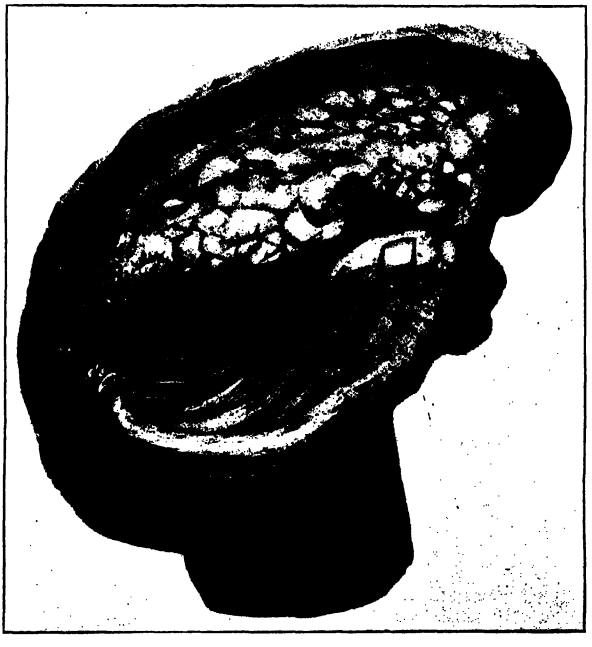


FIG. 71.—The brain of a young adult showing atrophic sclerosis and acquired porencephaly as described in the text (p. 223).

ATROPHIC SCLEROSIS AND CYSTIC FORMATION

In Fig. 71 a brain is depicted showing both conditions in the same hemisphere. The following features are observed: first, a marked diminution in the size of the affected hemisphere; and secondly, the presence of an extensive cyst corresponding to the area of distribution of

the posterior cerebral artery. The cyst is lined by a wall of dense fibrous tissue. The remainder of the hemisphere is in a state of sclerotic atrophy with microgyria; the convolutions are small and hard, the sulci are wide, and the surface is not unlike that of the kernel of a walnut. Microscopic examination shows the almost complete disappearance of the nerve elements and their replacement by dense neuroglial tissue.

The whole of one hemisphere may be affected, or only certain areas; the basal ganglia commonly escape. The blood-vessels are of normal appearance.

The atrophic area of the brain may be compensated partly by an excess of the cerebro-spinal fluid, and partly by an increase of the thickness of the calvarium over the affected regions.

Clinically, the cases of this condition are of acute onset. They may occur during intra-uterine life, and result from encephalitis or acute vascular lesions. The picture presented in the later stages depends upon the situation and size of the lesion, and may present all intermediate forms between infantile hemiplegia, diplegia, and simple mental deficiency.

Secondary degenerations occur in the lower parts of the brain and spinal cord as a result of the different cerebral lesions just described. When the lesion affects mainly the cortical motor areas, the pyramids and the crossed and direct pyramidal tracts are either absent, ill-developed, or degenerated. With extensive unilateral lesions, involving both the centrum ovale and the basal ganglia, there is a crossed cerebellar atrophy and wasting of the mesial fillet. The fronto-pontine and temporo-pontine tracts are degenerated after lesions more or less confined to the frontal and temporal lobes.

Physical conformation. These patients are usually undergrown, and present, in addition to the deformities of the limbs already described, other marked physical peculiarities. In the majority of the diplegic cases the head is small, though in a few it is larger than normal. In the hemiplegic cases, the skull over the affected hemisphere is smaller than over the non-affected, leading to marked cranial asymmetry. Other stigmata of degeneration may be seen in the high, narrow, and deformed palates presented by some cases. The

body and limbs on the paralysed are smaller than on the normal side, a change which is due to defective development of all the tissues. The want of growth is sometimes more apparent than real, owing to contracture and deformity, as measurements fail to bring out any definite shortening of the bones of the paralysed limbs.

Prognosis. The prognosis in the cerebral palsies of infants and children depends upon a number of conditions. The diplegic cases have, on the whole, a shorter life than the hemiplegic, for it is not common to find such patients living much beyond early adult life. If the child survive the acute onset of the disease, even in a partially paralysed state, life may be prolonged for many years. The onset of epileptic attacks, however, introduces an element of uncertainty and some danger. On the other hand, recovery from the acute onset may not occur. There are cases, however, in which recovery is so satisfactory that it is scarcely possible to detect any evidence of a local lesion. These cases, notwithstanding, may develop epilepsy about the time of puberty.

Pronounced athetosis is unfavourable in so far as it interferes with a satisfactory issue to treatment. Mental deterioration, marked rigidity, and paralysis are unfavourable, owing to the liability to intercurrent complications.

Treatment is confined almost entirely to physical exercises with a view to the correction of motor disabilities, rigidity, and contractures, and educational exercises to develop and improve the mental condition. It is scarcely necessary to describe these in detail, as they are conducted along similar general lines to those in healthy children. It is preferable that these children should be instructed either in special schools or under private tuition. Instruction in elocution is an important factor when speech defects are present.

Contractures in the feet are corrected by properly adjusted boots or by tenotomy. Epileptic attacks should be controlled in the manner described under Idiopathic Epilepsy (p. 579).

CEREBELLAR AFFECTIONS IN CHILDREN

A condition, analogous to cerebral diplegia, may occur from arrested development or early death (agenesia) of the nerve elements of the cerebellum. It is usually associated

with similar changes in the cerebrum, but little is known about it.

Symptoms. Acute cerebellar ataxia may come on suddenly, either during the course of, or shortly after, an acute febrile disorder. There are other cases, also, in which similar symptoms arise without any antecedent disease.

Although there are no certain pathological data that these cases are due to inflammatory or vascular lesions, such as have been already described in the cerebrum, yet presumptive evidence exists to show that they may be due to :—

(*a*) Encephalitis affecting the cerebellum and its mesencephalic connexions.

(*b*) Acute vascular lesions (hemorrhage or thrombosis) arising either during or after an acute specific disease.

These affections are characterised clinically by inco-ordination of the movements of the trunk, head, neck, limbs, and eyes, and of articulation. In some cases there is evidence that the disease simultaneously affects other areas—such as the midbrain and cerebrum. In one case personally observed (following upon measles), nystagmus, slight articulatory inco-ordination, ataxia of the upper limbs—but especially of the trunk and lower limbs—were present. The motor, sensory, and reflex functions showed no evidence of affection of any part of the brain other than the cerebellum. The mental condition was unaffected.

The **prognosis** in these cases is good, a slow and gradual improvement taking place. The ataxia of the trunk and lower limbs, when standing or walking, is the last symptom to disappear. The more definitely limited the symptoms are to the cerebellum, the more hopeful is the outlook for recovery.

Chapter V

INTRACRANIAL TUMOURS

The advances which have been made in the localisation of intracranial tumours, and in the knowledge of their nature and mode of growth, have placed their treatment within the range of practical surgery. Many of these growths, either from their position, their character, or their size, cannot be extirpated;

but even in such cases the employment of surgical interference as a palliative rather than a radical measure may be of the greatest benefit in relieving the general symptoms, preventing blindness, and in prolonging life.

Frequency and Character of Tumours

The relative frequency of the various forms of tumour growth affecting the brain differs materially according as statistics are drawn from children or adults. This difference is so marked that to give a collective table is entirely misleading.

In children, tuberculous tumours are as common as all other forms of new growth taken together. Gliomata are next most frequent; then the sarcomata, and then miscellaneous forms of tumour. In adults, gliomata form 48 per cent., sarcomata 31 per cent., tubercle 6 per cent, endotheliomata 5·5 per cent., carcinomata 5 per cent., gummata 3 per cent., and tumours of the choroid plexus 1·5 per cent. These percentages are drawn from the annexed table, which is based upon the post-mortem records of the Queen Square Hospital. These figures are mainly from adult cases, but include a small percentage of children.

	Single.	*Multiple.*	*Total.*
Glioma . .	95	1	96
Sarcoma . .	52	4	56
Endothelioma .	11	0	11
Tubercle . .	8	4	12
Fibro-sarcoma .	6	0	6
Gumma . .	4	2	6
Carcinoma . .	2	8	10
Tumours of the choroid plexus	3	0	3
Totals	181	19	200

New growths within the brain may be primary or secondary to tumour growth elsewhere.

Gliomata. The gliomata are the most common, and may occur at any age. They are rarely multiple. They may be situated in any part of the brain. They are of an infiltrating

character. Several varieties are found: (*a*) a small-celled, more or less circumscribed growth; (*b*) a large-celled, vascular, rapidly growing and infiltrating tumour; (*c*) forms of an intermediate type.

Those of rapid growth tend towards cystic degeneration, especially when situated in the cerebellum, and are not infrequently the seat of hemorrhage. They do not implicate the meninges, or bones of the skull. They may be seen spreading along small blood-vessels, and at the margin of the tumour small islands of new growth are observed surrounded by normal tissue. Where cystic degeneration has taken place, neuroglial cells may be seen arranged in several layers radiating outwards from the cavity. In the pons, where glioma occurs in the form of 'hypertrophy of the pons,' a wide infiltration of glial cells is seen, without much tendency to cystic degeneration.

Sarcomata arise in connexion with the meninges, periosteum, blood-vessels, and bones. They are found at all ages. They involve the brain tissue indirectly, and destroy the cerebral tissue by compression. They also radiate and extend along the cerebral blood-vessels. They are more often multiple than the gliomata. When growing from the meninges or periosteum they are fibro-sarcomatous in character, are usually of a dense, firm consistence, and often attain to large size.

Round-celled sarcomata are seen in connexion with the soft membranes and blood-vessels, and tend towards greater malignancy than the fibrous variety. Melanotic sarcoma, always a secondary tumour, is the most malignant type, and may be scattered throughout the brain, or widely spread over the meninges. Sarcomata, growing from the base of the skull, may attain a large size and be very widely diffused.

Tuberculous tumours may be solitary or multiple. They may be situated deeply or on the surface. They are found especially in children. The tumours are firm and non-vascular, tending towards caseation and sometimes purulent disintegration. The tubercle bacillus can be demonstrated in most instances. They may remain quiescent for long periods, or give rise suddenly to tuberculous meningitis.

Endotheliomata spring from the dura mater and extend on the one hand into the skull and on the other hand into the brain. They do not infiltrate the brain, but push it before

them and compress the nerve fibres. They may occur at any age, are always single, and may attain to a large size.

Gummata are sometimes single, but more usually multiple, or associated with gummatous meningitis. They invariably grow from the membranes. Commonly situated on the surface of the brain, they may be found, however, in the central parts growing from one of the deep folds of pia mater. They tend either to break down, or to form firm fibrous granulomatous masses. They do not suppurate. The surrounding brain is destroyed partly by pressure, and partly by vascular occlusion and inflammatory exudation.

Psammomata are found in connexion with the meninges and the pineal gland. They do not attain to a large size.

Adenomata may arise in connexion with the hypophysis cerebri (pituitary body).

Tumours of the choroid plexus are carcinomatous with a papillomatous structure. They arise from the choroid plexus and are situated inside the ventricles, but may infiltrate the surrounding tissue, or extend through the ventricular openings and appear upon the outer surface in the lateral recesses. Occasionally they give rise to secondary deposits in the central canal of the spinal cord, on the surface of the cord, or on the posterior nerve roots.

Carcinomata. The carcinomata are soft, often vascular and irregular in outline and distribution. They are found growing from the membranes, or deeply in the brain substance. They are usually multiple, and are more or less circumscribed. Of our ten cases the tumours were secondary to primary growths in the breast (three cases), rectum (four cases), supra-renal capsules (two cases), and lung (one case).

Parasitic cysts are of two varieties—the cysticercus and the echinococcus. They are rare in this country. They occur more commonly in connexion with the ventricles of the brain.

Situation of Tumours

Cerebrum. Cerebral tumours are more common in adults than cerebellar or pontine tumours. In children, on the other hand, pontine and cerebellar tumours are more frequent, the cerebrum being rarely affected except by tuberculous tumours.

The frontal area is rarely the seat of tumour in young

children. Glioma is the most frequent form in this locality, and usually develops between the ages of thirteen and thirty-five. Fibrosarcoma develops in adult and later life.

Glioma is the most common deep-seated tumour in the other regions of the cerebrum. The sarcomata are next in frequency and arise later in life, and involve the cortex more often than the deeper tissues. The gummata and the carcinomata are usually cortical, often multiple, and arise in adult and later life respectively.

Endothelioma occurs in early and mid-adult life, grows from the membranes, and does not infiltrate the brain tissue.

Cerebellum. This region is commonly affected in children by tubercle, glioma, and sarcoma. In adults, gumma, sarcoma, and carcinoma are the most common varieties of new growth.

Pons Varolii. The glioma is the most common form of tumour. It is found in children and young adults. Tubercle is also common in children.

Fourth ventricle. Gliomata, sarcomata, papillomata, and parasitic cysts may be found. The papillomata grow from the choroid plexus. These tumours may occur at any age.

Extra-cerebellar tumours do not occur in young children, but from the age of fifteen years onwards. They are usually fibro-sarcomata or neuro-fibromata.

The Table on page 231 shows the relative frequency of the different kinds of tumour in the several regions of the brain.

Anatomical and Physiological Factors

In order to obtain a fuller comprehension of the general effects of intracranial tumours, reference will be made to some anatomical and physiological factors, which play an important rôle in their production.

The brain is enclosed in the cranial box, from which there is only one large outlet—the foramen magnum, through which the brain is in continuation with the spinal cord.

The brain is a viscous mass completely filling the cranial cavity. It is surrounded by three membranes: (1) The *pia mater*. This is in close connexion with the surface of the brain, dips into the sulci, and sends processes into the cerebral

RELATIVE FREQUENCY OF THE DIFFERENT KINDS OF TUMOUR IN THE SEVERAL REGIONS OF THE BRAIN.

	GLIOMA.	SARCOMA.	TUBERCLE.	ENDOTHELIOMA.	GUMMA.	FIBROSARCOMA.	TOTALS.
Frontal area .	14	5	—	3	1	1	24
Rolandic „ .	2	8	—	1	1	1	13
Parietal „ .	6	—	—	1	1	—	8
Occipital . .	2	2	—	—	—	—	4
Temp. sphen. .	9	9	—	—	—	—	18
Corp. callos .	2	1	—	—	—	—	3
Centr. ovale .	8	1	—	—	—	—	9
Basal ganglia .	5	2	1	—	—	—	8
Mesencephalon	8	4	2	—	—	—	14
Pons Varolii .	17	—	—	—	—	—	17
Cerebellum . .	17	8	5	—	1	—	31
Extracerebellar	1	5	—	1	—	4	11
Latl. ventricle	2	—	—	—	—	—	2
Fourth ventricle	1	2	—	—	—	—	3
Insula . .	1	—	—	—	—	—	1
Falx	—	—	—	5	—	—	5
Pituitary body	—	2	—	—	—	—	2
Tentorium . .	—	1	—	—	—	—	1
Skull . . .	—	2	—	—	—	—	2
TOTAL . .	95	52	8	11	4	6	176

Tumours of the choroid plexus (third and fourth ventricles) and the carcinomata are omitted.

substance. (2) The *arachnoid mater*. This is somewhat thicker, is loosely connected to the pia, and bridges over the sulci between the convolutions. (3) The *dura mater*: a dense, strong, sensitive membrane in close relation with the skull. Within the dura are situated the cerebral venous sinuses into which the blood from the cerebral veins passes on its way to the emissary veins, which convey it from the cranial cavity.

The dura mater sends processes into the cranial cavity, dividing it into three more or less distinct chambers. In the first place, the tentorium cerebelli cuts off the posterior fossa from the anterior and middle fossæ. This is a particularly strong and resistant membrane, with only one opening in it, which is completely filled by the crura cerebri connecting the cerebrum with the pons. In the posterior chamber, beneath the tentorium, lie the pons, cerebellum, and bulb. Secondly, the cerebral chamber, which is above the tentorium, is divided into two halves by the falx cerebri, a process of dura mater which lies in the medial sagittal plane, and separates the two cerebral hemispheres in front and above the corpus callosum. This subdivision of the cerebral chamber is therefore much less complete, owing to the large size of the corpus callosum and the relative laxity of the partition between the two hemispheres.

Under the dura and arachnoid membranes are the subdural and subarachnoid spaces, which in normal conditions are merely potential cavities, but contain a small amount of cerebro-spinal fluid. These spaces communicate with the subarachnoid spinal space and with the intraventricular system. The dura and arachnoid membranes are also reflected along the optic nerves to form the optic sheaths and perineurium, so that the subdural and subarachnoid spaces are continued along the optic nerves outside the cranial cavity.

Inside the brain is a ventricular system consisting of the two lateral ventricles—one in each cerebral hemisphere—communicating by the foramina of Monro with the third ventricle, which is mesial and basal in position. From the hinder end of the third ventricle the aqueduct of Sylvius passes downwards into the fourth ventricle, which lies in the posterior chamber between the cerebellum, the pons, and the upper portion of the medulla oblongata.

The fourth ventricle communicates with the subarachnoid space by the foramina of Majendie and by openings in the

lateral recesses of the ventricle between the medulla and the cerebellum.

The subdural and subarachnoid spaces of the brain and cord are directly continuous through the foramen magnum.

The Cerebral Circulation

The arterial supply of the brain is provided by two sets of vessels, the internal carotid and vertebral arteries, which anastomose freely (p. 166). There are no valves in the cerebral veins, and there is no lymphatic system apart from the vascular system. This is provided with large perivascular spaces, and, probably also with small intra-adventitial spaces, which subserve the functions of a lymphatic system. In normal conditions the arterial pulsation is transmitted to the cerebral sinuses (Hill).

The brain expands within the cranial cavity. The expansion is of two kinds: (1) circulatory with arterial pulsation, and (2) respiratory. The experiments of Leonard Hill[1] show that the brain stands in such close relationship to the general venous pressure, that its greatest expansion is during expiration, owing to the rise of pressure which takes place in the right side of the heart. This dams back the blood into the cerebral sinuses and forces the cerebro-spinal fluid from the cranium into the less rigid vertebral canal. During inspiration, on the other hand, the blood flows from the cerebral veins and the cerebro-spinal fluid returns into the skull.

There is a definite relationship between the intracranial venous pressure and that of the cerebro-spinal fluid. The former cannot remain higher than the latter, as transudation takes place from the veins into the cerebro-spinal fluid until equilibrium is restored. Thus a rise in intracranial venous pressure implies a rise in the cerebro-spinal fluid pressure. The withdrawal of cerebro-spinal fluid therefore can only be of temporary benefit in the relief of increased intracranial tension. The one method of relieving intracranial pressure is to reduce the venous pressure within the skull either by venesection, purgation, or by removing a sufficiently large portion of the calvarium.

In tumour of the brain the increased intracranial tension is due to an increase of the contents of the skull which causes

[1] Leonard Hill, *The Cerebral Circulation*, 1896.

an obstruction to the cerebral circulation, thus raising the venous pressure and, *pari passu*, that of the cerebro-spinal fluid. (See under Cerebral Hemorrhage, p. 196.)

When the general intracranial pressure is raised, the brain is pressed against the dura mater and the cerebro-spinal fluid is

FIG. 72.—Represents a tumour of the left frontal lobe growing across the mesial plane, and secondarily affecting, by pressure, the right hemisphere.

forced from the cranial subdural and subarachnoid spaces into the corresponding spinal spaces and the optic nerve sheaths.

The determining factors in the production of the general effects of intracranial tumour therefore are: (1) the situation and size of the tumour; (2) the nature and rate of growth of the tumour; (3) the local effect of the tumour upon the brain substance. The focal effects depending upon the local lesion will be discussed later under Focal Diagnosis.

EFFECTS OF INTRACRANIAL TUMOURS UPON THE CRANIAL CONTENTS

1. *Tumours situated in the anterior or cerebral chambers.* A tumour in one or other of the anterior cerebral chambers gives rise to the following sequence of events.

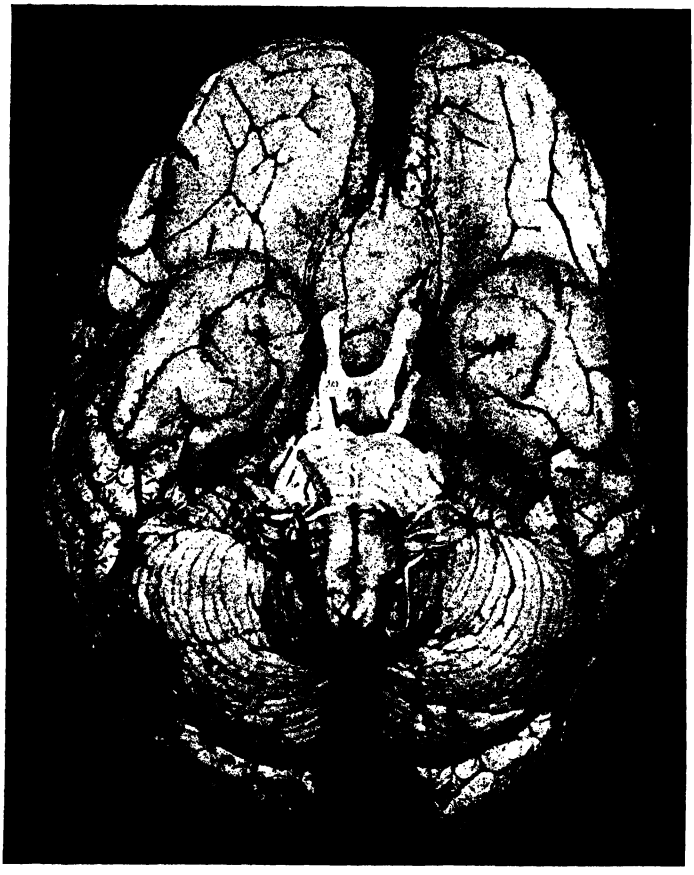


FIG. 73.—Shows the ventral aspect of the brain with a tumour involving the frontal lobes.

In the first place, there is an increase in the bulk of the contents of the chamber, with the result that a rise of pressure occurs in that chamber. Secondly, this is followed by a rise of pressure in the opposite anterior chamber and a consequent increase of the supra-tentorial tension;

and thirdly, by a rise of the general intracranial pressure both above and below the tentorium.

These general results are dependent upon an actual increase in the total solid bulk of the cranial contents; and if the brain be a constant quantity, the larger the tumour the greater the pressure. But if the tumour grows at the expense of the brain substance and destroys it, then the pressure is

FIG. 74.—Shows the formation of a 'pressure cone,' photographed from the side.

lessened proportionately to the amount of brain substance which is destroyed.

The constant effect is to displace the brain from the seat of tumour in the direction of least resistance; and this effect is first felt in the chamber in which the growth is situated. The brain may be displaced upwards, downwards, forwards, backwards, or across the middle line. The skull being practically unyielding, the nerves which run over the base of the brain may suffer from direct compression; on the other hand, the points of least resistance are the ventricles and the opposite hemisphere, and therefore the affected hemisphere tends to be displaced across the middle line. Thus a rise of pressure in both anterior chambers takes place, and eventually

becomes so great that the tentorium cerebelli is displaced downwards and a 'pressure cone' is formed by the blocking of the foramen magnum by the bulb. (Figs. 74 and 75.)

These stages, in the development of the increased intracranial pressure, may be recognised clinically, in the first stage, by the earlier occurrence of optic neuritis on the same side as the lesion in cerebral as contrasted with cerebellar growths;

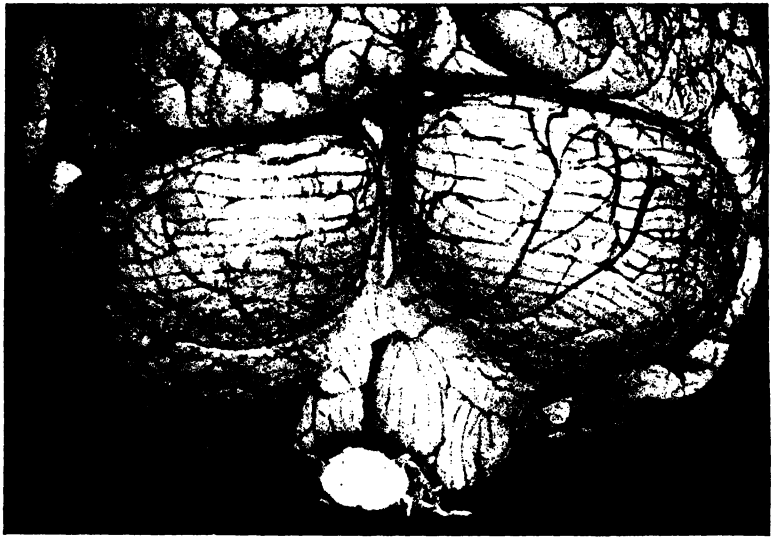


FIG. 75.—Shows a 'pressure cone' affecting the medulla oblongata, viewed from behind.

and by the signs of interference with the functions of the same cerebral hemisphere—such as changes in the reflexes and slight hemiparesis on the opposite side of the body—apart from the immediate local effects of the growth. The second stage, when the increased pressure affects both cerebral hemispheres, is demonstrated by the onset of mental changes and by evidence of impairment of function of the opposite hemisphere, so that changes in the reflexes are found on both sides. This accounts for the earlier onset of the mental symptoms in cerebral than in cerebellar tumours. The third stage is characterised by the failure of the respiratory centre from anæmia of the bulb owing to the formation of a 'pressure cone.'

2. *Tumours situated in the posterior or cerebellar chamber —subtentorial tumours.*

The posterior chamber is smaller than the anterior, its floor is formed by the posterior fossa of the skull, its sides and roof by the occipital bones and the firm resisting tentorium cerebelli. The place of least resistance is the foramen magnum.

Tumours in the posterior chamber may be situated: (1) outside the cerebellum, pons, or medulla; and (2) inside the cerebellum, pons, medulla, or fourth ventricle.

Of tumours situated *outside the cerebellum*, the most common are those in connexion with the auditory nerves. As a rule they are of slow growth and firm consistence, and tend to grow at the expense of the nervous tissue of the pons and cerebellum, which become indented and atrophied by the constant pressure upon them. It is therefore not until the growth has attained a sufficient size to force the bulb down into the foramen magnum that any great increase in intracranial tension takes place. When this occurs the communication between the cranial and vertebral cerebro-spinal spaces is cut off. In consequence, a rise occurs in the intraventricular and intracranial tension with resultant optic neuritis, provided the patient survives the effects of the bulbar anæmia. Clinically, we find in these cases early, well-defined, and long-continued local symptoms, with a late onset and rapid development of the general symptoms of intracranial tumour and often sudden death.

In tumours growing *within the cerebellum* displacement is towards the points of least resistance—the fourth ventricle and the foramen magnum. In some cases the ventricular system is obstructed with a consequent rise in the intraventricular tension. In other cases there is only partial blocking, with pressure exerted downwards upon the bulb, and interference with the communication between the cranial and vertebral subdural and subarachnoid spaces. Increasing rise of pressure leads to anæmia of the vital medullary centres.

Tumours growing into or in *the fourth ventricle* may extend and appear externally through the lateral recess between the medulla and cerebellum, and may eventually, along with the medulla, form part of the 'pressure cone.'

In these cases the ventricular system becomes distended,

the subarachnoid and subdural spaces obliterated, and optic neuritis develops early and simultaneously in both eyes. Mental symptoms are not common in cases of cerebellar tumour: as a rise in intracranial tension, sufficient to cause compression and interference with cerebral function, is not compatible with life.

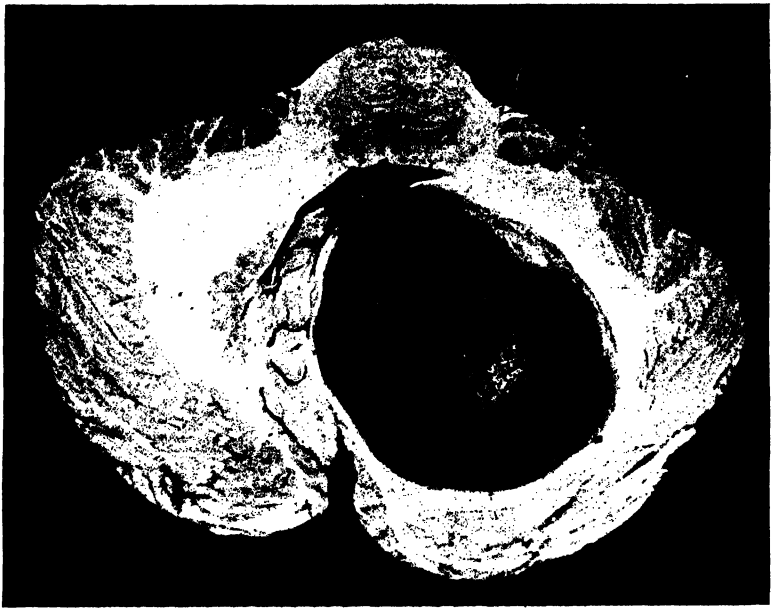


FIG. 76.—Shows a glioma of the right cerebellar lobe which has undergone cystic degeneration. It is displacing the middle lobe and pressing into the fourth ventricle.

Stupor and coma, therefore, are associated with respiratory failure as terminal symptoms.

The tendency for tumours in the posterior chamber to give rise to internal hydrocephalus is in strong contrast to what is seen in tumours of the anterior chambers. Cystic gliomas are of much more frequent occurrence in the cerebellum than in the cerebrum, and this may be partly explained by the smallness of the posterior chamber and the consequent earlier cutting off of the blood supply to the growth, with resulting necrosis and cystic formation. The effect of such cystic formation is to reduce the intracerebellar tension and

alleviate the pressure symptoms with re-establishment of the ventricular and subarachnoid communication. (Fig. 76.)

It is obvious that lumbar puncture as a therapeutic measure in such cases cannot do good, and may possibly even do harm by lessening the spinal resistance in the foramen magnum and permitting the further downward displacement of the bulb.

Tumours of the pons, if of an infiltrating or gliomatous nature, are slowly progressive, and only give rise to increased intracranial tension by obstructing the intraventricular system. In these cases there is a gradual rise in the intraventricular tension: the ventricle most affected being the third, which is the weakest of the supra-tentorial series. Its distension, therefore, may result in direct pressure upon the optic tracts and chiasma. This may account in part for the absence of optic neuritis in such cases, and the not infrequent occurrence of primary optic atrophy.

Tumours of whatever nature may have a fatal effect from their situation; but such cases are rare, *the true cause of death being anæmia of the cardiac and respiratory centres.*

The above facts explain in large part the origin of the so-called false localising signs, which are due to indirect pressure effects.

General Symptomatology

Headache. This is a more or less constant feature, varying in character from a dull aching to a sharp, radiating pain. It may come and go quite suddenly. Change of posture or movement of the head may bring it on. Frequently it is associated with a feeling of nausea or with attacks of vomiting. When the pain is localised to one particular spot, which is tender on pressure, it is evidence in favour of the growth being situated on the surface of the brain, or growing from the dura mater. The chief regions where general headache is complained of are behind the eyes, in both temples, and over the vertex. Occipital pain is suggestive of a growth in the posterior fossa, and unilateral frontal pain of one in the frontal region.

Vomiting. This may or may not develop till late in the history of the case. In its most characteristic form it is

associated with headache, and is independent of the taking of food. There is no pain or retching, and the patient has no nausea afterwards. Vomiting is less frequent than headache, but the majority of cases suffer from attacks of vomiting, often brought on by sudden change of posture or sudden increase in the intracranial pressure.

Optic neuritis. The time at which optic neuritis may develop is variable, as already explained (p. 287). As a general rule optic neuritis occurs early, and is very intense in cases of intracerebellar and intraventricular growths. In cerebral tumours its onset is more gradual, and in extra-cerebellar and intrapontine tumours the patient may die before any swelling of the disc has become visible. There is little relation between the situation of the tumour and the side on which the optic neuritis commences or is more intense. In cerebral tumours, especially of the frontal area, it more frequently commences first in the eye on the same side as the lesion, from which circumstance it may be of some localising value.

A high degree of swelling is quite compatible with good vision, and in many cases it is the ophthalmoscopic examination which first reveals its presence. The subjective sensations, of which complaint may be made, are mistiness before the eyes—often intermittent—and later on transient attacks of blindness. Permanent loss of vision from optic neuritis only commences when secondary atrophic changes take place. As the swelling subsides, the resulting fibrous tissues contract and give rise to degeneration of the nerve fibres. For this reason the longer the neuritis lasts, the more hopeless does the chance of the preservation of vision become.

Convulsive attacks. In addition to the general symptoms, already described, certain others are of common occurrence, and may be either of general or focal importance. Not infrequently, patients suffering from intracranial tumour are subject to various forms of seizures. These may be 'faints,' momentary losses of consciousness, local convulsive movements, or generalised convulsive attacks. Such seizures may simulate idiopathic epilepsy; and in no case should epilepsy be diagnosed until the possibility of cerebral tumour has been excluded. In a general convulsive seizure the patient falls down unconscious, becomes rigid with tonic

spasm, which passes into a fine and rapid clonic stage, and terminates by clonic jerkings, which become more violent and less frequent as the fit passes away. In such attacks the face may first be pale and then livid. During the clonic stage the tongue may be bitten. It is well to state here that not only may general convulsions occur as a symptom of cerebral tumour, but also minor or 'petit mal' attacks.

A focal or Jacksonian seizure is characterised by clonic movements commencing in a definite region of the body, and either confined to that region or spreading from it in a systematic order—'march of the spasm.' Consciousness is not lost until the attack becomes generalised. Such focal attacks may have a sensory or a motor commencement, dependent upon an irritative 'discharge' from corresponding cortical centres.

The importance of focal seizures will be pointed out in the account of the localising symptoms. They have their greatest diagnostic significance when occurring early in the history of the case, but when occurring late may be misleading and of false localising value.

The mode of onset of the focal seizures is of the utmost importance, and, failing this, the observation of the post-convulsive or exhaustion paralysis.

Vertigo. Giddiness is not an uncommon symptom, and often accompanies attacks of headache and vomiting. Its significance as a localising symptom will be considered later.

The mental state. Psychical symptoms are present in most cases of supra-tentorial tumour. The general mental symptoms, arising probably from the increase of intracranial tension which occurs in the later stages of the disease, are dullness, apathy, and irritability of temper, which may be followed by or develop into lethargy, stupor, and eventually coma. On the other hand, it is obvious that tumours situated in the higher psychical centres, or association areas, may be productive of psychical symptoms as early signs of localising value, but which are eventually obscured by those characteristic of the terminal stages of all intracranial new growths. The absence of early mental changes in sub-tentorial tumours is in striking contrast to their early development and persistence in cases of supra-tentorial growths.

TUMOURS OF THE FRONTAL REGION

This region is defined as that portion of the brain lying anterior to the ascending frontal convolution. (Fig. 1.)

The general symptoms of intracranial tumour are present. Headache is usually frontal, but not infrequently occipital in position. When the tumour is near the surface of the brain, localised tenderness on cranial pressure may be present. Optic neuritis may be late in appearing. As a rule it commences first, and has its greatest intensity in the eye on the side of the tumour.

The mental symptoms are of special importance. They are often slight in degree, but are of early onset and precede the general and the localising symptoms. The changes noted are inattention, inability to keep the mind fixed on any subject, loss of memory for recent events, and incoherence in conversation. Sometimes these patients are childish, casual and irresponsible in word and deed, and exhibit a tendency to make silly or pointless jests, without reference to the conversation or subject under discussion. There is frequently an alteration in character, temperament, and tastes—facts which can only be ascertained upon information supplied by relatives or friends. In the later stages delusions, stupor, and dementia supervene.

Irritative symptoms. The following varieties of seizure occur: giddy sensations, 'petit mal' attacks, generalised epileptic convulsions, and fits commencing with localised spasms. The last are of importance, and may be subdivided into two types: (1) those commencing with symptoms of a discharge from the centres in the post-frontal region, and (2) those commencing with symptoms of discharge from the motor centres in the precentral convolution, or ascending frontal gyrus.

1. In the first type, the fit commences with turning or jerking of the head and eyes to the opposite side. These may be the only movements which occur, consciousness being preserved. In such slight attacks the patients may complain of a feeling of rotation towards the side of the lesion, presumably due to the apparent displacement of the environment consequent on the rapid involuntary movements of the eyes. On the other hand, a fit commencing as above described may

spread to the face and mouth, arm, leg, and trunk, or it may become more severe and generalised with loss of consciousness. It is important to note that these attacks are never ushered in by a sensory aura. In addition to the character of the fits, which are frequently unobserved or incompletely described, the phenomena of the post-convulsive stage may be of great importance in the localisation of the disease. After severe seizures the head and eyes are in paralytic deviation to the side of the lesion, and there may be temporary hemiparesis of the contralateral limbs, but the recovery of motor power is rapid. The movement of the head and eyes to the opposite side is the last to recover. Such fits point to the primary irritation and secondary exhaustion of the centres in the post-frontal region. Temporary hemianæsthesia of the contralateral side may be present after a severe fit, but there is never hemianopsia. The loss of sensation passes off quickly.

2. The second type of focal fit occurs when the growth is situated in the left frontal region and the patient is right-handed. The onset is characterised by a sudden loss of speech, followed by twitching of the mouth and face on the opposite side (right), and often accompanied by the involuntary emission of indefinite articulate sounds. Consciousness may remain unimpaired and the patient signal for help, although unable to speak. The discharge may spread to the neighbouring centres, and a Jacksonian fit with the ordinary march supervene. If the fit be severe the power of speech returns slowly, but completely. The aphasia is purely motor in character, the patient being able to understand what is said to him. The association of such attacks with mental symptoms has more than once led to the supposition that a patient was suffering from general paralysis of the insane; but in contrast to what occurs in that disease, recovery from the fit is more rapid and the after-effects are less severe.

The occurrence of fits with focal beginning reveals plainly the side of the tumour, and the absence of a sensory aura makes any question as to the post-Rolandic situation of the growth untenable.

Cranial nerve symptoms are usually absent; but, when present, are due to the direct pressure of the growth. They occur upon the same side as the tumour, and indicate that the growth is situate on, or extends towards, the base of the skull.

Those chiefly affected are the olfactory and the third cranial nerves. The sixth nerve may also be affected, but as a late and indirect symptom. Olfactory symptoms are especially important from the point of localisation; and when other causes may be excluded, anosmia—especially when unilateral —is of especial significance. If the growth is basal and post-frontal, it may press directly upon the optic nerve, resulting in early loss of sight with acute optic neuritis.

The motor system. Paresis occurs only when the tumour impinges on the motor cortex or its efferent fibres, in which case contralateral hemiparesis of varying degrees may result. The most definite weakness is on the opposite side of the face, which seems to be represented more anteriorly than are the limbs, and, as in all cortical lesions, the paresis is greater in volitional than in expressional movement. Apart from such signs, which are not strictly of frontal origin, is the occurrence of tremor on the side homolateral to the tumour. This *tremor* may be observed in both the upper and lower extremities, but is more constant and better marked in the arm than in the leg. In character the tremor is fine, rapid, and vibratory, and may often be more easily felt than seen. It is absent during muscular rest, and can be best demonstrated by making the patient extend both arms horizontally in front of him, with the palms directed downwards and the fingers extended. It will then be noticed that the homolateral arm and hand are in a state of constant fine vibratory tremor, in contrast to the contralateral arm, in which such tremor is absent. This tremor may not be constantly present in any given case. It occurs in cases in which there is no evidence of interference with the motor system—such as paresis, as well as in cases where such paresis exists. In a few cases such tremor was observed in both hands, but it was more regular and constant and of the typical vibratory nature in that homolateral to the lesion.

Sensory system. Affection of sensation never results directly from a frontal lesion, and only occurs as a temporary phenomenon after severe fits, when the discharge has presumably passed across the motor centres to the post-Rolandic gyrus.

Reflexes. *Deep reflexes.* Tumours limited to the pre-frontal region do not as a rule cause any change in the deep

reflexes, but when the growth extends further posteriorly and impinges on the motor area, the contralateral deep reflexes are increased. When there is a considerable increase in the intracranial pressure, the deep reflexes are increased on both sides.

Superficial reflexes. The examination of the superficial abdominal reflexes is of the greatest importance, as they may be definitely affected in cases in which hemiparesis or other focal signs are absent. The first sign is absence or diminution of the epigastric or abdominal reflexes on the side opposite to the tumour, or on repeated stimulation the contralateral reflexes are more easily exhausted than the homolateral. This alteration of the superficial abdominal and epigastric reflexes is not a specific sign of frontal lesions, but merely the result of an affection, however slight, of the pyramidal system. Its value lies in the fact that it is the earliest demonstrable sign of a commencing hemiparesis.

Still more important as positive evidence of affection of the pyramidal system, is the presence of an extensor response on stimulation of the sole of the foot. In cases of frontal tumour the contralateral plantar reflex, when not extensor, may be of an indefinite type as compared with that of the homolateral side; when this is associated with loss or diminution of the corresponding abdominal reflexes, its significance is that of a typical extensor response.

It is unnecessary to dwell on the local external signs, such as localised tenderness on pressure over the skull, protrusion of the eye, or bulging of the cranium, though these may be of value in diagnosis.

TUMOURS OF THE PRE-CENTRAL REGION (MOTOR AREA)

The pre-central region consists of the ascending frontal convolution and its continuation upon the mesial surface. (Figs. 1, 2, and 3.)

The *general* symptoms are rarely absent when the tumour is situated in this locality; but as this region contains the motor area the focal symptoms usually occur early in the course of the disease and are invariably present.

The *focal* symptoms are of two kinds—irritative and paralytic.

Irritative signs. Convulsive seizures. If the growth is situated on or near the surface, convulsive attacks are frequent, and generally have a local commencement. The site of the tumour in the face, hand, arm, trunk or leg areas on one side of the brain determines the onset of the fit in the

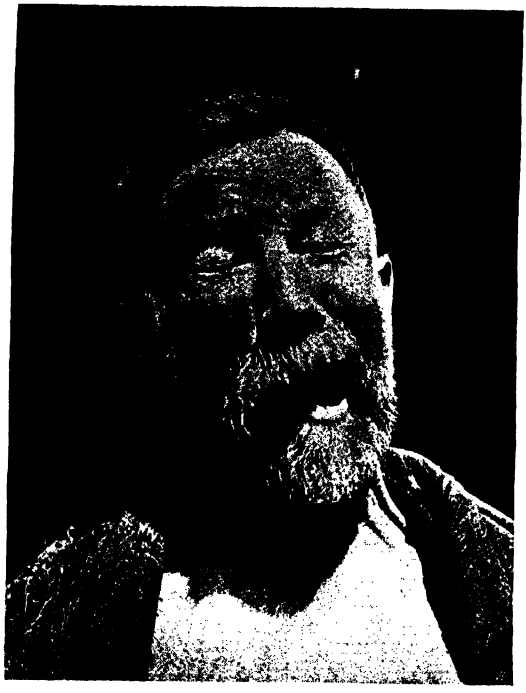


FIG. 77.—Shows a focal or Jacksonian fit involving the left side of the face.

muscles of the face, hand, arm, trunk or leg respectively of the opposite side of the body. These seizures are motor in character, but owing to the proximity of the sensory cortical centres behind the fissure of Rolando, sensory symptoms not infrequently coexist. (Fig. 77.)

A fit with focal commencement may result in a generalised epileptic seizure, and may determine the onset of epileptic attacks. A localised convulsion usually gives rise to a temporary mono- or hemiplegic weakness of the parts convulsed.

Paralytic phenomena. Progressive monoplegia or hemiplegia of the opposite side of the body is found in all cases of precentral tumour. This may be observed after a fit, or series of fits, and although at first transient soon becomes permanent and progressive. This is especially true of cases with a definite and constant local origin, the palsy being earliest observed in that part of the body in which the fit

FIG. 78.—Showing a subcortical tumour into which a hemorrhage has taken place.

commences, but the presence of such paralyses does not necessarily preclude the onset of further fits in the paralysed parts.

If the tumour is subcortical, convulsive attacks are less common, and if present, are general in character, and there is a greater tendency to progressive hemiplegia with spasticity. Rapid wasting of the hemiplegic limbs, which however remain spastic, is found in many cases of subcortical new growth.

It ought to be borne in mind that a sudden onset of hemi-

plegia does not negative the existence of a cerebral tumour, as hemorrhage into the tumour or surrounding tissue may occur. (Fig. 78.)

The close proximity of the post-frontal and post-central regions to the motor area proper (precentral) renders them liable to suffer, so that psychical and sensory changes are not uncommon. The fits are often associated with numbness or

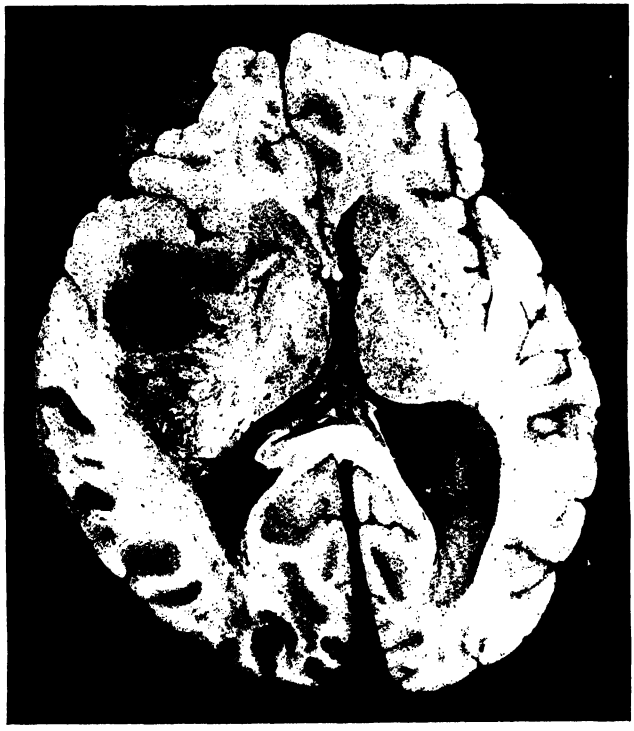


FIG. 79.—Showing a subcortical tumour involving the centrum ovale, and lenticular nucleus on the left side. There is considerable distension of the lateral ventricles, and the brain is pushed over towards the right side.

tingling in the parts convulsed, and temporary hemianæsthesia may coexist. In subcortical tumours permanent loss of sensation is frequent, owing to the propinquity of the motor and sensory paths, but hemianopsia is not present.

Motor aphasia of varying degrees may be observed when the tumour is situated in the left hemisphere: when the mental condition appears to be more affected than really is the case.

Reflexes. On the paralysed side the tendon jerks are increased, the plantar is of the extensor type, and the superficial abdominal reflexes are absent.

TUMOURS OF THE POST-CENTRAL AND PARIETAL REGIONS (SENSORY AREA)

This region embraces the ascending and superior parietal convolutions. The separation of the post-central convolution into a primary sensory portion lying in and immediately behind the fissure of Rolando, and a 'psycho-sensory' in close relation with the superior parietal lobule, has been already mentioned.

The post-central area has immediately in front of it, just across the fissure of Rolando, the pre-central or motor area. Posteriorly are the higher visual centres in the angular gyrus, and inferiorly, within and across the fissure of Sylvius, is the auditory centre in the superior temporal convolution.

It is therefore obvious that the extension of a tumour growth from the parietial region in a direction either forwards, downwards, or backwards will give rise to symptoms corresponding to the invaded area.

In man clinical evidence points to the post-central gyrus as the cortical area for the primary recognition of the senses of active and passive movement, of tactile localisation and discrimination, and possibly to a less extent of painful and thermal sensibility.

The representation of the sensibilities of active and passive movement and tactile localisation would further appear to be situated posterior to the corresponding motor areas and in close connexion with them.

The examination of cases in which there have been cortical lesions situated in the post-central gyrus and parietal region has demonstrated the following facts as regards the quality and distribution of the sensory loss: (1) Tactile localisation and the sense of movement are most impaired, even to complete abolition. Painful and thermal sensibility may not be affected at all; when affected there is not complete loss. A relative diminution of deep and painful pressure sensibility may also be present.

2. The sensory loss is more pronounced in the distal

than in the proximal parts of the limb. The affected skin areas have no relation to a peripheral or spinal sensory distribution, but appear to be sensory analogues of the motor disability following upon lesions of the corresponding areas the pre-central gyrus. The extent of the sensory loss may be restricted to more or less definite areas—such as the hand, a limb, or one side of the body. Where such sensory loss is well defined, all forms of sensibility are more or less affected.

According to Mills,[1] the *stereognostic sense*, or sense of recognition of the size, shape, and consistency of objects placed in the hand, is situated in the posterior part of the superior parietal lobule.

The idea of a stereognostic centre presumes an intact condition of the cutaneous and muscular sensibilities. The occurrence of cases, therefore, in which astereognosis is found without any other objective sensory loss, would favour the existence of such a centre. We have, however, been unable to satisfy ourselves, either by personal observation or from a study of the literature, that astereognosis exists without some defect of the primary sensibilities.

The *general* symptoms of intracranial tumour are present.

The *focal* symptoms of a tumour, strictly limited to this region, consist of loss or impairment of cutaneous sensibility and of astereognosis on the opposite side of the body. Cutaneous sensibility is rarely completely lost, but tactile sensibility—especially that to cotton wool—is impaired to the greatest extent. The senses of active movement and of passive position of the limbs are also impaired by lesions in this locality.

Irritative symptoms. These consist of a sensation of numbness and of tingling commencing locally in some portion of the body, but spreading so as to become general over the whole of the opposite side. It is rare for a seizure commencing with a local sensory aura to remain purely sensory, for the cortical discharge spreads to the corresponding motor area, and Jacksonian motor attacks follow immediately in the wake of the subjective sensation.

These phenomena were observed in three cases. In one, in which a glioma was limited to the post-central gyrus

[1] Mills, *Univ. of Pennsylvania Med. Bulletin*, 1906.

opposite the hand and face areas, the fit commenced with a subjective sensation of numbness, tingling, and 'drawing' of the face and fingers on the opposite side. Consciousness was retained, and, in answer to questions, the sensation was described as having reached the elbow thirty-five seconds from the commencement of the attack. The motor convulsion then started in the face and hand, and rapidly spread to the arm, trunk, and leg, and later became generalised, with loss of consciousness. On recovery the patient stated that the subjective sensation had reached the shoulder before the motor convulsion in the face, arm, and forearm had commenced. After the seizure, partial hemiplegia—chiefly in the hand and face—with complete hemianæsthesia to all forms of sensation, was detected. The deep reflexes were exaggerated, the abdominal reflexes were absent, and the plantar response was indefinitely extensor. Eventually there was complete return of motor power, and sensation became normal, except in the hand.

In two cases, in which a tumour was found in the ascending parietal gyrus—opposite respectively the leg and trunk areas of the motor zone—the fits were at first characterised by a sensation of movement in the foot and abdomen upon the opposite side, although no obvious movement was detected. Later on, in both cases, motor Jacksonian seizures appeared, commencing in those portions of the body to which the subjective sensations were referred.

Paralytic symptoms. These consist of loss or impairment of sensation upon the opposite side of the body, more particularly of tactile sensibility and the sense of passive position. Frequently vaso-motor disturbances, consisting of either elevation or depression of the surface temperature, are observed.

Motor system. Except in the early stages, motor weakness of the opposite limbs is present. Later on this may become considerable, and is not infrequently attended by some degree of ataxia. A further feature is the tendency to a somewhat pronounced wasting of the muscles of the paralysed limbs with rigidity, but occasionally with flaccidity. In the latter type, an increased myotatic irritability is observed.

Reflexes. Loss or diminution of the superficial reflexes is almost constant on the opposite side. The deep reflexes

vary; in our experience they have always been increased on the opposite side, but some observers (Mills and others) have described their diminution or absence.

TUMOURS OF THE OCCIPITAL LOBE

The *general* symptoms of intracranial tumour are usually present. Headache is occipital in position and optic neuritis is often intense.

The *focal* symptoms of a tumour of the occipital region are, subjective and objective disturbances of vision in the homonymous fields opposite the lesion.

The subjective symptoms consist of visual sensations, usually of light or of colours, sometimes accompanied by a feeling of numbness or paræsthesia in the limbs on the side of the body opposite to the lesion. The visual hallucinations usually have a hemianopic distribution. A combination of subjective visual symptoms and numbness may be mistaken for migraine.

Sooner or later, however, loss of vision of a definitely hemianopic type occurs. The subjective sensations may persist even after the development of the objective loss, but tend gradually to disappear. Although the defect of vision is usually hemianopic involving the half-fields, the loss is sometimes limited to one or other visual quadrant; but this is, perhaps, less frequent in tumours than in vascular lesions.

Although unaware of the hemianopic nature of the visual defect, the patient may early complain of loss of sight. On examination of the fundus oculi, optic neuritis may be observed. It is, therefore, important to test the visual fields, as it is easy to attribute the defect of sight to the influence of the local neuritic condition.

If the tumour is situated on the mesial aspect of the occipital lobe, homonymous hemianopsia to the opposite side is present. This is due to destruction of the primary visual centre situated in the lips of the calcarine fissure. Irritative symptoms, when present, are crude—such as flashes or sparks of light.

In some cases, where the growth is situated well forwards and encroaches upon the under surface of the occipital lobe, cerebellar symptoms may be present from direct pressure upon the cerebellum. The 'symptom-complex' in these cases

consists of homonymous hemianopsia, hemianæsthesia, and slight motor weakness on the contralateral side, in association with ataxia, hypotonia, and nystagmus, slower and more marked towards the side of lesion on lateral conjugate movements on the homolateral side. These last symptoms arise from pressure upon the lateral lobe of the cerebellum.

If the tumour is situated on the outer aspect of the occipital lobe, subjective symptoms of a more specialised type usually occur early; while the extension of the growth forwards along the convexity results in a mind-blindness, and word-blindness from involvement of the higher visual centres in the angular gyrus.

Hemianæsthesia and hemiplegia are also produced by a forward and inward extension of a tumour situated subcortically in the occipital lobe. These signs may be present without any subjective visual sensations.

Convulsive seizures may also occur in cases of tumour of the occipital region. These are either general or limited to the limbs on the opposite side, in consequence of an increase of the intracranial pressure. Localised convulsions, preceded by a sensory warning, may also occur.

The pupillary light reflex is unimpaired in cases of tumour limited to the occipital cortex or subcortical tissues.

Reflexes. As in all cerebral tumours, a loss or diminution of the superficial reflexes on the opposite side may be one of the earliest signs, even when hemianopsia is the only physical sign. In the later stages, with the occurrence of obvious motor symptoms, the deep reflexes are increased and a plantar extensor response appears upon the opposite side.

TUMOURS OF THE TEMPORO-SPHENOIDAL LOBE

For purposes of localisation this lobe is divided into two portions: (*a*) the tip of the lobe upon the mesial aspect of the brain—the uncinate gyrus; and (*b*) the superior temporal gyrus upon the convexity.

The *general* symptoms of intracranial tumour are present in most cases.

The *focal* symptoms are separately described, according as the lesion is situated in the uncinate region, or in the superior temporal gyrus.

1. The uncinate gyrus

A tumour situated in this locality reveals itself by the onset of seizures having a warning of a subjective sensation of smell or flavour. The sensations are sometimes of an unpleasant or perverted character, and may be so real to the patient that he dreads associating with others. The attacks are characterised by the crude warning sensation just described, which may or may not be associated with a 'dreamy state.' The dreamy state may be of the nature of a 'reminiscence'—as if that which is happening has been previously experienced; or of a feeling of unreality—as if surrounding objects were unfamiliar. An intense feeling of fear or of impending death may be associated with it. During the seizure smacking movements of the lips, chewing movements of the jaw, and sometimes spitting have been observed. Occasionally when the attack is over, the patient has a subjective sensation of intense hunger, or a desire to go to stool. During the unconscious stage, the arm on the side opposite the lesion may be moved about in a slow, deliberate, and apparently purposive manner.

These attacks may be succeeded by transient, bilateral, incomplete loss of the senses of taste and smell. In cases with destructive lesions of the uncinate lobe, there is never complete loss of taste or smell, but a bilateral impairment is not uncommon.

2. Superior temporal gyrus

In the early stages or in localised lesions, subjective sensations of sound are heard by the patient. When the tumour is more extensive, convulsive movements—often limited to the face and arm of the opposite side, but sometimes becoming unilateral with loss of consciousness—may occur, and are due to irritation of neighbouring centres; generalised epileptic seizures are also sometimes present. Incomplete cortical deafness of the opposite ear may also be observed, both after the seizures and as an independent focal symptom.

In right-handed persons a focal lesion of the left superior temporal gyrus causes not only cortical deafness in the opposite ear, but also word-deafness. A point of importance in differential diagnosis is the fact that, in abscess of the

temporo-sphenoidal lobe arising from unilateral suppurative middle ear disease, deafness due to the cortical lesion is in the ear opposite that which is diseased.

The *cranial nerves* are not usually affected.

Motor system. Fits when present commence in the face or arm. In the early stages paresis of the face or arm may be observed, which progresses to hemiplegia, if the tumour is of considerable size.

Reflexes. One of the earliest signs of tumour in this region is impairment or loss of the superficial abdominal reflexes on the opposite side. The deep reflexes may be increased, but have been found to be diminished in many cases, and a plantar extensor response develops in the later stages.

Sphincters. Sphincter control over the bladder and rectum may be impaired from lack of voluntary control, owing to the mental condition.

TUMOURS OF THE MIDBRAIN AND SUBTHALAMIC REGION

In addition to the *general* symptoms of intracranial tumour, more or less definite *localising* signs may be observed. When present they are found upon the same side as the lesion. They are :—

1. Weakness of the upward movement of the eyeballs.
2. Defective pupillary light reaction.
3. Ectopia or eccentric position of the pupil.
4. Paresis or paralysis of the third cranial nerve.
5. Defective sensibility over the fifth cranial nerve area.

Hemiplegic weakness, accompanied by more or less rhythmical tremor and ataxia, may be found in the limbs on the side opposite the lesion. Sometimes an associated hemianæsthesia is also observed. Occasionally these signs are bilateral. Should hemiataxia and tremor be present in the limbs upon the same side as the tumour, they are due to an associated involvement of the cerebellar and rubro-spinal systems.

Various forms of seizure may be associated with tumours in this locality. Sometimes generalised convulsive attacks are observed, at other times impairment of consciousness, which may deepen into coma, and be accompanied by transient residual third-nerve paralysis.

TUMOURS OF THE LATERAL VENTRICLE

The onset of the *general* symptoms, which are intense, is rapid. Definite *local* symptoms are often absent or delayed; but in all cases the supra-tentorial situation of the growth is made clear by the early diminution of mental acuteness, often

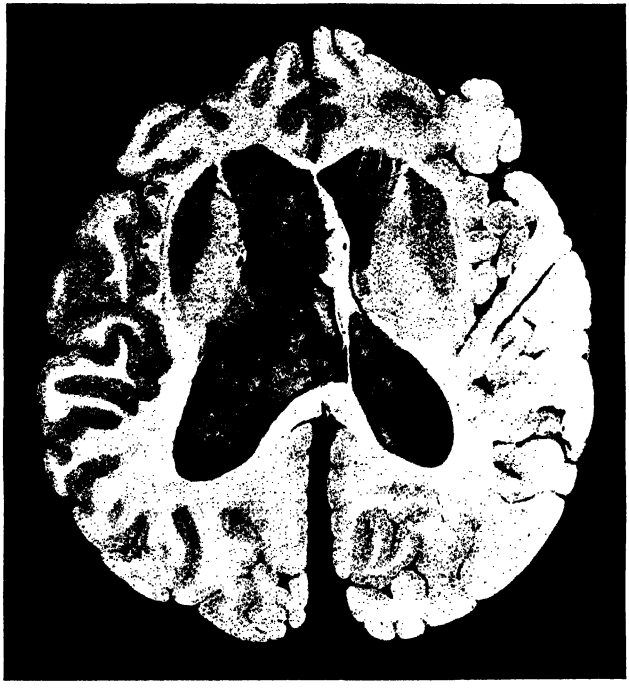


FIG. 80.—Shows a tumour of the choroid plexus involving the left lateral ventricle.

associated with psychical symptoms resembling those seen in cases of tumour of the frontal lobe. In addition, slight bilateral spasticity—sometimes more marked on the side opposite the tumour—an increase of the deep reflexes, diminution of the superficial reflexes, with either flexor or extensor plantar response, are observed. When an extensor plantar response is present, some degree of motor weakness of a spastic type will be detected. In the later stages, false localising signs arise from the increasing pressure in the infra-tentorial chamber. These are: giddiness, unsteadiness,

and general ataxia, sometimes associated with nystagmus and paresis of the sixth nerve. In the terminal stages symptoms of bulbar anæmia, characterised by respiratory and vaso-motor paralysis, foretell the impending death. (Fig. 80.)

TUMOURS OF THE THIRD VENTRICLE

The *general* symptoms may be of early or late onset The cerebral symptoms are more or less general and bilateral in character and consist of mental impairment, bilateral

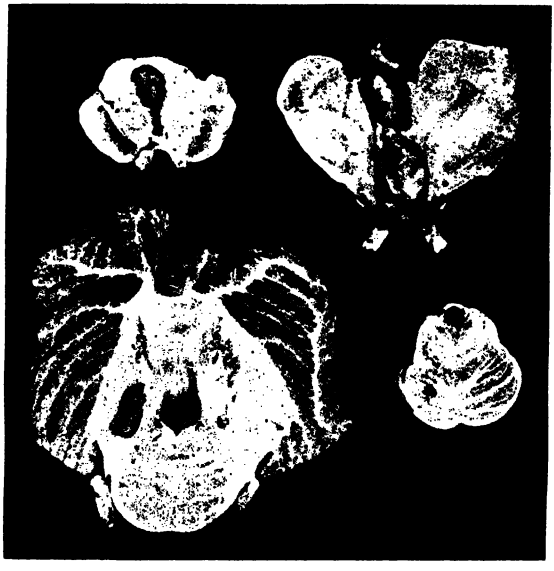


FIG. 80A.—Illustrates a tumour of the third ventricle, spreading downwards along the 'iter' and appearing in the fourth ventricle.

spasticity with some motor weakness, increase of the deep reflexes, diminution of the superficial reflexes and extensor plantar responses.

The only *localising* signs present in these cases are: (1) the early impairment of the reaction of the pupils to light; (2) the associated weakness of the upward movements of the eyes; (3) the occasional occurrence of ectopia pupillæ; (4) the rapid failure of vision at a time when the appearance of the fundus oculi indicates an acute stage of optic neuritis; or (5) early failure of vision without optic neuritis.

The first three symptoms are due to pressure upon the third nerve nucleus; the last, to pressure upon the optic chaisma by the anterior end of the distended third ventricle. (Fig. 80A.)

TUMOURS OF THE FALX CEREBRI (INCLUDING THE MESIAL ASPECT OF THE CEREBRUM)

Tumours in this locality are of slow growth, and are either endotheliomatous or fibro-sarcomatous in character,

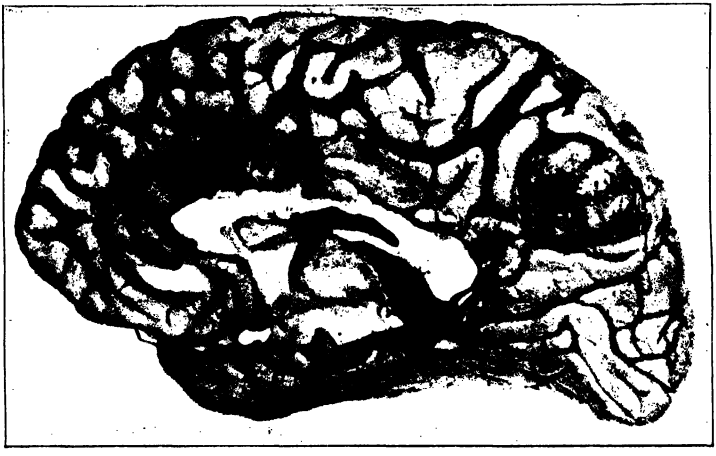


FIG. 81.—Shows a localised, circumscribed tumour involving the precuneus.

and tend to displace rather than infiltrate the cerebral substance.

The *general* symptoms are of slow development; but headache commences well in advance of vomiting and optic neuritis. The headache is commonly vertical in position and attended by pain on pressure.

The *focal* symptoms are early and are at first of an irritative character. The nature of the seizures depends upon the situation of the growth. If this is situated in the anterior half of the falx, bilateral and general epileptic attacks are present, sometimes on one side and sometimes on the other. These may be accompanied by an early development of mental failure. The clinical picture is that of a bilateral

frontal lesion, and may be readily mistaken for general paralysis of the insane.

If the tumour grows from the posterior half of the falx cerebri, the irritative focal symptoms consist of Jacksonian attacks, commencing simultaneously in both lower extremities, or they may commence first in one and then involve the other leg, or begin alternately in one or other foot.

The recurrence of such attacks is followed by a gradual and progressive paralysis of both lower limbs. It is rare for the motor system to be alone affected, sensory phenomena, both irritative and paralytic, occurring simultaneously.

The deep reflexes are increased on both sides, especially in the lower limbs, along with double extensor response and absence of the superficial abdominal reflexes.

In rare cases visual phenomena in the form of subjective flashes of light have been observed, but changes in the visual fields have never been definitely detected. In one case in which these symptoms were present, the mental condition and a well-marked degree of secondary optic atrophy precluded any trustworthy objective investigation.

Occasionally the growth extends entirely into one hemisphere, in which event, although the irritative symptoms may be bilateral, the paralytic phenomena are predominately on one side.

TUMOURS OF THE CORPUS CALLOSUM

The *general* symptoms are early and well defined; optic neuritis may be intense and mental symptoms pronounced.

A rapid loss of vision takes place in cases where the anterior portion and the knee of the corpus callosum are affected. This is a striking feature and a point of diagnostic importance, as the well-marked optis neuritis associated with blindness, but without atrophic changes in the disc, is quite unusual, and is to be accounted for by direct pressure of the growth upon the optic tracts.

General epileptic seizures are not uncommon, and paralysis of one or other, or both, sides of the body develops as the tumour infiltrates the centrum ovale. A particular feature observed on the hemiplegic side is the difficulty which the patient experiences in relaxing his grasp from any object

which he may have in his hand. Apraxia, or the inability to use an object for its proper purpose—although its name and the purpose for which it is used are known—is sometimes found, and when present may be regarded as a diagnostic sign of importance in lesions of this structure.

TUMOURS OF THE PITUITARY BODY

Tumours of the pituitary body may or may not be associated with acromegaly, and may or may not be characterised by the signs of intracranial tumour. Two types may be described.

In the *first type* there is a bilateral temporal hemianopsia with pallor of the inner (nasal) half of the optic discs, without evidence of optic neuritis. The general symptoms may be absent, but some degree of mental impairment is usually present—chiefly in the direction of loss of memory and slow cerebral action. The motor, sensory, and reflex systems show no change.

A *second type* is found in which the general symptoms of intracranial tumour are well marked; frontal headache, optic neuritis, and mental impairment being early and prominent features. In these cases acromegaly never develops, and the loss of vision is rapid. If the case is seen in the early stages, bitemporal hemianopsia may be observed; but this quickly gives place to blindness, affecting one side in advance of the other, so that temporal hemianopsia is found on one side and loss of vision on the other. This stage soon passes into that of complete blindness, and if now examined, the degree of blindness is out of all proportion to the changes in the fundus of the eye, little or no atrophy being detected. The mental symptoms are obtrusive—inattention, loss of memory, childish behaviour, and rapidly developing dementia.

In addition to the visual defects, interference with the functions of the third and sixth nerves on one or both sides is frequently observed. Occasionally pain over the ophthalmic branch of the fifth nerve is described. The other cranial nerves are not affected, except loss of smell, which may be bilateral and complete.

Generalised epileptic fits, muscular hypertonicity, and slight spasticity of the limbs without obvious paresis, are

occasional symptoms. Sensation is not affected. The deep reflexes are exaggerated, the superficial are diminished along with flexor or indefinite plantar responses.

Sphincter action is not impaired, but, owing to the mental condition, there is a want of control over the evacuations.

TUMOURS OF THE CEREBELLUM

The *general* symptoms of intracranial new growth are well-marked and early features. Headache is a constant and early symptom, being as a rule most intense and persistent in the occipital region. In a few cases it is frontal in position; but it is not uncommon for complaint to be made of pain radiating down the back of the neck. It may be associated with attacks of vomiting. Optic neuritis is an invariable accompaniment of cerebellar tumour. It is an early sign, intense in character, and often quite out of proportion to the localising symptoms. Its onset is acute, vision is impaired early—either temporarily or permanently—from consecutive atrophic changes in the optic nerve. In character the neuritis is similar to that seen in other intracranial tumours; but in some cases the appearance of the fundus resembles that seen in albuminuric retinitis. Vertigo or giddiness is another characteristic symptom, and may consist merely of an indefinite sensation of unsteadiness, or of a definite subjective sense of movement towards the sound side. External objects also appear to move in the same direction.

Cranial nerves. Symptoms referred to the cranial nerves are chiefly ocular in character, and consist of slight paresis of the external rectus muscle on the side of the lesion. This may occasionally give rise to diplopia, which is as a rule transitory, and unaccompanied by obvious strabismus. A slighter degree of paresis of the opposite external rectus may sometimes be present. Occasionally some weakness of conjugate movement towards the side of the lesion—particularly a want of sustained movement—may be observed. In cases with optic atrophy, the eyes when at rest are usually deviated towards the sound side.

In some cases, after acute attacks of vomiting and headache, the eye on the side of the lesion may be directed downwards

and inwards, and the others upwards and outwards. This is known as 'skew deviation,' and is probably an irritative rather than a paralytic symptom. (Fig. 82.)

Nystagmus is always present and is a valuable localising sign. In typical cases, it consists of slow and deliberate jerkings of both eyes on lateral conjugate movement to the side of the lesion; these are in marked contrast to the finer and more rapid movements, which take place on conjugate deviation to the opposite side. Nystagmus may also be present, but less well marked on upward and downward movements of the globes, and consists of rotatory upward and downward movement to the side of the lesion.

FIG. 82.—Showing 'skew viation' from lesion of cerebellum.

The pupils may be dilated, and in cases with pronounced optic atrophy the light-reflex is lost.

In rare cases of tumour of the lateral lobe the sensory division of the fifth nerve may be implicated. The anæsthesia is incomplete, and the motor root is never involved.

Occasional and slight weakness of the facial muscles on the side of the lesion may be noticed.

Subjective auditory symptoms are rarely present, and an affection of hearing is no part of the cerebellar 'syndrome.'

The other cranial nerves are not affected.

Motor symptoms. *Paresis* of slight degree, but of definite character, is present in the limbs on the side of the lesion. This weakness is most marked in acute cases and is best seen in the muscles of the back. A defective muscular tone (*atonia*) is associated with the muscular paresis of the homolateral side. The limbs when handled are found to be unusually limp and flaccid, and the muscles are soft and flabby. Atonia of this type may coexist with increased tendon reflexes—a point which is in striking contrast to the atonia seen in tabes dorsalis.

Ataxia is more or less characteristic. It is present in the homolateral musculature, although that on the opposite side

may be slightly affected. It is due to a lack of accurate co-operation and association of the synergic muscles effecting muscular movements, and not to the impairment of the muscular sense or sense of position of the limbs, as occurs in tabes, and it is not increased on closure of the eyes. Hence it is only present during the performance of a movement. In the 'finger-nose test,' for example, the ataxic deviation of the finger is marked at the outset, but becomes less pronounced towards the completion of the act, and finally ceases. The ataxia is most marked in the trunk and lower limbs.

FIG. 83.—Showing the attitude of the head in a case of cerebellar disease.

Attitude. Considerable attention has been devoted to the position of the head in cerebellar disease. When standing or sitting the head is generally tilted so that the occiput approximates to the shoulder on the side of the lesion, and the chin is deviated to the opposite side. But the converse position has also been described. When standing, the homolateral leg is slightly everted and abducted, so as to broaden the supporting base; the homolateral shoulder is held higher than its fellow, and there is definite lordosis.

The *gait* of cerebellar disease is described as reeling, staggering, or drunken. There is a tendency both to stumble and fall, as well as to deviate to the side of the lesion. The patient is fully conscious of this deviation, and endeavours to overcome it by rotation of the body towards the sound side.

There is a greater tendency to fall to the side of the lesion than to the opposite side, and if left alone the patient would tend towards a 'circus movement' to the side of the lesion. This, however, is corrected automatically by rotation of the whole body towards the sound side, which if overdone leads to an appearance of staggering and deviation towards the sound side. In any doubtful case it is advisable to test the

patient by asking him to stand on either leg alternately. By this means the greater weakness and unsteadiness of the affected side becomes apparent.

Reflexes. The deep reflexes are variable: sometimes diminished, at other times exaggerated, and often changing from day to day. The superficial reflexes are not affected, and the plantar reflexes are always flexor in uncomplicated cases. Sphincter control is not impaired.

Cranium. In children, bulging in the occipital region over the seat of the tumour has been observed.

Mental symptoms are absent until the intracranial pressure has risen sufficiently to cause impairment of the cerebral functions.

Tumours of the middle lobe or vermis cerebelli

The *general* symptoms are similar to those already described under Tumour of the Lateral Lobe. The ocular symptoms are bilateral, and consist of weakness of both external recti muscles, and of the conjugate lateral movements of the eyes; slow nystagmoid jerkings, equal in degree, occur on looking to either side. Inco-ordination and ataxia are bilateral, and most marked in movements in the antero-posterior axis. The patient when standing may fall either forwards or backwards, and also when he suddenly halts. In some cases the head is retracted. The gait is reeling equally to either side, with a sprawling action of the limbs.

Owing to the tendency of middle-lobe tumours to invade the fourth ventricle, internal hydrocephalus is developed early and indefinite spastic symptoms and irregular tremors complicate the clinical picture.

TUMOURS OF THE FOURTH VENTRICLE

General symptoms. Headache, vomiting, and giddiness, develop early, and are more or less constant. Headache is usually occipital in position, and may be associated with stiffness of the neck and pain on movement. Optic neuritis may appear early or late, but its development is rapid. Giddiness is general in character and unassociated with auditory phenomena. These symptoms may be present for one or two years, and local signs are strikingly in abeyance.

Loss of vision may occur early, before the advent of optic neuritis, and is probably due to dilatation of the third ventricle causing direct pressure upon the optic chiasma.

Mental changes are rarely in evidence until the terminal stages.

Examination of the *cranial nerves* reveals nystagmus on lateral deviation of the eyes to either side, similar in character and degree; paresis of both external recti muscles, weakness of the conjugate lateral movements of both eyes, and sometimes slight bilateral weakness of the lower facial muscles.

The absence of interference with the other cranial nerves is significant, although a temporary and variable inequality of the pupils may be observed. Paralysis of the third nerve with loss of the pupillary light reaction only occurs with grave secondary complications.

Motor system. Slight muscular hypertonicity associated with inconstant instability of gait is a characteristic feature and may persist for months. The onset of definite spasticity and of persistent inco-ordination indicate respectively implication of the pons and cerebellum. As a rule there is no demonstrable paresis of the limbs.

Sensory system. There is no alteration of sensibility.

Reflexes. The deep reflexes are brisk or exaggerated equally on the two sides. This may not be accompanied by any change in the superficial abdominal or plantar reflexes.

The absence of definite spasticity, of paresis, or of changes in the reflexes, in cases which at autopsy show not only internal hydrocephalus, but flattening and distortion of the pons and medulla, is accounted for by the fact that the increase in pressure has been sufficiently gradual to permit the circulation in these parts to adapt itself to the increased intracranial tension.

In some cases sugar is found in the urine.

In the terminal stages death ensues from the formation of a pressure cone within the foramen magnum, resulting in respiratory paralysis.

Cysticercus of the fourth ventricle is by no means rare, and has a slightly different symptomatology from that given above. The distinguishing features are: the long duration of the symptoms without definite physical signs, the sudden

onset of attacks of headache, vomiting and giddiness, the association of such attacks with sudden movements of the head, the long intermissions with complete freedom from all symptoms, the more frequent occurrence of glycosuria, the late onset of optic neuritis, and the frequency of sudden death.

In all cases presenting such symptoms the possible presence of a tape-worm (*tænia solium*) should be inquired into and the risk of infection from the mode of living considered.

The duration of the symptoms in tumour of the ventricle varies from five months to two years; but cases of cysticercus have been known to live for several years.

EXTRA-CEREBELLAR TUMOURS (TUMOURS OF THE CEREBELLO-PONTINE ANGLE)

Under this term are included those tumours which grow in the posterior fossa of the skull between the pons and the cerebellum, and originate commonly in connexion with the eighth cranial nerve, and less frequently with the fifth and seventh nerves. These tumours are usually of firm consistence and slow growth, and involve the pons and cerebellum secondarily by pressure. (Figs. 84 and 85.)

In contradistinction to intra-cerebellar growths, the general symptoms are late in appearance. As a rule, headache sets in first, and is nearly always associated with pain in the neck. Optic neuritis may be late in appearing or absent; when present it is either acute or subacute in degree.

The symptoms produced by such tumours are referred mainly to the cranial nerves implicated by the growth, and in lesser degree to the lateral lobe of the cerebellum and the pons. Unilateral cerebellar symptoms are therefore observed on the side of the lesion (paresis, ataxia, and atonia) combined with a spastic paresis of the opposite limbs from pressure upon the pyramidal fibres.

The *cranial nerve* symptoms are:—

(*a*) Paresis or paralysis of the sixth nerve, more complete than in intra-cerebellar growths, and nystagmus having the characters already described (p. 263).

(*b*) Paresis or paralysis of the fifth nerve—more especially its sensory division.

(*c*) The facial nerve is almost invariably affected at some stage in the course of the disease, when facial palsy of a peripheral type develops. It is surprising what great and long-continued pressure may be exerted on this nerve before clinical symptoms are produced.

(*d*) Nerve deafness on the side of the lesion is the earliest,

FIG. 84.—A case of extra-cerebellar tumour, growing in the right cerebello-pontine angle, displacing and compressing the pons and cerebellum.

most definite, and most constant symptom of the extra-cerebellar growths. It may precede the onset of the other symptoms by months or years. Noises in the ear—tinnitus aurium—are invariably associated with it. Vertigo differs from that of intra-cerebellar tumour, in that the subjective feeling of rotation is always from the sound to the affected side.

(*e*) In rare instances the bulbar nerves have been implicated on the side of the lesion.

Motor system. On the homolateral side a slight degree of paresis associated with definite atonia and ataxia is present. These symptoms are due to the pressure of the tumour upon one lateral lobe of the cerebellum. On the contralateral side considerable paresis of a spastic type may be present, owing to pressure on the pyramidal fibres above their decussation. In

FIG. 85.—Bilateral extra-cerebellar tumours.

old standing cases, when the pressure of the tumour is exerted on both sides of the pons, bilateral spasticity may be observed. The motor functions being affected in these two ways, the clinical picture is one of instability with paresis resembling disseminated sclerosis. The gait is of the ataxic spastic

paraplegic type: the ataxia being greater in the homolateral leg, the spasticity and paresis in the contralateral. (See Table, p. 428.)

Sensory system. Sensory changes are not present on the body or limbs.

Reflexes. The deep reflexes are increased, especially upon the contralateral side, where clonus may be obtained.

The superficial reflexes are diminished on the contralateral side, and associated with an extensor plantar response. Similar changes in the superficial reflexes of the homolateral side are only observed in the late stages.

The sphincter control may also be interfered with in cases with bilateral spastic symptoms.

TUMOURS OF THE PONS VAROLII

The most common tumours of this region are glioma and tubercle; sarcoma and gumma may also be found. Pontine tumours are more common in children than in adults.

The symptoms may best be described with reference to the two common kinds of tumour observed in this locality.

Glioma. The pathological appearances presented by these tumours led to the use of the term 'hypertrophy of the pons.' The general shape of the pons may be preserved, or the tumour may extend from the surface and completely surround the basilar artery and the emerging nerves. The symptoms presented during life are not proportionate to the extent of the pathological changes: a phenomenon which may be explained by the fact that, by Bielschowsky's method of staining nerve fibres, the axis-cylinders of the nerve fibres are not found destroyed by the new growth.

The *general symptoms* are, conspicuously absent. Headache only sets in with the development of internal hydrocephalus, and optic neuritis is often absent or only appears in the terminal stages. Optic atrophy, without obvious evidence of previous inflammatory changes, has been observed in quite a number of cases. In explanation of this it may be suggested that the slow increase of intra-ventricular tension, which results from the gradual compression and closure of the aqueduct of Sylvius, causes distension of the thin-walled third ventricle and pressure upon the optic tracts. Cases in which optic

atrophy is observed are usually of long standing, and in those in whom the symptoms began in early childhood.

Vertigo of an indefinite character is almost always present at some stage of the disease.

Cranial nerves. The first, third, and fourth nerves are not affected. The fifth to the twelfth are all liable to be paralysed, completely or incompletely. The paralysis is often bilateral, and may be of a supra- or infra-nuclear type; when nuclear, the paralyses are grouped according to their anatomical relations within the tegmentum pontis. For this reason bilateral paralysis of conjugate movement is a common symptom, owing to the juxtaposition of the sixth nerve nuclei. There may also be found paralysis of different nerves on opposite sides—such as the seventh on one side, and the twelfth on the opposite side. The pupils may show ectopia, or an eccentric relation to the iris.

Motor system. Bilateral paresis, often more marked on one side, and associated with spasticity and general ataxia, is usually present. Attacks of shuddering, as if from cold, are frequent and more or less characteristic of tumours of this region.

Sensory system. Hemianæsthesia is sometimes observed, but defects of sensibility may be absent during the whole course of the disease.

Reflexes. The deep reflexes are increased, often unequally; the superficial are diminished or absent, and the plantars are extensor in type.

Sphincter weakness may be delayed until a late stage.

The terminal stages. In children progressive hydrocephalus with loss of vision, defective hearing, and spastic paraplegia ensue; cranial nerve palsies are relatively slight, and the mental state shows little impairment until near the end. Attacks of respiratory failure, characterised by slow, periodic, and cyclic respiration, become increasingly frequent, and cause death in the absence of an intercurrent complication.

In cases commencing after puberty, although the general course of the malady is similar to that just described, the clinical picture resembles that of a somewhat rapid bulbar paralysis with difficulty in swallowing. Artificial feeding is usually necessary; these cases die of malnutrition and respiratory failure.

Tubercle. The pons may be the seat of a tuberculous growth which may be solitary, or one of several scattered throughout the brain. The symptoms at the onset tend to be more acute than in the gliomatous cases. Headache, vomiting, occasionally optic neuritis and febrile disturbances —associated with local signs more or less limited—are also points of distinction. Such general symptoms may subside, leaving merely the evidence of a local lesion. Such may be seen in paralysis of the seventh nerve, or of paralysis of lateral conjugative movement on one side with a crossed hemiplegia, with or without sensory loss; or the sensory division of the fifth nerve may be paralysed on one side with a crossed hemianæsthesia, with or without palsy of the seventh nerve.

This quiescent period may last for a few weeks to as many months, only to terminate in a gradual or sudden onset of symptoms of tuberculous meningitis.

TUMOURS OF THE BASE OF THE SKULL

These are relatively rare, and are usually sarcomatous in character. They are most frequently found in the middle and posterior fossæ, and are of slow growth.

The symptoms presented by these cases are referred chiefly to the cranial nerves; and local symptoms almost invariably precede by longer or shorter periods the onset of general symptoms.

The symptoms are best described with reference to the position of the new growth in the three fossæ.

Tumours of the anterior fossa. These are rare, and may be either sarcomatous or carcinomatous. They may extend through from the orbit, in which case they are recognised by the local orbital symptoms and unilateral anosmia and optic atrophy.

Tumours of the middle fossa. These are chiefly characterised by symptoms referred to the distribution of the fifth nerve. At the commencement complaint may be made of pain over one or all of its branches, and in the absence of objective sensory loss, the condition may be ascribed to trigeminal neuralgia. Sooner or later, sensation becomes impaired over the face, the corneal reflex is diminished or

lost, and trophic ulcers form upon the cornea. The motor affection of the fifth nerve may not develop until later. Not infrequently the extension of the growth causes paralysis of the third and sixth nerves; and by pressure upon the optic tract produces a homonymous hemianopsia to the opposite side. The general symptoms may remain in abeyance, although in some cases optic neuritis and vomiting become established.

It is important in all such cases to examine the hard palate, as the growth may extend into the mouth as well as into the cranial cavity. Not infrequently extension into the posterior fossa may also occur, or metastatic deposits develop on the opposite side, or indeed anywhere on the floor of the skull.

Subjective sensations of smell and taste arise from secondary involvement of the uncinate gyrus, and a slight degree of hemiplegia may be observed.

Tumours of the posterior fossa. The symptoms of growths in this locality are referred to the sixth, seventh, and eighth nerves. As the eighth is the nerve which offers the least resistance, it is first affected—noises in the ear, giddiness, and deafness appearing early. Extension of the tumour towards the foramen magnum induces paralysis of the ninth, tenth, eleventh, and twelfth nerves; but a forward extension is not uncommon, so that the fifth nerve shares in the paralysis.

The symptoms often tend to become bilateral owing to metastatic deposits on the opposite side.

In addition to the local symptoms of cranial nerve paralysis, a slow and progressive spastic weakness of the opposite side results from pressure upon the crus, pons, or bulb, and cerebellar symptoms on the same side may also develop indirectly from pressure.

The differential diagnosis of this condition from primary tumour of the eighth nerve (p. 267) is made by the more rapid and more extensive involvement of the other cranial nerves, by the occurrence and greater persistence of pain in the head and in the distribution of the fifth cranial nerve, by the less complete deafness, by the later onset of cerebellar symptoms, and by the paralysis of the external rectus muscle preceding the development of nystagmus.

The presence of a tumour in the roof of the mouth, or a history of pre-existent malignant disease, confirms the diagnosis. Occipital pain, especially on movement of the head, is a symptom of some significance in these cases.

General Diagnosis of Intracranial Tumours

The diagnosis of an intracranial tumour is based upon the study of the symptoms and signs presented by each case—phenomena which necessarily vary according to the nature and situation of the growth—as detailed in the account of the regional diagnosis just described.

Certain general symptoms, however, are common to intracranial tumours and other disorders. These are more particularly headache, vomiting, optic neuritis, and convulsive seizures.

Persistent headache, especially if associated with vomiting and optic neuritis, points strongly in favour of a tumour within the skull, but may also be present in meningitis, abscess of the brain, anæmia, and chronic renal disease.

The existence of these symptoms without any signs of *Nephritis* or of *Anæmia* is presumptive evidence of an intracranial new growth, which will receive confirmation by the presence of other signs—motor, sensory, and reflex—of organic disease of the brain. On the other hand, the absence of such organic signs, but the presence of obvious grave anæmia, or of albuminuria, cardiac hypertrophy, and arterio-sclerosis, are usually sufficient to explain the symptoms. Cases, however, are from time to time seen in which signs of organic cerebral disease are found in association with chronic renal disease or anæmia. These present great difficulty in diagnosis; but the history and the mode of onset of the symptoms, or the character and persistence of the physical signs, or the effect of treatment, will usually permit a decision being made.

Cases commencing with generalised convulsive seizures, or with attacks of minor epilepsy, are difficult to distinguish from *Idiopathic Epilepsy*. A definite local aura as the mode of onset of the fit, and the presence of a transient post-convulsive hemi- or monoplegia, are signs favouring an organic cause of the seizures; but it is not until the development of

a progressive paralysis, or of optic neuritis, that the diagnosis of tumour can be placed upon a firm basis.

Symptoms of a similar nature may usher in *General Paralysis of the Insane*; but in this disease the pupillary changes, characteristic tremor, and mental condition usually indicate the true nature of the malady.

The symptoms of tumours of the cerebellum sometimes present a striking resemblance to those of *Disseminated Sclerosis*; but in the latter condition, their relapsing character, the presence of marked subjective paræsthesia, early sphincter trouble, the alteration in the reflexes characteristic of a pyramidal affection, and the absence of the general symptoms of cerebral tumour, usually suffice to distinguish them. (See Table, p. 428.)

The early symptoms of brain tumour, when occurring in young women, may be ascribed to *Hysteria*. It is therefore most important to look for the existence of 'stigmata' of this disease—such as hemianæsthesia, and contraction of the visual fields without organic changes in the fundus oculi. Optic neuritis is never present, nystagmus does not occur, and the reflexes do not show the alterations found in organic disease. (See Table, p. 428.)

Prognosis

A few facts may be given upon the prognosis of life in intracranial tumours. It is obvious that in the majority of cases little more than palliation can be rendered by surgical means. Some forms of tumour, either from the slowness of their growth or from their situation, are more amenable to surgical treatment; in these the outlook is more favourable.

1. *Cerebral tumours.* In a general way the outlook, as regards the life of a patient suffering from cerebral tumour, is not so unfavourable as in cerebellar tumour. Gummata, endotheliomata, and fibro-sarcomata may be removed with a fair prospect of relief and prolongation of life. Rapidly growing gliomata and sarcomata are most unsatisfactory, although in favourable cases the duration of life after operation may be from six months to two years. In less malignant forms life may be prolonged for six, eight, or more years after operation without recurrence of the growth. Tuberculous

tumours are unfavourable. The prognosis as regards life is bad in gliomata, sarcomata, and carcinomata, if no operation is performed.

The prognosis of meningeal and cortical new growths is decidedly more favourable than in those which are subcortical or deeply placed.

2. *Cerebellar tumours.* In intra-cerebellar growths, not submitted to operation, the prognosis is bad; but as cystic degeneration of the growth sometimes occurs, life may be indefinitely prolonged, provided always that the patient survives the stage of acute symptoms. Operations for cerebellar tumours, although attended with considerable risk, are not only justifiable, but imperative as a palliative measure, and on the whole the results are good.

Extra-cerebellar tumours are usually of slow growth, and even without operation life may be prolonged for a number of years. The palliative operation may indefinitely prolong life, but the radical operation usually results in death.

3. In *pontine tumours* the prognosis depends upon the site and the rate of the growth. If the bulb is involved or compressed, death occurs early. Intra-ventricular growths are unfavourable.

The possibility of sudden death in some cases of intracranial tumour should be borne in mind in estimating the prognosis.

Treatment

The advances in cerebral localisation have made the treatment of intracranial tumours almost entirely a surgical matter. As soon as the presence of an intracranial tumour has been diagnosed, and its position, if possible, localised, no delay should occur in explaining the nature of the malady to the patient and laying before him the proposed method of treatment—its dangers and its advantages.

A preliminary course of medicinal treatment is unnecessary, except in cases in which the tumour is probably of gummatous nature, when a month or six weeks' course of energetic anti-syphilitic treatment should be carried out. If at the end of this period the symptoms persist, or are increasing, no further postponement of surgical interference is advisable.

Surgical measures are undertaken with a twofold object: one, *palliative*, for the relief of the general symptoms—more especially headache, vomiting, and optic neuritis; the other, *radical*, with a view to the removal of the growth.

The *palliative* operations are undertaken with the object of relieving the excessive intracranial tension present in most cases of tumour. It is not necessary that the exact position of the tumour should be known before trephining: all that is required being a general indication, from the extent and degree of the symptoms, as to whether the growth is above or below the tentorium cerebelli.

If the tumour is supra-tentorial in position, the trephine opening should be made either in front of, or behind, the Rolandic area. If the growth is believed to lie behind the motor area and to be deep in the subcortical tissues, the opening in the skull should be made over the occipital region, preferably on the side of the tumour. If there is no clear evidence that the tumour is behind the motor area, the trephine opening should be made over the frontal lobe and as near the middle line as possible.

In all cases a large portion of bone should be removed and the dura mater freely incised by the flap method, so as to insure relief of pressure, freedom from strangulation of a portion of brain through the opening in the dura or skull, and avoidance of subsequent paralysis.

When the skull has been opened for the relief of pressure, the tendency is for the compressed brain around the tumour to become the seat of a transient œdema, and to protrude at the artificial opening in the skull. It is for this reason that the operation should be carried out in front of, or behind, the motor area, according as the tumour is supposed to lie anterior or posterior to the Rolandic fissure. In this way the transmission of the pressure across the motor zone with consequent hemiplegia and sometimes aphasia is avoided.

If the growth is subtentorial and not localisable to one or other side, a large opening should be made on either side of the middle line over the cerebellum; but the theca should not be opened in the first stage, as the removal of the bone alone affords sufficient temporary relief, and enables the parts to gradually accommodate themselves to the altered

pressure conditions. At a later stage the dura mater may be opened on both sides, or only on one side, if the tension shows great unilateral excess. By this method the serious risk of collapse from sudden relief of pressure is obviated. Moreover, it affords an opportunity of radical interference at the second stage.

The effect of such palliative operations is sometimes remarkable. Headache and vomiting are relieved, optic neuritis and epileptic seizures gradually subside, and stupor or coma gives place to a brighter and clearer mental condition.

The outstanding complication of palliative trephining lies in the tendency towards hernia cerebri.

Palliative operation only is recommended in cases of sarcoma of the base of the skull, and in tumours of the auditory nerve. Although the latter are in many respects most suitable for extirpation, the risks of their removal have in the majority of cases proved far too serious, and the permanent condition of the patient is not materially improved.

Cerebellar tumours are frequently cystic, and tapping, without removal, may be resorted to with success.

Radical operations, with a view to the removal of the new growth, ought to be undertaken only when the tumour is clearly and definitely localised. For this purpose the trephine opening is made over that part of the brain to which the localising symptoms point as the position of the tumour.

The importance of a large opening is great, as the surgeon is enabled to see and define the limits of the tumour and the point of maximum pressure.

If the tumour grows from the dura mater, it may be removed at once along with the membrane to which it is attached. If it is within the brain, the parts should be carefully inspected and its limits defined. If it is obvious that the growth is extensive and of an infiltrating nature, no attempt should be made to remove it. If no tumour is visible, the brain may be carefully incised and the subcortical tissue examined. If a growth of small size is found, it may be removed provided that its removal will not increase paralysis or lead to aphasia.

Chapter VI

INTRACRANIAL ABSCESS

Suppuration within the cranial cavity may implicate both membranes and the brain. When it affects the membranes it may be either generalised, as in suppurative meningitis, or localised, as in extra- and intradural abscess. Suppuration may also affect the brain and membranes suppurative meningo-encephalitis.

Etiology. Intracranial suppuration is the result of local and general infection by staphylococci, streptococci, and pneumococci.

The local infections are due to the following causes:—

1. *Traumata of the scalp, skull, membranes, and brain*, in consequence of compound fractures, gunshot, or other injuries. Intracranial abscess may also arise as the result of *contrecoup* a blow on one side of the skull, causing injury to the brain on the opposite side which may become the seat of abscess. An injury to the scalp alone may give rise to abscess. Such injuries may be followed by suppuration within thirty-six hours; or after four or five weeks' interval. On the other hand, all symptoms may be deferred for a number of years—up to twenty years, in one of our cases. An abscess may be situated superficially or deeply. When superficial, symptoms arise early; when deep the occurrence of symptoms may be delayed.

2. *Suppurative otitis media.* Intracranial abscess may be secondary to both acute and chronic suppuration in the middle ear. The routes by which infection may be carried are:—

(*a*) Through the roof of the middle ear and antrum. When carried by this route suppuration is found in the middle fossa of the skull, involving the temporo-sphenoidal lobe and its meningeal coverings.

(*b*) Through the posterior wall of the antrum, the labyrinth and the internal auditory meatus. Lateral sinus thrombosis and cerebellar abscess are commonly brought about by infection along this route.

(c) Through the venous and lymphatic channels between the ear and the cranial cavity, and by the eighth nerve.

The most frequent intracranial complications of acute otitis media are suppurative meningitis and sinus thrombosis; those of chronic otitis are temporo-sphenoidal and cerebellar abscess, but both meningitis and sinus thrombosis may also occur.

3. *Disease of the nose and accessory cavities.* The intracranial complications due to these causes may result from operative interference upon the nose or from acute and chronic suppuration of the sinuses. Infection may occur directly from carious bone or indirectly through the orbit, optic nerve, and ophthalmic veins, or by the venous and lymphatic channels. Disease of the ethmoidal and sphenoidal sinuses is most prone to give rise to intracranial suppuration.

It may be stated in general terms that acute infective disorders of the ear, nose, and accessory sinuses, operative procedures and injury are liable to give rise to acute suppurative meningitis, whereas chronic infections are more prone to induce abscess of the brain.

4. *Other local infective causes* of cerebral abscess are—tuberculous and syphilitic affections of the cranial bones, acute osteomyelitis of the skull, carbuncle, and erysipelas of the scalp.

5. The *general infections* which give rise to intracranial suppuration are: pyæmia, bronchiectasis, empyæma, gangrene of the lung, peritonitis and pericarditis, abscess of the liver, appendicitis, suppuration of joints and of the tonsils, acute fevers—such as cerebro-spinal meningitis, enteric, scarlet fever, measles, and influenza—streptothrix, and actinomycosis.

A so-called *idiopathic* abscess of the brain has been described. The causes of this are frequently to be found in an old and forgotten scalp wound, a healed ear suppuration, a nasal focus of disease, previous cerebro-spinal meningitis, erysipelas, and influenza. The majority of idiopathic abscesses are in reality due to old suppurative ear disease which may have escaped detection.

As the causes which give rise to intracranial suppuration are numerous, the initial symptoms are varied and complex,

being in part those of the causal condition, and in part those of the intracranial complication.

Pathology. Brain abscess may be either acute or chronic. In the acute form pus is found occupying a cavity which may or may not have any definite separation from the surrounding brain substance, which is soft and pultaceous. The abscess is usually in the white matter, and is separated from the surface of the brain by what appears to be healthy tissue. In this type of abscess infection has taken place through the vascular and lymphatic channels.

In the more common, or chronic abscess, adhesions have formed between the affected bone, the dura, the pia-arachnoid membranes and the cerebral cortex. Owing to the lesser resistance offered by the white matter, pus makes its way more readily in the subcortical tissues; so that abscesses of this character are of mushroom shape, the stalk being attached to the dura mater at the seat of infection. They are usually encapsulated by a wall of dense tissue. They may rupture either into the subarachnoid space or into the ventricles.

Heiman[1] records the relative frequency of abscess in different localities as follows: Out of 818 cases of intracranial suppuration, 539 were cerebral and 279 were cerebellar abscess. In the cerebral cases the temporal lobe was much the most commonly affected, being involved seven times more often than the rest of the cerebrum.

When due to direct extension from ear disease, abscess of the temporal lobe is situated in the third temporo-sphenoidal gyrus, or between the base of the lobe and the convexity. The abscess rarely occurs in the anterior part of the lobe. It may be superficial with a local meningitis, or healthy brain may be present over the abscess, or the abscess may be deeply placed in the lobe.

The majority of cerebellar abscesses are found near the groove containing the sigmoid sinus. They are laterally placed when infection is through the sinus, and mesially when infection is through the labyrinth.

Pontine and crural abscesses are rare.

The two sides of the brain are equally affected.

[1] Heiman, *Zeitchr. f. Ohrenheilk*, vol. xxxii.

Traumatic and otitic abscesses are usually single, and metastatic abscesses are multiple.

Symptoms. The symptoms of intracranial abscess are both general and local: the latter varying according to the part of the brain directly implicated, or to pressure upon nerves or structures situated at some distance from the seat of the disease.

General symptoms. The general symptoms are, in the main, similar to those produced by tumour, or other cause of increased intracranial pressure. Headache is usually an early and sometimes a prominent symptom. It is, however, by no means a constant feature, nor does it invariably correspond to the site of the abscess. There is no necessary relation between the seat of the abscess and localised tenderness on cranial percussion.

Mental symptoms are frequently early as well as constant features in most cases. The most marked is a tendency to somnolence or lethargy, with excessive lassitude, loss of the power of attention, and inability to answer questions or to carry on conversation. Concurrently with the progress of the disease, the lethargy deepens, and the stupor becomes more pronounced until it terminates in unconsciousness and, finally, coma.

The temperature varies from a normal, or slightly subnormal, curve to one showing moderate pyrexia. A sudden rise to 104° or 105° F. would indicate rupture of the abscess into the ventricle. In cases complicated with sinus thrombosis, the chart is of the pyæmic type with temperature rising daily to 105° or 106° F. and dropping to a normal or subnormal level with rigors and profuse perspiration.

The pulse is usually slow and regular, and even when the temperature is raised, a corresponding increase in the pulse frequency is not observed.

Optic neuritis is sometimes present, but when it occurs it is a late symptom. Many cases of cerebral abscess pass through their whole course without the appearance of this sign.

Vomiting is an early, but not a constant symptom. In cerebellar abscess its persistence is more marked than in abscess in other localities.

Rapid and pronounced emaciation and cachexia are not

uncommon symptoms of cerebral abscess, and when present are very characteristic.

In all cases of chronic purulent otorrhœa, the onset of headache, with irritability or any other sign suggesting an alteration in the patient's normal mental condition, an attack of causeless vomiting, or a slight degree of pyrexia, should arouse suspicions as to the likelihood of intracranial suppuration. A sudden acute increase in the discharge from the ear, or a sudden arrest of a chronic otorrhœa ought, if accompanied by symptoms such as have been described, to be looked upon as signals of a dangerous kind.

Although three stages are usually described in the development of the symptoms of intracranial abscess, it is impossible to define their limits, as one is merely an intensification of that which precedes it, and in turn merges into that which follows. The first stage, however, may be described as that of the *prodromal* phenomena irritability, change of temperament sometimes attributed to hysteria, slight pyrexia, headache, and one or more attacks of causeless vomiting; the second stage as that of the *full development* of the abscess, with a slow pulse—sometimes pyrexia, at other times a subnormal temperature—headache, and a tendency to lethargy and somnolence. This merges into the *terminal* stage of stupor and coma.

Cerebro-spinal fluid. This may present a milky appearance when withdrawn by lumbar puncture. A cytological examination may reveal the presence of polymorpho-nuclear cells with or without the presence of micro-organisms. The latter may also be present without any leucocytes.

Localising symptoms. The temporo-sphenoidal lobe and the lateral lobe of the cerebellum are the two commonest situations of cerebral abscess.

An alteration in the percussion note of the skull has been stated to be a sign of cerebral abscess. Over the abscess the percussion note becomes clear and high-pitched when the head is struck with a percussion hammer. This is best detected by aid of a stethoscope placed upon the forehead, or on a bald patch.

Temporo-sphenoidal abscess. The general symptoms already described are present, on whichever side the abscess may

be situated; but should the suppuration be present in the left lobe in right-handed persons, a form of partial aphasia has been found as one of its earliest signs. This consists of an inability to name objects, although a knowledge of their function is still retained and may be expressed.

An early sign of especial significance is loss or impairment of the epigastric and abdominal reflexes on the side opposite the abscess; this may be found even without any sign of motor or sensory paralysis.

Cerebellar abscess. Symptoms pointing to involvement of the lateral lobe of the cerebellum are: retraction of the head and rigidity of the neck, giddiness often so pronounced that the head is with difficulty raised from the pillow, and a reeling or staggering gait. Some patients assume in bed a characteristic attitude, lying on the side with the legs flexed, and that aspect of the face upwards, which corresponds to the side of the abscess. Slow and large range nystagmus to the side of the abscess, and inco-ordination of the movements of the limbs upon the same side, are the chief localising signs.

The localising symptoms of abscess of other parts of the brain do not differ from those described under Intracranial Tumours.

Prognosis. Acute abscess is a serious malady, which unless treated surgically will cause the death of the patient within a few weeks. If the abscess is uncomplicated and operation for its evacuation undertaken early, the prognosis is, on the whole, favourable. This differs to some extent upon the seat of the abscess: temporo-sphenoidal abscess presenting a more favourable outlook than that situated in the cerebellum.

The prognosis in chronic abscess varies. If of small size, its presence may be undetected, providing it does not give rise to localising symptoms. When it gives rise to general symptoms, the prognosis is grave, and the condition calls for immediate surgical interference.

Diagnosis. The differential diagnosis of abscess of the brain requires to be made from suppurative meningitis and infective sinus thrombosis. The points in the diagnosis are given in the Table upon p. 288.

Treatment. The prophylatic treatment consists, in traumatic cases, in rendering aseptic all damaged tissues. Where the bone is injured and dirty, the injured portion should be removed. If the dura mater or brain be injured, the former should be freely exposed and opened to permit the inspection of the lepto-meninges and brain; and, where necessary, the infected portions should be removed. When the brain has been injured it is advisable not to close the wound, but to drain for a time.

Any local suppurative disease of the accessory cranial sinuses should be attended to, and operation for its removal effected without delay.

All chronic otorrhœas require careful attention and treatment. Macewen[1] gives the following indications for opening the mastoid antrum:—(*a*) Repeated inflammations in the mastoid antrum and cells with swelling over the mastoid process or a fistulous opening in the bone. (*b*) An acute inflammation of the mastoid antrum and cells with retention of pus. (*c*) When there are initial symptoms of intracranial involvement associated with chronic purulent otorrhœa. (*d*) Persistent chronic otorrhœa, arising from carious disease of the temporal bone, which has resisted ordinary treatment. (*e*) If the discharge is highly offensive or contains virulent pathogenic organisms.

Extradural and Intradural Abscesses

An abscess may form between the infected bone and the dura mater. This is rare, except in cases of ear disease. This form of abscess may not give rise to any specific signs—being found by the surgeon during the course of an operation for mastoid disease. It is not infrequently in direct communication with the tympanic cavity, and may give signs of its presence by the welling out of pus from the bone after this cavity has been cleansed.

An intradural abscess may also be present, the pus being prevented from infecting the subarachnoid space by the formation of adhesions during the slow process of meningeal infection. The symptoms of this type of abscess are similar to those of abscess within the brain.

[1] Macewen, *Pyogenic Diseases*, 1893.

INFECTIVE SINUS THROMBOSIS.

This is an infective or inflammatory condition occurring mainly in adult persons. It is of local origin, and affects the sinus nearest the site of the primary disease. In contrast to the marasmic form it is found mainly in the bilateral sinuses. In the later stages it is associated with meningitis and pyæmic abscesses of the brain.

Etiology. The common cause is a chronic suppurative inflammation of the middle ear, with a cario-necrotic affection of the surrounding bone, infecting the sigmoid sinus. It may also arise from trauma of the skull, and from infective diseases in the orbit, nose, and tonsils; from erysipelas of the face and scalp, dental caries with periostitis and retropharyngeal abscess.

It may be due either to direct extension, or to a thrombus in and extending from a small neighbouring vein. The result of this infection is the gradual formation of a thrombus within usually the sigmoid vein. This in time becomes infected, and gives rise to a general infection of the system through suppuration in and disorganisation of the clot.

The local infection may extend downwards into the jugular vein, or upwards into the cavernous sinus. A general systemic infection may also occur directly from the ear without the intervention of a localised sinus phlebitis.

Symptoms. The symptoms are both general and local: the former being those of pyæmia, and the latter depending upon the particular sinus affected.

The *general* symptoms are, vomiting and headache—the latter usually severe, and either widespread or referred to the seat of the disease.

The temperature is remittent, rising and falling rapidly, associated with a small rapid and irregular pulse, profuse perspirations and rigors. The tongue is dry and coated; anorexia is common, and diarrhœa more frequent than constipation. Optic neuritis may also be present. Macewen has described two types of pyæmic infection: (1) the pulmonary—whose symptoms are cough, pain in the chest,

dark-coloured sputum, and fetor of breath; and (2) the abdominal—characterised by a dry cracked tongue, vomiting, tympanites, diarrhœa, or the typhoid state.

The *local* symptoms point to thrombosis of particular sinuses.

(*a*) *The superior longitudinal sinus.* Thrombosis of this sinus is usually a marasmic condition characterised by œdema of the scalp and distension of the veins, epistaxis, convulsions, and more rarely paralysis.

(*b*) *The cavernous sinus.* Both sinuses are usually involved as a result of infective disorders of the orbit, face, mouth, nose, throat, and pharynx. The symptoms consist of pain over the supra- and infra-orbital branches of the fifth nerve, protrusion of the eyeballs, œdema of the eyelids and side of the nose, ptosis and ocular palsies. Optic neuritis may also be present.

(*c*) *The sigmoid sinus.* The symptoms may be of only slight intensity. When present they consist of œdema and pain over the mastoid bone, phlebitis and thrombosis in the internal jugular vein and deep veins of the neck, pain on pressure over the upper part of the jugular vein, which may be palpable, and sometimes enlargement of and suppuration in the cervical glands.

The **differential diagnosis** has to be made mainly from abscess of the brain and suppurative meningitis. The leading points of difference are given in the Table on p. 288.

Prognosis. This is essentially unfavourable. If surgical interference is carried out in the early stages, the outlook is more hopeful. The duration of an untreated case is only a few weeks.

Treatment. Treatment is entirely surgical, and limited to the affections of the sigmoid sinus. It consists in exposing, clearing out, and ligaturing the affected sinus or vein, as well as removing the primary source of infection. Owing to the occasional coexistence of abscess, attention should be given to the cerebellum and the temporo-sphenoidal lobe.

DIFFERENTIAL DIAGNOSIS OF SUPPURATIVE MENINGITIS, CEREBRAL ABSCESS, AND INFECTIVE SINUS THROMBOSIS.

	MENINGITIS.	ABSCESS.	INFECTIVE THROMBOSIS.
Onset	Rapid	Relatively slower	Rapid
Temperature	Pyrexia, 100° Fahr. to 104° Fahr.	Normal, subnormal, or slight pyrexia	Pyæmic type with sudden rises and falls accompanied by rigors, &c.
Pulse	Rapid, irregular, and intermittent	Slow and regular	Rapid and irregular
Focal signs	Twitchings of limbs, strabismus, rigidity of neck, Kernig's sign present	Signs of lesion of temporo-sphenoidal lobes, or of cerebellum	Mastoid tenderness and swelling; tenderness along jugular vein; exophthalmus on one side; œdema of scalp
Blood	Leucocytes from 17,000 to 21,000 per c.mm.	Leucocytes may not exceed 14,000	Leucocytes about 20,000
Cerebro-spinal fluid	Purulent or semi-purulent under increased pressure, large polymorpho-nuclear counts. Strepto-, staphylo- and pneumococci present	May be milky. Polymorpho-nuclear cells may be present. Micro-organisms may be present without leucocytes	Same as abscess

CHAPTER VII

HYDROCEPHALUS

This is a condition characterised by distension of the ventricles of the brain with cerebro-spinal fluid. It arises in three ways:—

1. As a congenital or developmental condition, either alone, or in association with defects—such as spina bifida, hydromyelia, or other malformations.

2. As a primary condition, coming on at any period after birth.

3. As a secondary condition, resulting from obstruction and damming up of the cerebro-spinal fluid within the ventricles.

Etiology. The causes of primary hydrocephalus are not clearly understood.

It would appear to be due to an inflammatory affection of the lining membrane of the ventricles, more especially that covering the choroid plexus. In two cases—the one a child, the other an adult—the ventricular ependyma was studded with small, hard, whitish nodules composed of proliferated ependymal cells, with evidence of old inflammatory lesions in the choroid plexus. The presence of thickening of the intima of the choroidal vessels suggested a syphilitic origin.

The secondary forms arise in connexion with tumours of the brain, especially when subtentorial and affecting the ventricular systems. Subsequent inflammatory changes cause blocking of the veins of Galen, with resulting hyperæmia of the choroid plexuses. Most frequently it arises from meningitis obstructing the communication between the ventricular system and the spinal subarachnoid spaces, especially in posterior basic and epidemic cerebro-spinal meningitis. It is rare in tuberculous meningitis.

Symptoms. (1) *Congenital hydrocephalus.* The most striking feature is enlargement of the head. This may be present at birth, or be observed to take place a few weeks after birth. The enlargement is limited to the cranial vault, the bones of which become separated sometimes to a surprising extent. Owing to this the forehead is abnormally prominent, and the parietal region directed backwards. In comparison to the large head the face appears small or shrunken, and, owing to the displacement of the orbital plates of the frontal bones, the eyes are turned downwards. The veins of the scalp may be enormously distended. The sutures of the skull are widely open.

In association with the cranial enlargement a number of nervous symptoms may be present. These consist of convulsions, attributable to the abnormal pressure exerted upon the cerebral cortex against the calvarium by the enlarged and distended ventricles; paralysis and spasticity of the limbs, with muscular contractures, exaggeration of the deep reflexes, and extensor plantar responses are common. The mental symptoms are those of marked mental enfeeblement or amentia.

Vision is frequently impaired from pressure of a distended third ventricle upon the optic chiasma, or from optic atrophy.

Nystagmus and convergent strabismus are accompanying symptoms.

As this condition is usually congenital or arises in early life, its recognition is easy.

2. *Primary hydrocephalus.* When it occurs in adults it is difficult to recognise. It is liable to be confused with cerebral tumour or intracranial aneurism. It would appear to be one of the conditions giving rise to 'pseudo-tumour.' The symptoms are those of intracranial tumour in so far as headache, vomiting, optic neuritis, and giddiness are concerned. These are often attended by convulsions, either general or focal. The important symptoms in favour of primary internal hydrocephalus are the early impairment of upward movement of the eyes, and of the light reflex, at a stage when the optic neuritis is acute and the vision unimpaired, especially as regards the perception of light. Early failure of vision often occurs when the ophthalmoscopic appearances are characteristic of acute papillitis and without visible evidence of atrophy.

In the convulsions with local onset the commencement is not always in the same locality: one seizure beginning in the arm and another in the leg. This variability in the local seizures is important in distinguishing the condition from a localised tumour.

Besides the convulsive phenomena the motor system exhibits a progressive spasticity, which is bilateral and equal on the two sides, and associated with slight general weakness. The reflexes are characteristic of upper motor neurone lesion.

3. *Secondary hydrocephalus.* The symptoms and signs of the secondary or obstructive form are grafted upon those of the pre-existing cerebral lesion.

In cases of acute meningitis enlargement of the head may be noticed; but in the early stages, at all events, it is quite impossible to differentiate the symptoms of the two conditions.

Where hydrocephalus is secondary to slowly growing intra-pontine tumours or chronic meningitis, in addition to enlargement of the head, which depends upon the amount of ossification of the cranial bones, a change in the percussion note of the skull may be observed, often associated with the development of an intracranial murmur.

The nervous phenomena are headache, convulsions usually generalised but sometimes local, spasticity of varying degrees associated with increase of the deep reflexes, diminution of the superficial abdominal reflexes, and the presence of the extensor type of plantar response.

Very characteristic are the ocular phenomena. These are (1) early loss of upward movements of the globes, associated with (2) diminution or loss of the pupillary light reflex—presumably due to the pressure of a distended third ventricle upon the third-nerve nucleus; and (3) failure of vision without or with only slight optic neuritis, going on to optic atrophy from pressure upon the chiasma.

In rapidly growing intracranial tumour with acute pressure symptoms, hydrocephalus is less extreme and optic neuritis more pronounced, and the symptoms generally are those of increased intracranial pressure.

The **diagnosis** is not difficult in infants when enlargement of the head has taken place. The enlarged head of the rachitic child differs from that of the hydrocephalic by its squarer shape, by the absence of prominent fontanelles, and by the other associated signs of rickets.

When hydrocephalus arises as a primary condition in later childhood or early adult life, greater difficulty may be experienced in diagnosis, as the symptoms are more comparable to those of intracranial tumour.

The diagnosis of this condition rests first upon the presence of severe general symptoms without associated localising signs; secondly, upon the characteristic development of the ocular and visual symptoms; and thirdly, upon the onset of bilateral spasticity.

Lumbar puncture is often of value in the diagnosis of primary hydrocephalus—a highly albuminous fluid being withdrawn.

Prognosis. In the majority of cases of developmental hydrocephalus the outlook is bad, and death ensues within a comparatively short time. A few cases of mild and non-progressive character may live for a number of years with impaired mental capacity.

In the acquired cases the prognosis is bad. In some instances, on the other hand, the malady is arrested and life is prolonged with impaired mental faculties and epilepsy.

In the type arising in consequence of cerebral tumour, or of the meningitides, the prognosis is that of the causal condition.

Treatment applies almost solely to the developmental form with marked enlargement of the head. The pressure of bandages or of a plaster applied to the head is an old method, and may be tentatively adopted. Tapping the ventricles and lumbar puncture are of little use—the former being, moreover, a highly dangerous procedure. Ligature of the internal carotid arteries has been of use in some cases.

A method of establishing a communication between the ventricular and subarachnoid spaces has been adopted with some success by Cheyne and Sutherland.

In the primary and secondary forms, trephining with a view to decompression should be performed on both sides of the skull over the inferior and post-parietal regions. Repeated lumbar puncture might also be of some service in relieving intracranial tension.

Part VII

DISEASES OF THE MEMBRANES

EPIDEMIC CEREBRO-SPINAL MENINGITIS (CEREBRO-SPINAL FEVER)

This is an acute infective inflammation of the pia-arachnoid membrane over the brain and spinal cord, due to the presence of the meningococcus, or diplococcus intracellularis meningitidis. It occurs in both an epidemic and a sporadic form, and there is reason to suppose that the condition known as posterior basic meningitis is a mild, or atypical example of the same disease.

Etiology. Numerous epidemics of cerebro-spinal fever have occurred during the past century; but attention has been recently redirected towards it owing to severe epidemic outbreaks in New York in 1905, in Glasgow in 1906, and in Belfast in 1907. Epidemics of cerebro-spinal fever seem to differ from those of scarlet fever, or of smallpox, by the irregularity of their distribution—the disease cropping up in disconnected places. The majority of cases occur in young people under fifteen years of age—the first quinquennium showing the greatest number of cases. Adult life, however, does not confer complete immunity, although it is rare over twenty-five or thirty years of age. The epidemics have appeared chiefly during the winter and spring months, the number of cases sensibly diminishing during the summer to reappear in the late autumn and winter.

An epidemic may be preceded, or succeeded, by some increase in the number of the sporadic cases. In the intervals between epidemics small accessions to the average number of cases usually observed during the winter have been noted, but not amounting to an epidemic. Posterior basic meningitis, mainly met with in infants under twelve months, and by some

regarded as a sporadic form of this disease, has also been found to occur in epidemic form.

The disease is favoured by over-crowding and insanitary conditions, as it rarely shows itself outside the poorer quarters of large cities. It is not communicable as a rule from one person to another, as are the ordinary forms of infectious disease.

The meningococcus. The essential cause of the disease is the diplococcus intracellularis meningitidis. It is difficult to cultivate and of low vitality. It is found in the polymorphonuclear cells of the meningeal exudation. It has also been found in the secretions of the nose, mouth, and conjunctiva, which would appear to be its normal habitation. It is probable, but by no means certain, that it effects an entrance into the subdural space through the lymphatics of the tonsils and the sphenoidal sinuses.

Morbid anatomy. The meninges of the brain and spinal cord present an inflamed appearance and are covered with a purulent exudation. This is especially seen over the base with an extension upwards to the convexity. The cerebro-spinal fluid is increased in quantity, and of turbid appearance. The ventricles may also be distended, and their walls red and œdematous.

Microscopically, the pia-arachnoid membrane is found to be the seat of pronounced inflammatory changes, the blood-vessels being engorged and surrounded by a profound perivascular. cell infiltration. In chronic cases, or in cases observed after the acute stage has passed away, the pia arachnoid shows considerable cicatrical thickening. The nerves, both cranial and spinal—but especially the former —are similarly affected, and may be involved in the cicatrical tissue.

Symptoms. Although a period of incubation is probable, there are no well-determined facts regarding it. In the majority of cases the disease comes on more or less suddenly with headache, restlessness, and vomiting. The other nervous symptoms at this stage vary with the intensity of the disease, but may consist of extreme restlessness, irritability amounting to delirium, convulsions and coma, ending rapidly in death in a few hours in severe instances (fulminant type).

In the less acute cases more obviously meningeal symptoms

are observed—such as twitchings of the face, arms and legs, squinting, and photophobia. Amongst other symptoms detected in well-defined cases are retraction of the head; or if less severe, rigidity of the muscles of the neck on attempts to bend the head, pain at the back of the head and in the neck, or a tendency to opisthotonos. Kernig's sign may be present. This consists of an inability to extend the leg upon the thigh

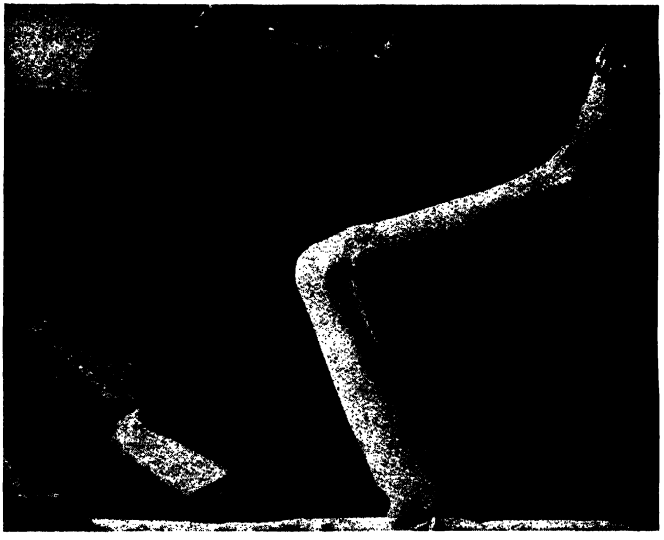


FIG. 86. Photograph of a child presenting Kernig's sign. Note the contraction of the hamstring muscles.

when the child is lying on the back, with the thigh in a semi-flexed position. (Fig. 86.)

There may be intervals of consciousness, during which complaint is made of severe headache—frontal or occipital; and pain along the spinal column is a frequent symptom. In favourable cases the symptoms gradually subside, with the exception of the head retraction, which may persist for a long time.

The knee joints are sometimes swollen and painful, and there is a state of general hyperæsthesia, so that the child cannot be moved or even touched without inducing great pain and discomfort.

There is no *temperature curve* characteristic of the disease. According to Osler it may assume forms: (*a*) differing little from that of an average case of enteric fever; (*b*) resembling

tuberculosis in its remissions and exacerbations; and (*c*) suggestive of malaria.

The *pulse* is rapid and irregular, in which it differs from that of tuberculous meningitis, which is habitually slow.

Paralysis of the *cranial nerves* may be severe, and is sometimes permanent even in the cases which recover. Most common is paralysis of the oculo-motor nerves with diplopia. Loss of vision is sometimes found. Deafness also occurs partly from a complicating otitis media as well as from damage to the auditory nerve. A slight degree of facial paralysis has also been found, but more as an early and transient than as a permanent symptom.

Mental. Mild dementia has been observed to persist after the acute symptoms of delirium and stupor have passed away. This has been attributed to an accumulation of fluid within the ventricles, and may be relieved by lumbar puncture. The symptom tends to become permanent with chronic hydrocephalus, mental impairment, and sometimes epileptic fits.

There is also a chronic type of the malady, lasting for many weeks, with remissions and exacerbations of fever, and corresponding improvement or relapse of the nervous symptoms, delirium, stupor, and coma. In these cases the patient may be reduced to a state of profound marasmus and extreme emaciation.

The rash. In its true form this would appear to be a rare condition. It consists of hemorrhagic spots, appearing in crops, over various parts of the body—such as the thighs, legs, hands, abdomen, and chest. They may also be seen in the eyelids and under the conjunctivæ. They appear early in the disease. The spots vary in size from a pin-point to that of a sixpenny-piece, are of a deep purple colour, and do not disappear upon pressure. Other forms of this skin eruption are seen in a roseola like that of enteric fever, and herpes may occur in various parts of the body.

The blood. There is a well-marked polymorpho-nuclear leucocytosis in all acute cases, sometimes amounting to 50,000 per c.mm. In the chronic cases this is not observed. It is of little value as a means of differentiating this malady from tuberculous meningitis.

The cerebro-spinal fluid. This is usually under considerable pressure, and is opaque, turbid, or purulent. A cytological

examination reveals a large number of polymorpho-nuclear leucocytes, which are much in excess of the lymphocytes. In tuberculous meningitis the mononuclear cells preponderate. Suitable staining will reveal the diplococcus lying within the polymorpho-nuclear cells, but occasionally outside them. It is found as a rule during the persistence of the disease.

Diagnosis. The diagnosis has to be made from the other forms of meningitis. The chief points are given in the Table upon p. 304.

Prognosis. In the acute or fulminant form death occurs in a few days. In the less acute form recovery ensues in a small proportion of the cases, while in the chronic relapsing type, recovery may be expected in a certain number of cases.

The outlook, however, is in all cases of this disease obviously grave. In the event of recovery, permanent paralyses of some of the cranial nerves especially the auditory and the optic—are found, and the mental condition may pass into one of mild dementia.

The duration of the disease varies from a few days in the severe cases to several months in the chronic types.

Treatment. Treatment should be conducted along general lines, with abundant nourishment, and stimulants administered when necessary. Cold applications locally to the head and spine may give comfort, while frequent bathing at a temperature of 100° F., and perhaps cupping over the neck, may relieve distress. Sedatives—such as the bromides and chloral—may be advantageously used, and hypodermic injections of morphia have been recommended by some writers, but would seem harmful in many cases.

Lumbar puncture, with the removal of any large quantity of cerebro-spinal fluid, is not likely to be of much benefit in the early and acute cases, unless there are signs of a rapid rise in the intraventricular pressure, but may be of temporary service in the more prolonged and relapsing cases.

The indication for puncture lies in an increase of the pressure in the subarachnoid and ventricular spaces. As the fluid is expressed from the needle in a considerable stream in many of these cases, marked benefit will be temporarily obtained by this operation. If, on tapping, no fluid comes away, or the stream is so small as to suggest diminution of pressure, lumbar puncture should not be further attempted.

POSTERIOR BASIC MENINGITIS

This disease is probably a sporadic form of cerebro-spinal meningitis. It may occur at any season. It affects both sexes equally. It is most common in the first year of life, but also occurs in later years. It is due to the presence of the diplococcus intracellularis in the subarachnoid space, which is probably infected through the posterior nares.

Pathology. The morbid changes are more strictly confined to the posterior basal portions of the brain than in cerebro-spinal meningitis. The purulent exudation is found chiefly in the interpeduncular space and upon the anterior surface of the pons and crura cerebri. It tends to spread along the base of the temporo-sphenoidal lobes towards the ventricles, so as to cause blocking of the foramen of Majendie, and eventually internal hydrocephalus.

Symptoms. The onset is acute or subacute. The child becomes peevish and irritable. Convulsions and vomiting may ensue, and it is noticed that movements of the head cause pain. In a few hours or days the malady becomes fully developed. The child then lies with its head retracted and sometimes in a state of extreme opisthotonos. Cranial nerve paralysis, especially of the sixth nerve, may be present, and there may be difficulty in swallowing.

This state of irritation gives place to one of stupor and coma. If death does not ensue, a slow process of recovery sets in. The mental condition improves and the rigidity becomes progressively less. Kernig's sign, which is present early in these cases, eventually disappears.

Blindness is observed in some cases on recovery from the acute stage. As a general rule this is a transient symptom, although it may persist for as long as six months. On examination the fundus oculi is normal and the pupillary light reaction is unaffected. In other cases, where blindness is permanent, the pupillary reaction is lost, and the examination may reveal atrophy of the optic disc. When such is not present, obvious evidence of hydrocephalus is found.

The after-condition depends upon the degree of obstruction to the flow of the cerebro-spinal fluid through the foramen of Majendie. If complete obstruction has taken place, secondary hydrocephalus results, which may either

result in death at a late stage, or in mental and physical impairment.

The cerebro-spinal fluid is under pressure and turbid, and contains an excess of polymorpho-nuclear cells. The diplococcus intracellularis is present, especially in the early stages.

The **differential diagnosis** is found in the Table upon p. 304.

The **treatment** consists of repeated lumbar puncture and the withdrawal of cerebro-spinal fluid.

TUBERCULOUS MENINGITIS

This is most common in children during the period of the first dentition. It is also, though less commonly, found in adults. In quite young children, especially under one year, the non-tuberculous form of meningitis, or posterior basic meningitis, is more common.

It is doubtful whether tuberculous meningitis is ever a primary condition, though the source of infection may be difficult to find. In two cases the only evidence of tubercle, after careful examination, was in one instance a small caseous bronchial, and in the other a small caseous mesenteric gland. The commonest source of infection is tuberculous disease of glands; then, in order of frequency, tuberculous disease of the lungs, joints, bones, testicles, and the ear. A solitary tuberculous tumour within the brain may be a source of infection. Operations for tuberculous disease of the spine are particularly liable to set alight the meningeal inflammation.

Morbid anatomy. In the earliest stages the membranes of the base may present a greasy or sticky appearance; but in the later, when meningitis is well developed, small white nodules, varying from the size of a pin-point to a pin-head, are seen studded over the meninges, along the fissure of Sylvius, the fissure between the cerebellum and cerebrum, and along the lines of the great vessels. The exudation is of a serous, sero-fibrinous, or fibrino-purulent character. The tubercles are formed of collections of small round cells in the perivascular sheaths of the smaller arteries. In some cases giant cells and caseation in various stages are present.

The arteries may exhibit more or less acute endarteritis.

The brain itself is infiltrated with small round cells, especially along the vessels, and the cortical cells and fibres show softening and degenerative changes.

In most cases the ventricles are distended and sometimes contain a turbid fluid. The ependyma may be granular, and the choroid plexus is distended and tortuous, and may be covered by an exudation. The sub-ependymal tissues are frequently softened. In many cases the foramen of Majendie is blocked, and this may give rise to ventricular distension. In other cases the veins of Galen are obstructed. Owing to intra-ventricular distension, general flattening of the convolutions of the convexity is observed. The dura mater is not affected.

Symptoms. The mode of onset is usually insidious; more rarely it is acute. When the latter, it is a terminal complication of active tubercle elsewhere. The symptoms vary somewhat according to the age of the patient. In children, when the onset is insidious, there is a well-defined prodromal stage lasting from two up to twelve weeks. This is shown by general malaise, and occasionally slight headache. The child is languid and irritable, and often has a dull frowning expression. Constipation and a foul breath, accompanied by occasional vomiting at night without obvious cause, are common accompaniments.

At this period the temperature may be slightly raised up to 100° F. or 101° F., and the pulse quickened.

The further course of the disease may be for descriptive purposes divided into the stages of irritation and of paralysis.

In the *irritative stage* the symptoms assume an acuter phase, which is due to the development of a definite meningeal lesion. These are headache (often severe), generalised convulsions, and rigidity of the neck without marked retraction of the head. The child may lie upon its side with the limbs drawn up and the abdomen retracted. A general hyper-sensitiveness to touch, to movement of the limbs, and to light, may be present. The temperature and pulse rate fall at this period, and the latter is frequently irregular. As this stage progresses, local paralytic symptoms begin to appear. The pupils, which may have been dilated, become irregular and variable in size; paralysis of one or other ocular muscle may result in squint and diplopia; and irregular nystagmoid movements of the eyeballs are observed. The convulsions tend to become more localised, although they may appear in different parts of the body at different times. A condition of general rigidity—at first slight in degree—develops, and

weakness or paresis of one limb or of one side of the body and aphasia may become distinct. All these symptoms are more or less transient, but improvement is never continuous.

In the later phases of this stage, rigidity becomes more marked, convulsions more generalised, and the pupils are inactive to light. These symptoms are due to the development of internal hydrocephalus, with distension of the ventricles. The mental condition is one of stupor, though irritability is seen when the patient is moved. The irritative merges into the second or *paralytic stage*. Here the pulse is quickened, soft, and less irregular. The stupor deepens, difficulty in swallowing comes on, with more complete and persistent paralysis of the limbs. Independent wandering movements of the eyeballs are also seen. The corneal reflexes are lost, the superficial reflexes impaired, and in older patients an extensor plantar response is obtained. Retention of urine in the early stage or incontinence later, Cheyne-Stokes respiration, increasing coma, and an occasional convulsion terminate the scene, which is sometimes accompanied by a considerable rise of temperature.

Optic neuritis develops during the irritative stage, but is rarely severe in type. Tubercles of the choroid have been observed, and are described by some writers as one of the diagnostic signs of the disease.

Sugar may be present in the urine in the later stages.

In cases with an acute onset, active tuberculous disease is found elsewhere in the body. Here sudden headache, vomiting, convulsions, and delirium or stupor, with rigidity of the neck and cranial nerve palsies, are found.

In adults, the headache is in excess of somnolence. The clinical picture is more like that of an intracranial tumour, although the meningeal symptoms suffice usually to distinguish the two conditions. On the other hand, it is not infrequent for meningitis to be grafted upon the symptoms of an already existent tuberculous tumour.

The *cerebro-spinal fluid* may be turbid when withdrawn, and on examination is found to contain the tubercle bacillus. The mononuclear cells or lymphocytes preponderate over the polymorpho-nuclear cells.

The *blood* shows a leucocytosis, but not to the same extent as in cerebro-spinal meningitis. Calmette's reaction is not of

much assistance in diagnosis, as it may be present in association with tuberculous disease in any part of the body.

Diagnosis. In the early stages of the disease, when the symptoms are vague and indefinite, it is next to impossible to state what the course of the malady may be. When more acute symptoms make their appearance—and especially in adult cases—the diagnosis has usually to be made from enteric fever and apical pneumonia. The almost universal adoption of precise methods of diagnosis in doubtful cases is nowhere of more value than in these conditions. A lymphocytosis of the cerebro-spinal fluid, especially if associated with the presence of tubercle bacilli in the centrifugalised deposit, is conclusive evidence of tuberculous meningitis; while the Widal reaction of the blood would establish the existence of enteric fever. From lobar pneumonia the diagnosis is made by the auscultatory signs present in that disease. (See Table, p. 304.)

Prognosis. The disease is invariably fatal within two or three weeks from the onset of the initial symptoms. The cases which have been reported as cured have probably been sporadic instances of cerebro-spinal meningitis rather than the tuberculous variety.

Treatment. This is conducted on general principles. The patient should be kept in bed in a darkened room, the head shaved, and an ice-bag applied to it. Symptoms ought to be treated as they arise—more especially the constipation, which is a feature of the malady. A lumbar puncture, done at the outset in order to establish the diagnosis by an examination of the cerebro-spinal fluid, may have effected a transitory improvement in the symptoms; but it is unlikely that a repetition of this operation will lead to any permanent benefit.

SUPPURATIVE MENINGITIS

Etiology. This is an infection of the lepto-meninges by streptococci, staphylococci and pneumococci. Pneumococcus meningitis may occur in consequence of acute middle ear disease, and probably also as a sporadic condition. The sources of infection are the same as those already described under Abscess of the Brain, p. 279. They are: traumata of the head, suppurative middle ear disease, disease of and operations upon the nose and accessory cavities, acute fevers

—such as pneumonia, influenza, typhoid fever, scarlet fever, and measles—and sometimes a terminal infection in renal, cardiac, and pulmonary diseases.

Meningitis is also described as a result of infection by the gonococcus and the bacillus coli.

Symptoms. The onset of acute cerebral symptoms such as headache, delirium, and convulsions in association with pyrexia, rapid pulse, and vomiting, occurring in the course of any of the above-mentioned conditions is suggestive of a secondary inflammation of the pia-arachnoid membrane.

The headache is severe and continuous, and is accompanied by delirium passing into stupor. The temperature runs a more or less continuous course, not rising and falling as in pyæmic conditions or sinus thrombosis.

The pulse is rapid at first, but eventually becomes intermittent and finally slow.

Patients in this condition are hyperæsthetic to light, to sounds, and to handling of the limbs and to movement. Muscular twitchings and retraction of the neck are also frequent.

Localising symptoms may be present, according to the position of the initial inflammation.

In meningitis of the base of the brain, oculo-motor palsies, strabismus, twitchings of the facial muscles, and sometimes optic neuritis are observed. In meningitis of the convexity, mono- or hemiplegic weakness may develop, and in the later stages convulsions of a generalised character.

In whatever part of the brain the inflammation commences, the general symptoms as described above progress, and the stupor deepens into coma, in which the patient dies.

The *cerebro-spinal fluid* varies from an opalescent to a yellowish or truly purulent fluid. The pressure of the fluid is raised, and as much as 15 or 20 c.cm. may be readily withdrawn. Albumen is present in considerable quantity. The polymorpho-nuclear cells are much increased. Cultivations reveal the presence of staphylococci, streptococci, and pneumococci.

Diagnosis. The presence of polymorpho-nuclear cells alone does not prove the existence of suppurative meningitis: as they may be present without meningitis—in brain abscess, suppurative labyrinthitis, and sinus phlebitis. Micro-

organisms have also been found alone in the fluid in sinus phlebitis, septicæmia, and brain abscess.

Suppurative meningitis should only be definitely diagnosed when a large number of polymorpho-nuclear cells are present along with the above-mentioned organisms in the cerebro-spinal fluid.

The course of the disease is rapid, and invariably fatal. According to the intensity of the symptoms it varies from a few days to two or three weeks. Its ordinary course may be complicated by the existence of a sinus thrombosis or of abscess of the brain.

Treatment is of little avail once the meninges have become infected. Rest, the application of ice to the head, purgation and sedatives, may be prescribed with temporary relief. The abstraction of a quantity of cerebro-spinal fluid has been followed by temporary improvement.

TABLE GIVING THE POINTS OF DIFFERENTIAL DIAGNOSIS BETWEEN THE SEVERAL FORMS OF MENINGITIS ABOVE DESCRIBED

	CEREBRO-SPINAL FEVER.	POSTBASIC MENINGITIS.	TUBERCULOUS MENINGITIS.	SUPPURATIVE MENINGITIS.
Age at onset	Children and young adults	Infants under one year	Children & adults under 50 years	All ages
Infection	Primary, sporadic, epidemic	Primary, sporadic epidemic	Secondary to tubercle elsewhere	Secondary to fractures, operations, or suppurative disease elsewhere in body
Organism	Diplococcus intracellularis	Diplococcus intracellularis	Tubercle bacillus	Streptococcus, staphylococcus, pneumococcus
Cytology of cerebro-spinal fluid	Polymorpho-nuclears largely in excess of lymphocytes	Polymorpho-nuclears in early, lymphocytes in late stages	Lymphocytes considerably in excess of polymorpho - nuclears	Chiefly polymorpho-nuclear cells
Course of the disease	A few days in acute cases—commonly 2 to 6 weeks; chronic cases may persist for several months	One week up to three or four months	Two to eight weeks	A few days to two or three weeks
Special features	Erythematous, herpetic, and petechial rashes, arthritic swellings, cranial nerve palsies	Marked and persistent head retraction Profound marasmus and emaciation	Irregular pulse and temperature; marked constipation; strabismus	Convulsions, delirium, stupor, and coma
Prognosis	Recovery not uncommon	Usually fatal	Invariably fatal	Invariably fatal

Part VIII

DISEASES OF THE SPINAL CORD

Chapter I

ANATOMY AND PHYSIOLOGY OF THE SPINAL CORD

A transverse section of a spinal cord shows a central mass of grey matter surrounded by white substance. A postero-lateral projection of grey matter known as the posterior horn, and covered by a cap of gelatinous substance, subdivides the white matter into a posterior and an antero-lateral column. The antero-lateral column is further subdivided by the issuing anterior nerve roots into an anterior and a lateral column.

The grey matter of the two halves of the cord is connected by the posterior or grey commissure, in which is situated the central canal. The grey matter contains both nerve cells and nerve fibres. The nerve cells are grouped so that some occupy the entire length of the anterior horn throughout the cord, others the intermedio-lateral horn between the eighth cervical and the second lumbar, and a third group, the column of Clarke, occupies the base of the posterior horn in the lower dorsal and upper lumbar regions. Other cells are scattered throughout the posterior horn and intermediate zone of grey matter. (Fig. 87.)

The white columns of the cord are made up of a number of tracts having different origins and terminations, and conducting upwards or downwards. These tracts have been mainly differentiated by the method of secondary degeneration, and may be readily recognised under low powers of the microscope.

Most of the tracts of the spinal cord have been already

incidentally described under the various systems on an earlier page, but it is necessary to refer to them here in relation to the localisation of function in the cord.

1. The *posterior columns* are formed, for the most part, of fibres arising in the posterior root ganglia, which enter the

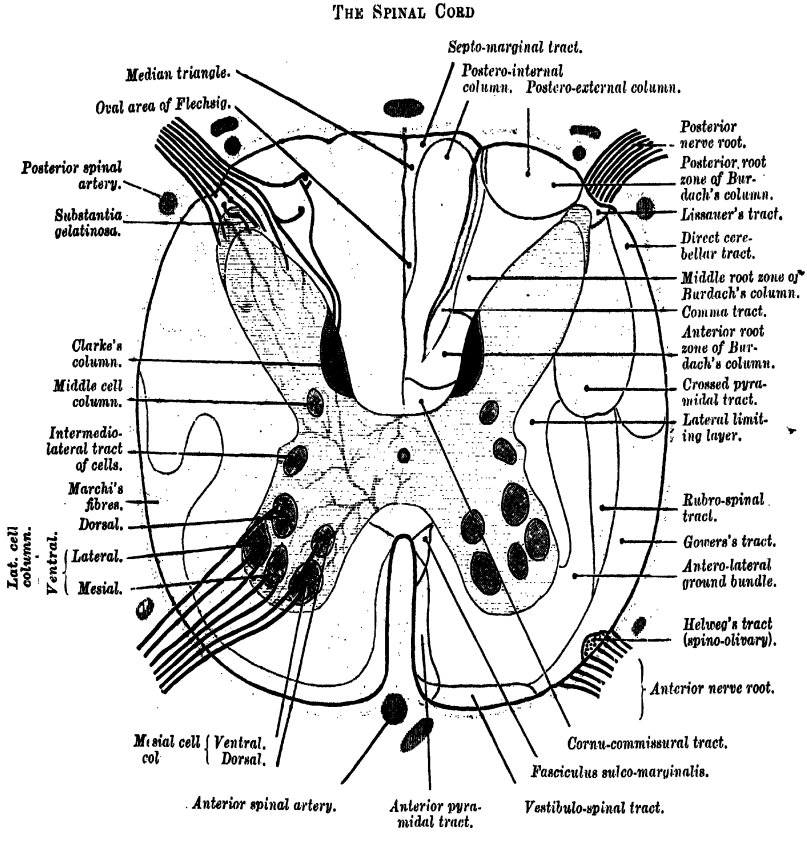


Fig. 87.—Diagram showing the tracts, cell columns, and vascular supply of the spinal cord. (Modified from Morris's 'Anatomy.')

cord at different levels and ascend towards the brain. The degenerations seen in a section of the cervical region after a transverse lesion of the cord vary according to the level of the lesion. If the lesion is low down (lumbar), the degeneration is limited to the postero-internal columns; if high up (cervical), the postero-external columns are also degenerated.

The posterior columns also contain fibres which degenerate below a transverse lesion. In the cervical region, occupying a position between the postero-internal and postero-external tracts, is a strand of fibres which degenerates downwards for a short distance after a transverse lesion in this locality. This is the 'comma-shaped' tract of Schultze, formed of endogenous fibres, probably commissural, between adjacent segments of the cervical region. (Fig. 88.)

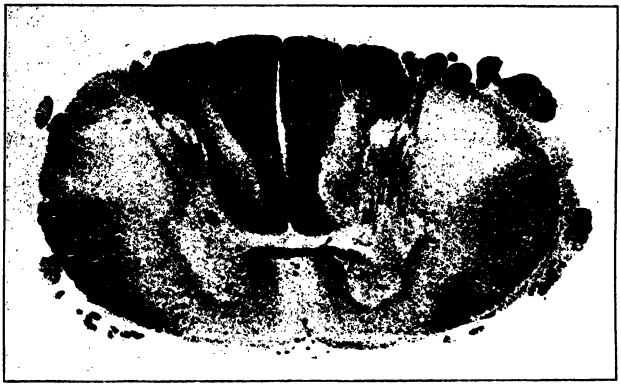


FIG. 88.—Showing the degenerations descending from a transverse lesion in the cervical region. The degeneration in the posterior columns is the 'comma tract' of Schultze; an extensive degeneration is also seen in the antero-lateral columns.

Other small descending tracts—the septo-marginal tract, the oval field, and the ventral posterior field—are probably portions of the same system of endogenous fibres at different levels of the cord.

2. The *antero-lateral columns* contain both ascending and descending tracts, some of which are of exogenous and others of endogenous origin.

(*a*) DESCENDING TRACTS. If we look, in the first place, at the tracts which degenerate below a transverse lesion of the cord, it will be seen that they occupy both the posterior and antero-lateral parts of the lateral column and the anterior column. (Fig. 88.)

The *crossed pyramidal tract* is situated in front of the posterior horn and is separated from the periphery, except in the lumbo-sacral region, by the dorsal cerebellar tract. If two sections of the cord be compared, one taken from below

a transverse lesion of the cord and the other from a case of cerebral hemiplegia, it will be seen that the descending tract of degeneration in the lateral column in the latter case is much smaller than in the former. In the cerebral lesion we see the fibres of the crossed pyramidal tract alone; in the cord lesion another descending tract—the rubro-spinal—is observed in front of the pyramidal bundle. (Figs. 88 and 89.)

The *rubro-spinal* tract descends from the opposite red nucleus through the pons and medulla, enters the cord at the decussation, and lies immediately in front of the crossed

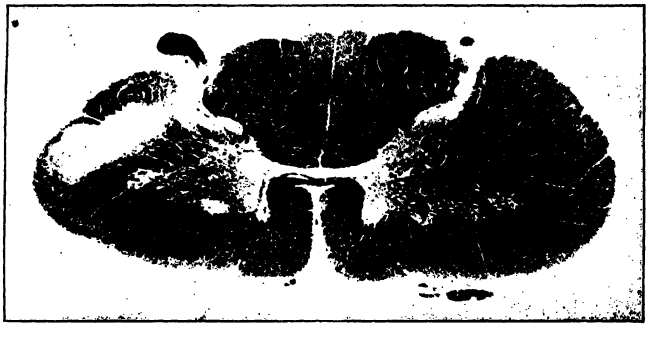


FIG. 89.—Showing the degeneration of one crossed pyramidal tract resulting from a cerebral lesion (hemiplegia).

pyramidal tract and partly mixes with its fibres. It can be traced into the mid-thoracic region.

The *direct pyramidal tract* occupies the marginal area along the anterior median fissure, and mixed with it are *descending sulco-marginal* fibres.

Occupying an antero-lateral position in the periphery of the cord is the antero-lateral descending, or *vestibulo-spinal* tract, an exogenous bundle of fibres issuing from Deiters's nucleus. (Fig. 87.)

(*b*) ASCENDING TRACTS. A study of the tracts which degenerate in an ascending direction in the antero-lateral columns will reveal the following:—

The *dorsal cerebellar tract* of Flechsig, which arises in the cells of Clarke's column and occupies the postero-lateral periphery of the lateral column (p. 19).

The *ventral cerebellar tract* of Gowers, occupying the antero-lateral periphery of the cord (p. 19).

Along the margin of the anterior fissure, and partly mixing with the fibres of the direct pyramidal tract, is the *ascending sulco-marginal tract.*

A small triangular bundle of fibres, which would seem to pass to the inferior olivary body, has been found in the upper cervical region, and is known as *Helweg's bundle.*

Although the tracts just described form the great mass of the white substance of the spinal cord, those in immediate relation to the grey matter have received special designation. The white matter lying between the crossed pyramidal tract and the lateral portion of the grey matter is known as the *lateral limiting layer*; and the white substance in immediate relation to the anterior horn is called the *anterior ground bundle.* These areas of white matter, with the posterior ventral field of the posterior columns, are probably composed of endogenous and association fibres, linking up adjacent segments as well as those situated somewhat more distantly.

RELATION OF THE SPINAL SEGMENTS TO THE VERTEBRAL SPINOUS PROCESSES

The spinal cord is built up of a number of superimposed segments, corresponding more or less approximately to the vertebral bodies. Each segment is furnished with a pair of spinal nerve roots—anterior and posterior—which enter and leave the spinal canal by the intervertebral foramina, at varying distances below the corresponding spinal segments. As the spinal cord itself terminates at the lower border of the first lumbar vertebra, the nerve roots corresponding to the lower lumbar and sacral segments pass vertically downwards for some distance within the canal towards their foramina of exit. Here they form the *cauda equina,* in which the higher or lumbar roots lie external to the lower or sacral roots.

It is therefore apparent, from this disposition, that the seat of a lesion within a segment of the cord is at a higher level in the vertebral canal than that indicated by the anæsthesia of the corresponding posterior nerve roots. As the relation of the nerve roots to the vertebral spines and the segments of the spinal cord is surgically important, a table

has been constructed to show their relative position throughout the spinal column.

TABLE SHOWING THE RELATION BETWEEN THE POSITION OF THE SPINAL SEGMENTS AND THE VERTEBRAL SPINOUS PROCESSES

Level of spinal segment.	*Vertebral spine.*
C 1	above C 1
C 2	above C 1
C 3	C 1
C 4	C 2
C 5	C 3
C 6	C 4
C 7	C 5—6
C 8	C 6—7
D 1	C 7
D 2	D 1
D 3	D 2
D 4	D 2—3
D 5	D 3—4
D 6	D 4—5
D 7	D 5—6
D 8	D 6—7
D 9	D 7—8
D 10	D 8
D 11	D 9
D 12	D 10
L 1	D 10—11
L 2	D 11
L 3	D 11—12
L 4—5	D 12
L 5— S1	D 12
Sacrum	L 1

These observations show that in the upper cervical and upper dorsal regions, the nerve roots issue about one vertebral body below the level of their corresponding spinal segment; that in the lower cervical and mid-dorsal regions the difference amounts to about a body and a half; and in the lower dorsal region to two vertebral bodies. In the lumbar

region, owing to the rapid shortening of the spinal cord, all the lumbar segments correspond to the eleventh and twelfth dorsal, and the sacral segments to the first lumbar vertebra.

Vascular Supply of the Spinal Cord

The arteries of the spinal cord are: the anterior spinal artery, which passes along the front of the cord and gives off a median branch distributed to the anterior horns and central grey matter; and two smaller posterior spinal arteries, which pass along the line of the posterior nerve roots and give branches into the posterior horns. (Fig. 87.)

In addition to these main arterial supplies, numerous small vessels pierce the periphery of the cord in all directions from the encircling sheath of pia-arachnoid membrane.

The grey matter is more richly endowed with blood-vessels than the white: a fact which probably explains the greater tendency for lesions of a vascular nature—such as poliomyelitis and hemorrhage—to occur in this part of the cord.

The perivascular lymphatic spaces communicate with the subarachnoid space outside the spinal cord.

LOCALISATION OF FUNCTION IN THE SPINAL CORD

The following description is in no way a full account of the functions of the spinal cord, but is intended as a guide to the clinical localisation of lesions within the cord.

The spinal cord has the following functions:—

1. It conducts motor impulses from the cerebrum by the crossed and direct pyramidal tracts.
2. It conducts sensory impressions to the cerebrum by the posterior columns and certain ascending tracts.
3. It conducts afferent and efferent cerebellar impulses: the former by the dorsal and ventral spino-cerebellar tracts, the latter by the vestibulo-spinal tract.
4. It contains within its segments centres for reflex action.
5. It contains trophic and vaso-motor centres.
6. It contains tracts and fibres connecting the various segments with each other.

1. Motor conduction in the spinal cord

A lesion of the pyramidal system in any part of its course is followed by degeneration of the pyramidal tracts below the level of the lesion.

The symptoms consequent upon a lesion of the pyramidal tracts are :—

(*a*) A spastic paralysis or paresis of the muscles below the lesion, characterised by—

(1) An increase of the muscle tonus.

(2) Exaggeration of the deep reflexes.

If the spastic paralysis involves the muscles of the abdomen and lower limbs,

(3) Diminution or loss of the superficial abdominal and epigastric reflexes.

(4) An alteration in the character of the plantar reflexes, whereby the normal flexion of the big toe is replaced by extension (p. 44).

(*b*) Impairment of sphincter control—

(1) As regards the vesical sphincter, either precipitancy and incontinence, or hesitancy and retention.

(2) As regards the rectal sphincter, retention of fæces, except on the administration of aperients, when incontinence is the rule.

2. Sensory conduction in the spinal cord

The sensory tracts and the conduction of sensory impressions in the spinal cord have been already described on pp. 9 to 18.

The following summary may be given here :—

(*a*) The senses of discrimination of two points and of passive position of the limbs pass up the homo-lateral posterior columns, and cross in the decussation of the fillet.

(*b*) Tactile sensibility and its localisation and the sense of pressure pass up the homo-lateral posterior columns for an indefinite distance and then decussate to the opposite antero-lateral column.

(*c*) All forms of painful and thermal sensibility decussate at once and pass up by way of the opposite antero-lateral columns.

It is not possible to say precisely what are the symptoms of lesion of the individual sensory columns, but as far as is known they are the following:—

Symptoms of lesion of the posterior columns:

(*a*) Loss of the sense of passive position of the limbs.

(*b*) Loss of cutaneous discrimination.

(*c*) Impairment of light touch, and cutaneous localisation, dependent upon the extent or degree of the lesion.

Symptoms of lesion of the antero-lateral columns:

(*a*) Abolition or impairment of thermal sensibility.

(*b*) Abolition or impairment of painful sensibility on the opposite side—the sense of temperature to probably a greater degree than that of pain.

3. Cerebellar conduction in the spinal cord

Two tracts, the dorsal and ventral spino-cerebellar tracts, convey impressions from the posterior nerve roots, through the spinal cord, to the cortex of the cerebellum.

It is difficult to state what the functions of those tracts are individually, and apart from lesion of the adjacent tracts. There are a few disorders, chiefly of a degenerative character, in which these tracts are diseased, and in such cases the chief symptom is cerebellar instability (p. 478).

Lesion of the cerebellar cortex is followed by some degree of hypotonia of the homo-lateral side. It might therefore be argued that, as these tracts form the afferent limb of the cerebellar arc, their destruction would also result in diminished muscular tone.

The functions of the efferent vestibulo-spinal tract are likewise unknown, but it would appear probable that this tract is concerned in the regulation and co-ordination of muscular movements.

4. Reflex centres in the spinal cord

Each segment of the spinal cord contains reflex centres for the corresponding segment of the body and limbs.

In its simplest form *reflex action* consists of an involuntary muscular contraction excited by a peripheral stimulation through a spinal segment. For reflex action it is necessary to have an afferent or sensory nerve with its posterior root, a

connexion between the posterior root and the anterior horn cells, an anterior root and motor nerve and a peripheral muscular mechanism.

Normal reflex action indicates a healthy condition of the limbs and central portion of the reflex arc in a particular segment. But it is obvious, from a consideration of the symptoms following lesions of the conducting paths of the spinal cord, that the reflex centres are connected both with the higher and the lower parts of the nervous system, as shown by the alterations in the character of the reflexes, associated with disease of these structures. For example, the knee jerk or the ankle jerk may be abolished by a lesion of the conducting strands between the posterior and the anterior horns; the plantar reflex, which in health consists mainly of flexion of the big and small toes on stroking the sole of the foot, is converted into a movement of extension of the big toe when the pyramidal tracts are disorganised. Under a like condition the normal contraction of the abdominal muscles ceases when the skin of the abdomen is stroked, but the tendon jerks show varying degrees of exaggeration.

An examination of the reflexes, therefore, throws light not only upon the condition of the spinal segment as a reflex centre, but also upon the condition of the conducting paths in the brain and spinal cord.

Alterations of the spinal reflexes, therefore, indicate either an interference with the arc itself, or with the superior controlling structures.

1. *Tendon jerks.*

(*a*) Interruption of the primary reflex arc causes impairment or abolition of the jerks.

(*b*) Lesion of the pyramidal system causes exaggeration of the jerks.

(*c*) Complete severance of the cord causes abolition of the jerks below the lesion.

2. *Superficial reflexes.*

(*a*) Interruption of the primary reflex arc causes abolition of the reflexes.

(*b*) Lesion of the pyramidal system leads to ready exhaustion, impairment or abolition of the reflexes.

(*c*) Complete severance of the cord causes their abolition.

3. *Plantar reflexes.*

(*a*) Interruption of the primary arc causes abolition of the reflex.

(*b*) Lesion of the pyramidal system leads to an extensor response.

(*c*) Complete severance of the cord causes abolition.

The substitution of an extensor for the normal flexor response depends upon some affection of the pyramidal system.

Segmental localisation of the principal spinal reflexes

Tendon.		*Superficial.*	
Biceps	C 5—6	Palmar	C 8—D 1
Supinator	C 6—7	Epigastric	D 5—D 9
Triceps	C 6—7	Hypogastric	D 10—12
Quadriceps	L 3—4	Cremasteric	L 1—2
Achilles	S 1—2	Plantar	S 1—2
		Anal	S 5

5. Segmental localisation

Each segment of the cord contains spinal centres for the muscles of the body and limbs, and receives by the corresponding nerve roots sensory impressions from definite skin areas.

(*a*) Motor Localisation

Each spinal segment contains motor cells for several muscles, but one muscle, or a group of muscles, derives its chief supply from one segment. The intercostal muscles appear to be supplied from a single segment, but, according to Sherrington, most muscles receive nerve fibres from three spinal segments. Each spinal segment in the cervical and lumbar enlargements presides over several muscles. A lesion of a single segment weakens the action of a number of muscles, although the paralysis falls especially upon one particular muscle or group of muscles.

The symptoms of lesion of the anterior horns vary according to the individual segment or segments affected, but the following general features are characteristic of paralysis of this region:—

Flaccid paralysis of the muscles supplied by the affected segment characterised by—

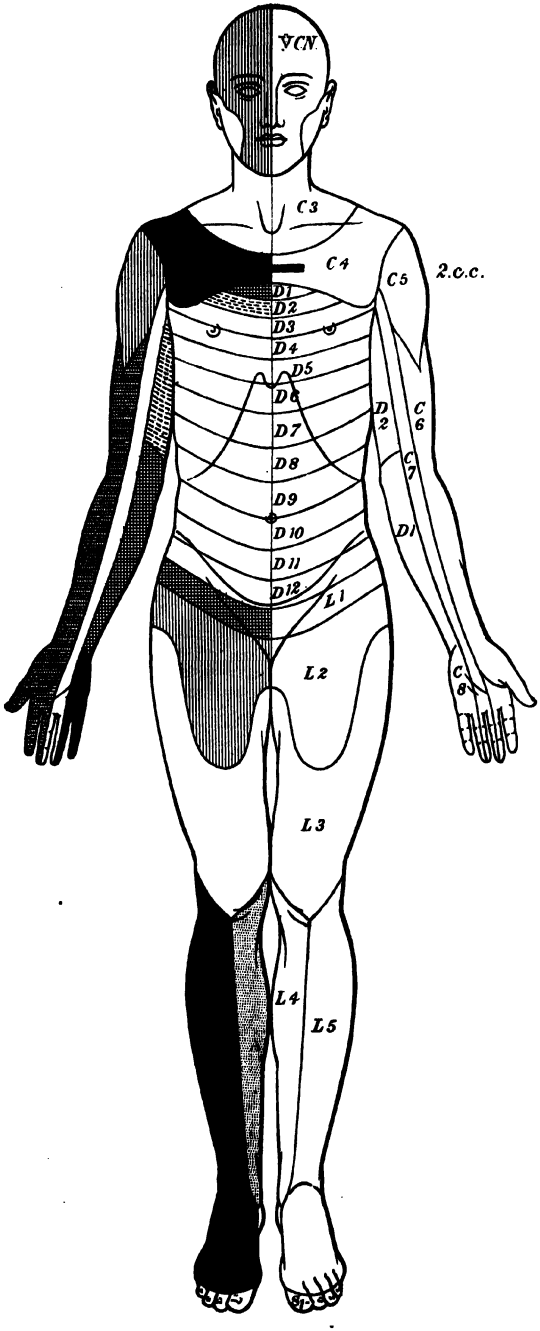


Fig. 90.—Front view of the body and limbs showing the skin areas corresponding to the spinal segments.

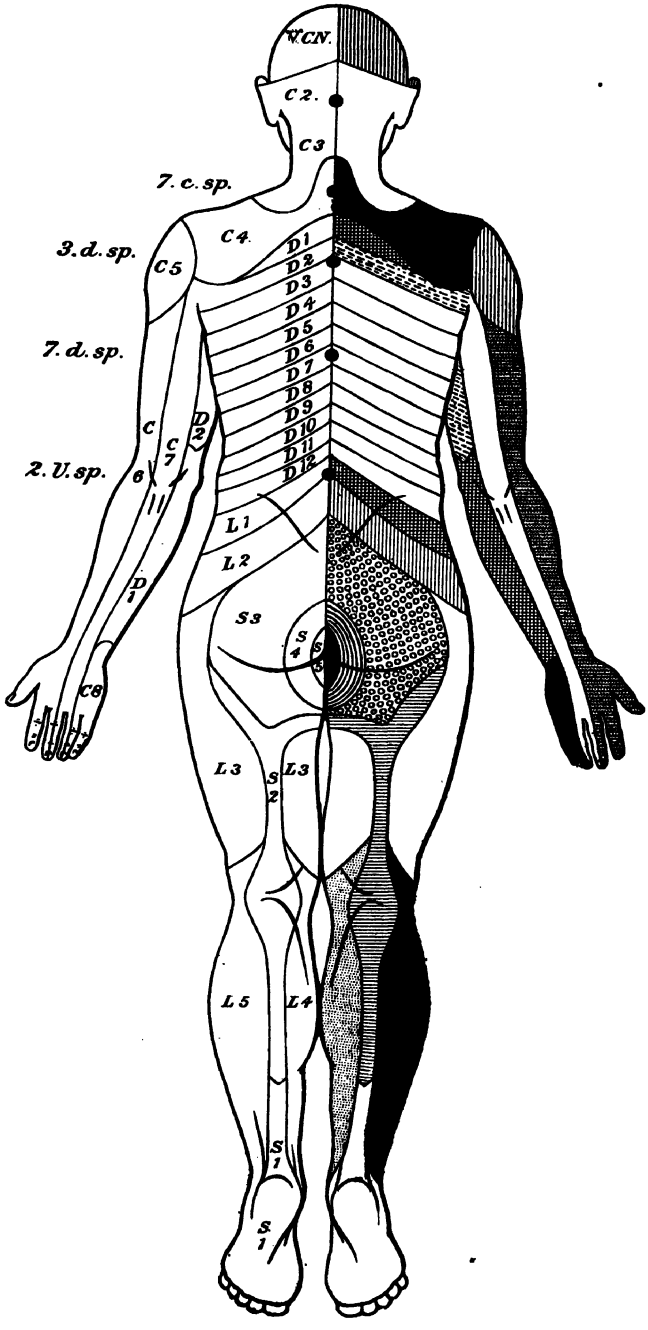


FIG. 91.—Back view of the body and limbs showing the skin areas corresponding to the spinal segments.

(*a*) Muscular atrophy.
(*b*) The reaction of degeneration (p. 51).
(*c*) Loss of the deep and superficial reflexes subserved by the affected muscles.

TABLE SHOWING THE PRINCIPAL MUSCLES AND THEIR SPINAL REPRESENTATIONS

C 2—3	**Deep muscles of the neck,** trapezius, sterno-mastoid.
C 3	**Levator anguli scapulæ,** trapezius, sterno-mastoid, diaphragm scaleni.
C 4	**Diaphragm,** trapezius, levator anguli scapulæ, scaleni, rhomboids, deltoid, spinati, biceps, supinator longus.
C 5	**Deltoid, spinati, teres minor, rhomboids,** diaphragm, biceps, supinator longus, serratus magnus, pectoralis major, brachialis anticus, coraco-brachialis.
C 6	**Biceps, coraco-brachialis, brachialis anticus, supinator longus,** deltoid, spinati, teres major, serratus magnus, pectoralis major, subscapularis, pronators, extensors of wrist.
C 7	**Triceps, extensors of wrist and fingers,** pronators, pectoralis major, subscapularis, latissimus dorsi, teres major.
C 8	**Flexors of wrist** and **long flexors of fingers, interossei,** lumbricales, thenar and hypothenar muscles.
D 1	**Muscles of thenar** and **hypothenar eminences,** interossei, lumbricales, flexor carpi ulnaris, oculo-pupillary fibres.
D 2—12	**Intercostals, rectus abdominis,** serratus posticus, internal and external obliques.
L 1—2	**Ilio-psoas, quadratus lumborum,** sartorius, cremaster, quadriceps.
L 3	**Quadriceps,** sartorius, quadratus lumborum, adductores, obturator externus.
L 4	**Adductores,** quadriceps, sartorius, tensor fasciæ femoris, tibialis anticus, extensor communis, extensor hallucis.
L 5	**Tibialis anticus, extensor communis digitorum, extensor hallucis, peronei,** abductors and external rotators of hip, gastrocnemii, flexor longus digitorum, hamstrings, glutei.
S 1	**Gastrocnemii, hamstrings, long flexors of toes,** peronei, abductors and external rotators of hip, glutei.
S 2	**Glutei, intrinsic muscles of foot,** gastrocnemii, hamstrings, long flexors of toes.
S 3—5	**Muscles of perinæum** connected with **defæcation, micturition, erection, ejaculation.**

NOTE.—Thick type indicates the principal supply of each segment.

(*b*) Sensory Localisation

It has been demonstrated, both clinically and experimentally, that a lesion which involves the whole transverse area of the cord in any one segment, produces a loss or diminution of sensibility below the level of the lesion, and a slight alteration in the sensibility in the areas supplied by the segment above. Each spinal segment receives by its posterior nerve roots sensory impressions from more or less definite areas of the body; but inasmuch as the sensory supply of any one of these areas is provided for, not by fibres entering one posterior root alone, but partly by fibres which enter by the adjacent superior and inferior roots, a complete lesion in one segment results in (1) complete loss of sensibility below the level of the lesion; (2) considerable, but not complete, loss in the area supplied chiefly by the posterior roots of the affected segment; and (3) slight impairment of sensibility in the area supplied by the posterior roots of the segment above.

The quality of the sensory loss differs from that seen in lesions of the peripheral sensory nerves owing to the readjustment of the grouping of the various forms of sensibility in the spinal cord. And it also follows that partial lesions of the spinal cord cause loss of some forms of sensibility more than of others—dissociation of sensibility according to the involvement of the various afferent paths within the cord.

For the size, shape, and position of the several skin areas, the reader is referred to the annexed charts. (Figs. 90 and 91.)

The following is a summary of the symptoms of **a total transverse lesion of the cord**:—

(*a*) Complete flaccid palsy below the level of the lesion, with wasting of the muscles and loss of their faradic excitability.

(*b*) Complete abolition of sensation below the level of the lesion, marked impairment corresponding to the lesion, and slight impairment corresponding to the segment above it.

(*c*) Abolition of all the reflexes.

(*d*) At first retention, later dribbling of urine and constipation.

(*e*) Trophic changes and bed-sores.

Chapter II

INTRINSIC DISEASES OF THE SPINAL CORD

1. ACUTE MYELITIS

Although the term 'myelitis' really signifies an inflammation of the spinal marrow, it has come to be applied to a number of spinal disorders which are not of an inflammatory character. More intimate study of the nature and pathology of the diseases of the spinal cord has reduced the number of different forms of so-called myelitis, the great majority of such cases being really due to thrombotic lesions of the spinal blood-vessels, arising chiefly from syphilitic endarteritis.

The following are the disorders to which the term 'myelitis' has been applied :—

1. *Thrombotic myelitis*, a primary condition of softening of the spinal marrow (myelomalacia) associated with blocking of the spinal blood-vessels, and usually of syphilitic origin (described on p. 374). A chronic form, known as senile paraplegia, is seen in old people.

2. *Pressure myelitis*, a chronic form, attributable to vertebral disease, meningeal lesions, or to the pressure of tumours without or within the theca spinalis (described on p. 346).

3. *Infective myelitis*. This may be primary, or secondary to such general conditions as enteric fever, smallpox, dysentery, malaria, measles, tonsillitis, gonorrhœa, and cystitis.

We propose to limit the term *acute myelitis* to a somewhat rare inflammatory condition of the spinal cord.

Infective Myelitis—Acute Myelitis

Etiology. This may be primary, or secondary to such conditions as enteric fever, smallpox, dysentery, malaria, measles, tonsillitis, gonorrhœa, and cystitis.

Pathology. The morbid appearances are characteristic of an acute infective process in the spinal cord. It is not more frequent in the dorsal region, but may extend, more or less, throughout the whole of the spinal cord and

upwards into the bulb. Both white and grey matter share in the inflammation, but the posterior columns are generally the seat of the most intense changes. The arteries are distended and engorged with blood, and around them a marked proliferation of small cells is observed with a few polymorphonuclear leucocytes. Around this is an area of proliferated neuroglial cells. The myelin sheaths are swollen and broken up, and there may be destruction of axis-cylinders. The large cornual ganglion cells may, or may not, be affected.

A similar vascular engorgement and perivascular proliferation is seen in the spinal meninges.

In old-standing cases sclerosis is seen in the affected areas, and sections of the cord show ascending and descending degenerations.

The cerebro-spinal fluid has been found to contain leucocytes and diplo-bacilli. According to Farquhar Buzzard,[1] the character of the lesions just described suggests a bacterial disorder resulting from an infection of the lymphatic system.

Acute myelitis occurs in three forms—acute transverse myelitis, acute ascending or disseminated myelitis, and acute suppurative myelitis or abscess of the spinal cord.

Acute Transverse Myelitis

Symptoms. The onset of the spinal symptoms is preceded, or accompanied by, general symptoms of malaise, fever, and pain in the back. The onset is usually rapid, without being sudden, that is to say, the full effect of the lesion may take several hours to develop. It is impossible to lay down any rule as to the length of time which may elapse between the onset of the first symptom and the full development of the paralysis.

The first symptoms are usually subjective sensations of pain, numbness or burning. When present, the painful sensations are referred to the upper level of the affected segments, the numbness and tingling to the limbs below the level of the lesion. Pain at the level of the lesion is universal in cases in which the lesion is a meningo-myelitis, and as it is rare for an infective condition to be limited to the spinal cord, pain is usually present.

[1] Farquhar Buzzard, *Brain*, 1907.

Numbness or paræsthesia of the limbs may precede weakness, but some degree of motor paralysis is usually coexistent. In the early stages, reflex spasms of the extremities below the lesion give rise to pains and cramps, sometimes of a severe character. In other cases, the motor weakness develops before the sensory functions are involved. This may consist first of a feeling of heaviness or weight in the legs,

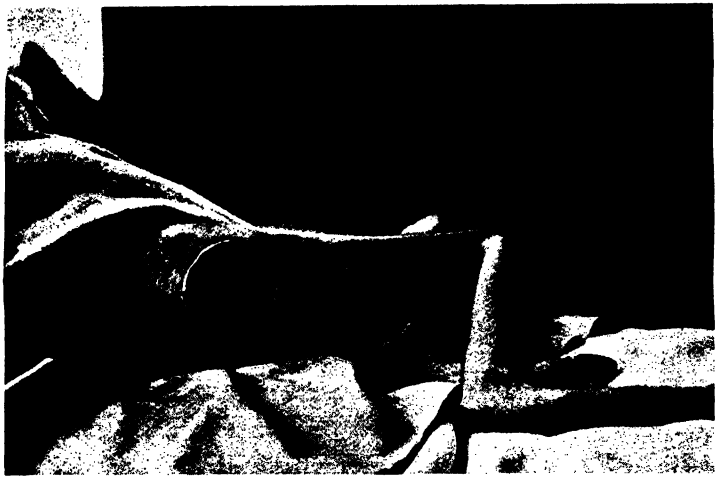


FIG. 92.—Shows a large bed-sore over the great trochanter in a case of acute myelitis.

which soon gives place to obvious and complete motor paralysis.

Loss of sphincter control may be an early symptom, but in the acute cases the sensory, motor, and sphincter functions usually suffer together with the establishment of a complete transverse lesion of the cord.

The clinical picture presented by such a case is as follows:

1. *Symptoms below the level of the lesion.*

(*a*) Complete flaccid motor paralysis.

(*b*) Complete loss of the reflexes—superficial and deep.

(*c*) Complete loss of all forms of sensation.

(*d*) Complete abolition of sphincter control, so that incontinence of urine and fæces are present.

(*e*) Trophic changes—such as bed-sores. (Fig. 92.)

2. *Symptoms corresponding to the level of the lesion*, which may extend over two or more spinal segments.

(*a*) Flaccid motor palsy of the muscles supplied from the anterior cornual regions.

(*b*) A loss of all forms of sensation over the corresponding area. At the upper margin sensation may not be completely abolished, and subjective sensations of pain and constriction are frequently present in association with objective hyperæsthesia.

(*c*) Abolition of the reflexes innervated from the corresponding segments.

If the lesion be *transverse but incomplete* in degree, the clinical picture is modified chiefly in respect of the motor and reflex signs. The voluntary power is lost or impaired below the lesion, but the condition of the muscles is that of spastic paralysis with increase of the myotatic irritability, the deep reflexes are exaggerated, and clonus is easily obtained.

The superficial abdominal reflexes are abolished, and the plantar response is of extensor type. There may also be incontinence of urine and loss of expelling power of the bladder and rectum.

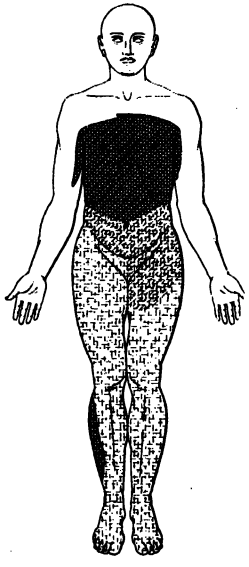


Fig. 93. Showing the distribution of the sensory loss in a case of acute meningo-myelitis. The dark area corresponds to the seat of the lesion and represents complete abolition of sensation; over the stippled zone the sensory loss is incomplete.

The sensory symptoms conform to those seen in the complete transverse lesion, in so far as the upward extent of the affection of sensibility is concerned; but the degree of impairment varies and some degree of dissociation may be observed. (Fig. 93.)

The trophic functions are also disturbed, as in the complete cases.

Prognosis. Recovery in the infective cases is rare. If it occurs, it is characterised by a lessening of the sensory impairment and of the trophic disturbances, by the development of spastic paraplegia with contractures—especially of the flexor and adductor muscles—and slow return of voluntary power.

The permanent condition is, as a rule, that of persistent paraplegia and contractures.

Treatment. The patient should be placed upon a water bed. Great care should be taken to prevent the development of bed-sores; constant attention is necessary, all points of pressure should be protected, and the skin kept clean and dry. The use of the catheter requires to be carried out with rigid antiseptic precautions. In the event of cystitis being present, the bladder should be washed out daily with a sterile solution of boracic acid, and urotropin given internally every six hours. Careful attention should also be paid to the bowels—the rectum being emptied at a fixed time each day; the action of a mild aperient being aided if necessary by an enema.

Drugs are of little use in the general treatment of the disease, although they may be of service in relieving symptoms. Flexor spasms of the legs may be allayed by the bromides, cannabis indica, or veronal.

After the acute symptoms have passed away, massage and passive movements to prevent contractures and deformities may be successfully employed. Spasticity is counteracted by passive movements, carefully regulated exercises, and if necessary extension by weights.

Acute Ascending Myelitis

(syn.: acute disseminated myelitis, acute ascending meningo-myelitis)

Pathology. According to Buzzard, the pathological changes consist of a lepto-meningitis and scattered patches of softening in the white and grey matter. Hemorrhages are not infrequent, and the brain may be hyperæmic.

Microscopically, foci of acute inflammation are found either in patches or involving the whole transverse section of the cord, both of the white and grey substance. The vessels are engorged and there is a cellular proliferation in the adventitial lymphatic sheath. Proliferation of the neuroglial cells, which are swollen, multi-nuclear and sometimes vacuolated, is observed round the vessels. The nerve fibres may be swollen or may have entirely disappeared. The blood is not coagulated.

Around the vessels are mono-nuclear cells, and further afield are polymorpho-nuclear leucocytes.

Cellular proliferation is also detected in the meninges. Amyloid bodies are found in the periphery of the cord and around the blood-vessels. Degeneration of the ganglion cells is in proportion to the existing inflammation. In contra-distinction to the changes found in acute poliomyelitis, the cells may be swollen and chromatolytic without inflammatory exudation, except in the adjoining white matter.

These changes may be due to many varieties of bacteria, and are of the type seen in typhoidal myelitis, and in old cases of 'urinary paralysis.' According to Buzzard, it is a bacterial infection of the lymphatic system of the spinal cord—a spinal lymphangitis.

Symptoms. The clinical picture presented by this malady is that of a rapidly ascending motor and sensory paralysis. The disease commences suddenly, often with pain or a feeling of constriction round the abdomen or chest, which is followed, some hours later or the next day, by weakness and numbness of the lower limbs and sphincter trouble. During this period the deep reflexes may be increased and an extensor plantar response may be present. The weakness develops, often quite suddenly, into a complete paraplegia, so that the condition now presented is that of complete flaccid paralysis with sensory loss and abolition of the deep and superficial reflexes, including the plantar reflex, below the level of the lesion, but the electrical reactions remain normal. The palsy may spread upwards segment by segment, or symptoms pointing to a focal lesion higher up the spinal cord may supervene. The skin is dry, and there is great liability to the formation of bed-sores.

Although clinically the symptoms may suggest either a steady upward advance of the disease, or the existence of two or more focal segmental lesions, the pathological examination of such cases reveals widespread and disseminated myelitis, which may be hard to reconcile with the clinical appearances and symptoms.

Prognosis. The prognosis is as a rule unfavourable, the disease ending fatally, but cases are on record in which recovery has taken place. In those which do not die, recovery may be relatively complete, the patient being able to walk

with some spasticity of the limbs and impaired sphincter control.

Treatment. This is the same as for transverse myelitis.

Acute Suppurative Myelitis

Suppurative myelitis, or abscess of the spinal cord, apart from its occurrence in the course of a suppurative meningitis, is a rare affection. It has been found in association with

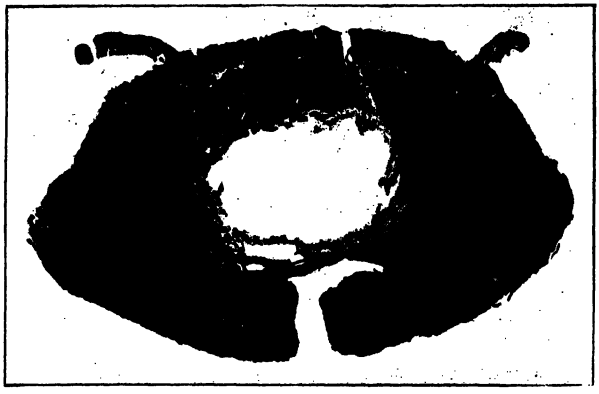


Fig. 94.—Photograph of an intraspinal abscess, secondary to old-standing tuberculous disease of the spine.

tuberculous and carcinomatous disease of the vertebræ, which have led to evascularisation and necrosis of the corresponding spinal segments, and therefore favoured the attacks of bacilli from the bladder or elsewhere. It may also occur as a primary condition. The pus in these cases spreads upwards and downwards from the original lesion and occupies a position chiefly in the anterior regions of the posterior columns and horns. In some places definite signs of an abscess wall may be discerned, but the spread of the pus seems to be along the lines of least resistance, causing secondary destruction of the adjacent white matter. (Fig. 94.)

The abscess cavity occupies a position similar to the lesion in syringomyelia, and to hemorrhage within the spinal cord.

Symptoms. When primary, the onset is similar to that of acute transverse myelitis. When secondary to tuberculous or carcinomatous disease of the vertebræ, the onset may be subacute.

As a general rule, it is impossible to distinguish this condition from acute transverse myelitis; but abscess has been diagnosed during life in the secondary cases by the presence of dissociated sensibility and the signs of an intramedullary lesion.

The **prognosis** is most unfavourable.

Treatment. This is the same as for transverse myelitis.

2. CAVITIES IN THE SPINAL CORD

There are four conditions in which cavity formation is found in the spinal cord.

(*a*) Hydromyelia, or distension of the central canal, unaccompanied by structural changes other than those due to compression of adjacent tissues. No symptoms may be present, but its importance lies in the fact that injuries to the spine may lead to hemorrhage into the cavity.

(*b*) Gliosis, or new formation, occurring in the central grey matter, within which cavity formation occurs as a result of degenerative changes.

(*c*) Cystic degeneration of gliomatous and sarcomatous tumours within the spinal cord (p. 336).

(*d*) Tuberculous, vascular, or inflammatory lesions, which may result in softening and cavity formation (p. 374).

(*e*) Syphilitic pachymeningitis.

As the two last conditions are described under their respective headings, the condition known as *spinal gliosis* will receive consideration only in this place.

Syringomyelia or Spinal Gliosis

Etiology. Syringomyelia occurs more commonly in men than in women; the most frequent period for its commencement is between twelve and thirty years of age. There is no predisposing cause; but in many cases the history of a spina bifida may be obtained, or a cicatrix detected, on examination of the back. Injuries to or falls on the lower part of the spine have been cited as exciting causes, but conclusive evidence on this point is lacking.

Pathology. The essential feature of this disease is the occurrence of a gliosis with cavity formation. This is

probably the result of a proliferation of the ependymal cells and tissues surrounding the central canal. Retrograde changes occur in the centre of the new growth, resulting in the formation of cavities, which may contain a serous or gelatinous fluid. The cavity is not lined with ependymal cells, except in the rare cases in which it opens into the central canal. The gliosis is characterised by a profusion of glial fibres, amongst which is scattered a relatively small number of neuroglial cells.

The growth occupies the grey matter, and tends to invade the anterior horn, the anterior portion of the posterior columns, and to extend as a fissure along the posterior horn.

Examination by the naked eye shows the cord at the level of the cavities either increased or diminished in size. When increased, the cord feels to the touch like an india-rubber tube. In transverse section the walls of the cavity present the appearance of a glistening thick membrane, and ascending and descending degenerations may be visible in the columns of the cord.

The portions of the cord chiefly affected are the cervical and upper dorsal regions, the lumbar region, and sometimes the medulla oblongata (syringo-bulbia).

The view that this condition is congenital is supported by those cases in which the remains of a spina bifida, or meningocœle, are found in association with the cavity formation and when the cavity is lined with ependymal cells. In many cases, on the other hand, there is no evidence of a congenital origin, and the condition ought to be regarded as a neuroglial overgrowth.

Symptomatology. The symptoms of the disease vary according to the situation and extent of the lesion. If the cervical and upper dorsal portions of the cord are alone affected, the local symptoms will be referred to the upper limbs and to the cervical sympathetic; if, on the other hand, the condition extends upwards to the medulla, bulbar symptoms will be present.

Syringomyelia produces motor and sensory symptoms in one or both of two ways.

(*a*) By interfering with incoming sensory and outgoing motor fibres through lesion of the central grey matter of the affected segment or segments. In the early stages

these may be the only symptoms, and consist of segmental loss of either thermal sensibility, thermal and painful sensibilities, or later, of all forms of sensation in the corresponding root areas, which may or may not be associated with segmental muscular atrophies. The afferent sensory fibres, especially those which decussate immediately after their entrance into the cord, are apt to be involved by the growth, which extends posteriorly from the central grey matter into or along the posterior horn. This explains the dissociation of sensation which is found corresponding to the level of the lesion, as the fibres which subserve painful and temperature sensibilities cross at once, while those for tactile sensation pass mainly up the same side (p. 17).

(*b*) The growth of the lesion may induce a further series of symptoms by pressure upon the long ascending and descending conduction strands. The symptoms of the later stages are, therefore, impairment of all forms of sensibility and spastic paralysis below the level of the lesion, of either unilateral or bilateral distribution.

It is possible that if the lesion does not extend eccentrically so as to interfere with the functions of the grey matter and adjacent commissural fibres, a cavity of considerable length may exist without giving rise to symptoms. This explains the finding of syringomyelic cavities at autopsies upon cases in which, during life, no symptoms had been detected.

In the majority of cases the clinical picture presents the following features:—

1. *At the level of the lesion.*

(*a*) A dissociation of thermal and painful from tactile sensibility.

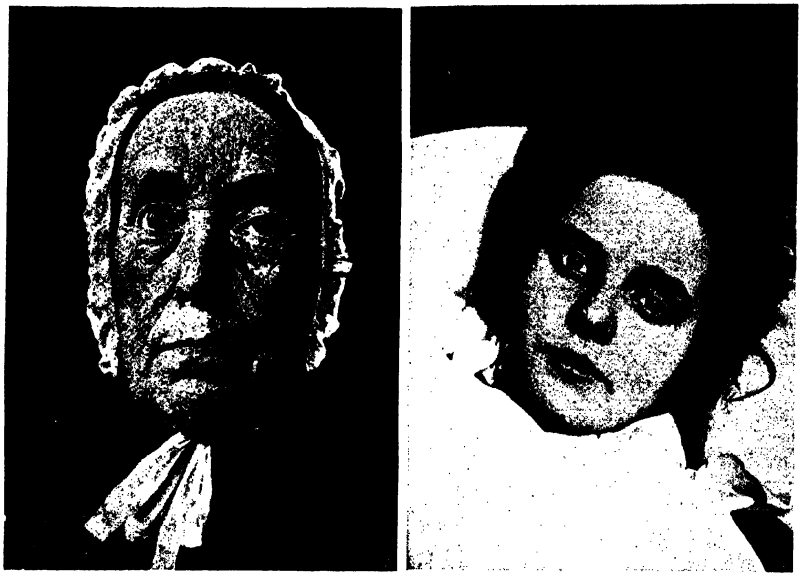
(*b*) Progressive muscular wasting.

(*c*) Vaso-motor and trophic changes.

2. *Below the level of the lesion.* In the late stages loss of sensation and spastic paralysis.

The disease shows itself primarily by disturbances of sensibility, by the occurrence of painless burns, or by trophic alterations. As the lesion commonly involves the cervical region of the cord, these changes are observed in the upper limbs. Pains radiating down the arm or up the neck may be an early symptom, but numbness and a feeling of deadness are the most frequent subjective sensations. These symptoms

may be accompanied, or shortly followed, by some degree of motor weakness in the arms or shoulders, and later by atrophic paralysis. On the one hand, this condition may remain stationary for varying periods. On the other, a progressive or intermittent increase of the symptoms may supervene, with signs indicating interference with the



FIGS. 95 AND 96.—Illustrate paralysis of the cervical sympathetic nerve on the left side. The partial ptosis, enophthalmos, and contracted pupil are well seen.

functions of the tracts of the spinal cord above or below the level of the focal symptoms.

Optic nerves. Optic atrophy and contraction of the visual fields have been observed, but are rare.

Oculo-motor nerves. Nystagmoid jerkings, or nystagmus on extreme lateral deviation, are present in a large number of cases. Ocular palsies, especially of the sixth nerve, are also found. The pupillary light-reaction is retained, but inequality of the pupils may be observed from paralysis of the sympathetic nerve.

Fifth nerve. The sensory division is occasionally affected, more especially on one side, either with pains and hyper-

æsthesia, or with diminution or loss of all forms of sensibility; in some cases, however, tactile sensibility is retained. Trophic disturbances and motor paralysis are rare.

Seventh nerve. Facial palsy of the peripheral type may be present, with wasting and atrophy of the facial muscles.

The *eighth nerve* is rarely involved.

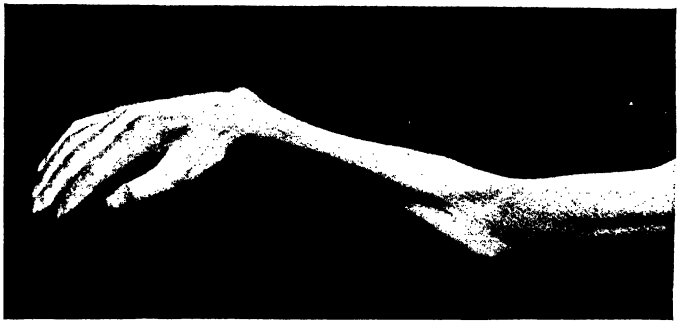


FIG. 97.—Illustrates muscular atrophy in a case of syringomyelia.

Bulbar nerves. Unilateral or bilateral paralysis of the tongue, vocal cords, pharynx, and palate are commonly observed.

Cervical sympathetic. This is more often affected than the other nerves by reason of its situation in the cervical region. Occasionally irritative phenomena, but more frequently paralytic symptoms, are observed—narrowing of the palpebral fissure, enophthalmos, and a contracted pupil, which does not dilate on stimulation of the skin of the neck or to cocain, but reacts to light and accommodation. (Figs. 95 and 96.)

The order of implication of the cranial nerves in syringomyelia is from below upwards, and is due to the spread of the disease from the spinal cord into the bulb, whereby the bulbar nerves are affected first, and the sixth pair are involved before the third nerves.

Motor system. The earliest motor symptom is a proneness to fatigue, often accompanied by a tired aching pain, generally in the upper extremities. Later on, wasting of the muscles with fibrillation is noted. In the majority of cases this commences first of all in the small muscles of the hands—interossei, thenar, and hypothenar groups—eventually

spreading into the forearm, arm, and shoulder. In a smaller proportion of cases, the scapulo-humeral muscles are the first affected. This is seen by inability to raise the arms, which hang flail-like by the sides. The wasting is bilateral, but often predominates on one side. There is also a type of the disease in which the lower limbs participate in the muscular wasting. In these cases the syringomyelia either commences in the lumbar enlargement or secondarily involves it.

Fig. 98. Showing wasting of the small muscles of the hand and ulnar side of forearm.

The muscular wasting does not necessarily spread in a definite order throughout the limb, but may miss portions; for example, it may proceed from the hand to the shoulder, leaving out the arm. The paralysis is often more marked than would be suggested by the atrophic state of the muscles. Spastic paralysis from interference with the pyramidal system may be bilateral or unilateral; and if severe in degree is always accompanied by sensory changes.

Sensory system. The disturbances of sensibility, as already explained, are of two kinds—one, segmental, corresponding to the segment or segments of the cord involved; the other, involving all forms of sensation in the body and limbs below the level of the lesion.

1. *Local changes.* In the earliest stages subjective sensations of heat and cold, with or without coexistent sensations of a painful character, are present, but are shortly replaced by objective thermo-anæsthesia, moderate degrees of temperature being felt merely as sensations of contact. In early cases the loss of temperature sensibility is limited to moderate degrees of heat and cold, but where there is complete loss of the extremes of temperature, the moderate degrees are also

abolished. Loss of sensation to heat or cold is not necessarily coterminous, as either the one or the other may be abolished over a greater area.

The loss of the sensibility to painful impressions may be preceded, in rare cases, by subjective sensations of a painful character, associated with cutaneous hyperalgesia. Analgesia, both superficial and deep, is the next most common sensory defect, and is so profound that burns, cuts, and wounds may be inflicted without the patient being aware of them.

Analgesia is not necessarily present as the earliest symptom, nor is it always coterminous with the area of thermo-anæsthesia. Once established it persists, although variations within small limits may occur. Its distribution is usually segmental, yet in many cases this is lost, or so modified as to resemble the 'glove' analgesia of hysteria.

In the early stages and in typical cases tactile sensibility is unimpaired; but in certain cases, especially in an advanced stage, impairment or loss of tactile sensibility may be observed, usually over areas not corresponding to those of thermal and painful anæsthesia.

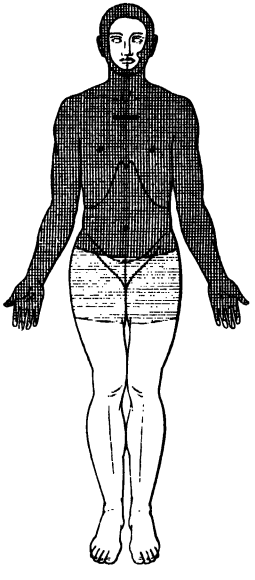


Fig. 99.—Chart of the sensory loss in a case of syringomyelia. The vertical lines indicate the area of thermal and painful loss; the horizontal lines indicate the area of tactile loss.

The local sensory changes, although not necessarily confined to the upper limbs and upper part of the chest and face, are more commonly found in these localities, owing to the position of the lesion within the cervical region of the spinal cord. (Fig. 99.)

2. *Distal changes.* Impaired sensibility may also be present below the level of the lesion, from interference with the conducting tracts. If the lesion is bilateral, impairment of all forms is found; but more commonly, owing to one side of the spinal cord being more involved than the other, the sensory loss assumes the features of the Brown-Séquard

'symptom-complex,' painful and thermal sensibilities being lost on the side opposite the lesion, whereas tactile sensibility may be impaired on the same side (p. 15).

Reflexes. The reflexes of the affected segments are abolished; but below the lesion the deep reflexes are increased, the epigastric or abdominal abolished, and the plantars extensor in type.

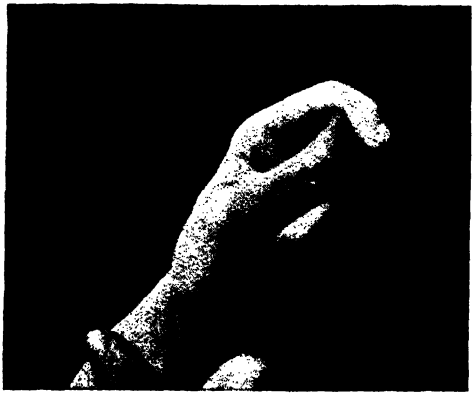


FIG. 100.—Shows the pincer hand ('main en pince') of syringomyelia.

FIG. 101.—Shows the succulent hand ('main succulente').

The *sphincters* are rarely affected in the cervical cases, but in cases with a lumbar lesion incontinence is an early symptom.

Trophic disturbances. The cutaneous changes are acute hyperæmia, redness, and local elevation of temperature, persisting for several hours or days. Passive hyperæmia is a late phenomenon, and often appears over bony prominences, and is due to mechanical obstruction to the circulation. Another form of passive hyperæmia is characterised by slight swelling of a bluish tint and reduction of local temperature. Secretory changes are found in increase or decrease of sweat secretion, the former more commonly in the earlier stages, and generally over the areas of impaired sensation. A vesicular eruption in certain nerve areas, lasting for a few days, without pain or irritation, and often confined to the analgesic zones, has been observed sufficiently frequently to deserve mention.

The succulent hand ('main succulente') depends upon vaso-

motor disturbance. The hand is swollen, especially over the dorsum, the skin is of a normal colour, pits on pressure, but soon recovers. The local temperature is raised. A similar condition is found in hemiplegics, where the hand is kept in a dependent position. (Figs. 100 and 101.)

Painless whitlows (Morvan's disease) appear upon the

FIG. 102.—Illustrates painless whitlows (Morvan's disease) in a case of syringomyelia.

fingers. These may be an early feature of the disease, but in many cases they are absent throughout. (Fig. 102.)

Arthropathies, similar to those seen in tabes dorsalis, may occur, usually in the upper limbs, affecting chiefly the shoulder, elbow, and inter-phalangeal joints. They are often the first symptoms of the disease. Spontaneous fractures are also liable to occur.

Scoliosis, or kypho-scoliosis of the spinal column, has been ascribed to articulatory changes, but is more probably due to weakness of the spinal musculature. It may be an early or a late symptom. It is always found in the upper dorsal region.

Diagnosis. In typical and well defined cases of syringomyelia the diagnosis is easy, as there is no other condition in which an association of muscular atrophy with dissociation of

sensibility as above described occurs. As the sensory changes are usually the earliest signs, there is little likelihood of the malady being confused with amyotrophic lateral sclerosis.

The two affections which are most likely to be confused with syringomyelia are: (1) spinal tumour with root symptoms, and (2) cervical rib.

In *spinal tumours*, the local paralyses correspond to the affected nerve roots; the sensory loss is of the peripheral type and usually unilateral; the pressure symptoms develop first upon the same side as the root symptoms.

In cases of *cervical rib*, the palsy is usually unilateral and confined to certain root areas; the sensory loss is of the peripheral type; there are no associated spinal symptoms, and an X-ray examination shows an additional rib.

Leprous neuritis, in the early stages, may present a close resemblance to syringomyelia, owing to the patchy character of the anæsthesia, and muscular atrophy. The previous history, thickening of the nerves and the absence of evidence of interference with the functions of the spinal cord, are the determining points.

Prognosis. The disease is progressive, but may persist for many years. Recovery is unknown, although arrest of the symptoms sometimes takes place.

Treatment. This is conducted on general principles; massage, faradisation, and the treatment of complications as they arise. Special care must be taken to avoid injury or burning over the anæsthetic areas.

3. INTRAMEDULLARY TUMOURS

The common forms of tumour within the spinal cord are glioma, sarcoma, tubercle, and gumma. Hydatids have occasionally been found.

Gliomata, the commonest form of new growth within the brain, are rarely met with in the spinal cord. These tumours tend to break down and form one of the varieties of cavity formation within the cord. This may be primary, analogous to that seen in the cerebellum, or it may be secondary to hemorrhage within the tumour. It is distinguished from syringomyelia by the presence of numerous neuroglial cells,

with few neuroglial fibres, and by the preservation in an intact state of the central canal and its ependymal lining. These growths primarily originate within the grey matter, and commonly spread into the white substance.

Sarcomata are either primary within the cord, or secondary to growths in the cerebrum, cerebellum, spinal roots, or meninges. Meningeal sarcoma may invade the spinal cord

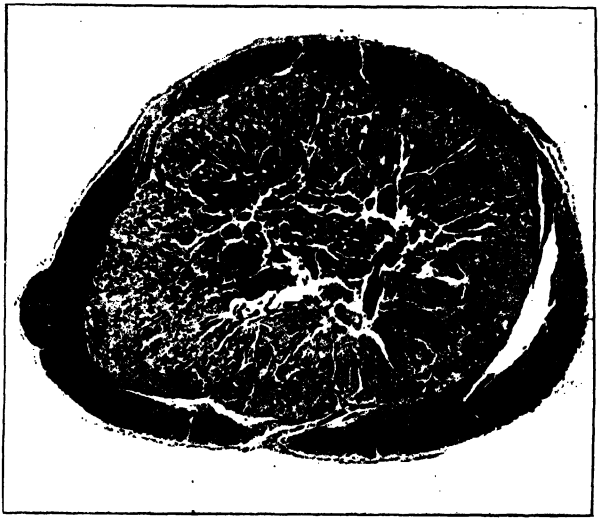


FIG. 103.—Showing a large intramedullary-tumour in the cervical region of the spinal cord.

along the pial vessels, the vessels of the central grey substance, or along the deep fissures. In two cases in which a primary growth invaded the fourth ventricle, secondary deposits were found growing from the interior of the central canal. Usually the cells are of the small or large round type. It is rare for such growths to attain any size without degenerative changes and cavity formation occurring in their central parts.

Tuberculous tumours occur in association with general tuberculous infection of the meninges, but have also been found as solitary growths similar to those occurring in other parts of the central nervous system.

Gummata are always found in connexion with the pia-arachnoid membrane or spinal blood-vessels.

The following are the ages at which the different types of intramedullary tumour are most common :—

Under 12 years . .	Tubercle, sarcoma, glioma.
12 to 40 years	Glioma, tubercle, gumma.
Over 40 years	Gumma, sarcoma.

Symptomatology. Intramedullary tumours give rise to symptoms depending upon their situation rather than their

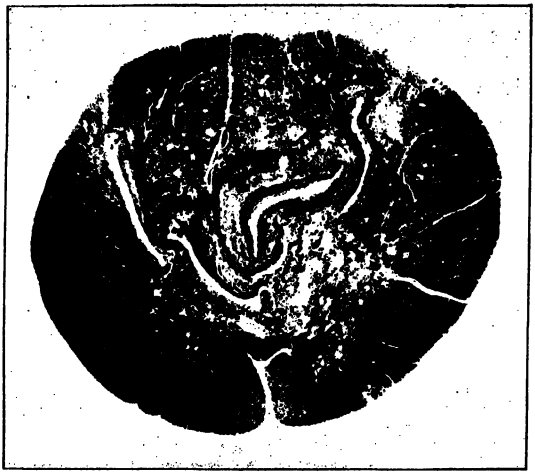


FIG. 104.—Photograph of the spinal cord showing cavity formation in association with spinal gliosis.

nature. They form a connecting link clinically between syringomyelia and the intradural form of extramedullary growths. Pure intramedullary tumours, or those which have not extended from the meninges, are rare, and present a symptomatology in all respects similar to that described under syringomyelia (p. 328).

Intramedullary tumours growing from the meninges and secondarily involving the cord present symptoms corresponding to those found both in syringomyelia and extramedullary tumours, but with the important clinical distinction that the early symptoms are those of an extramedullary growth (p. 350).

These may be for a time the only symptoms, but the physical examination reveals signs which cannot be explained

entirely by the existence of an extramedullary growth, and point to the presence of an intramedullary lesion.

The earliest signs are found in the realm of *sensation*.

1. Local segmental dissociation of sensation (loss of painful and thermal, with retention of tactile sensibilities).

2. The occurrence of the Brown-Séquard 'symptom-complex' below the level of the lesion (loss of painful and thermal sensibility on the side opposite the motor paralysis).

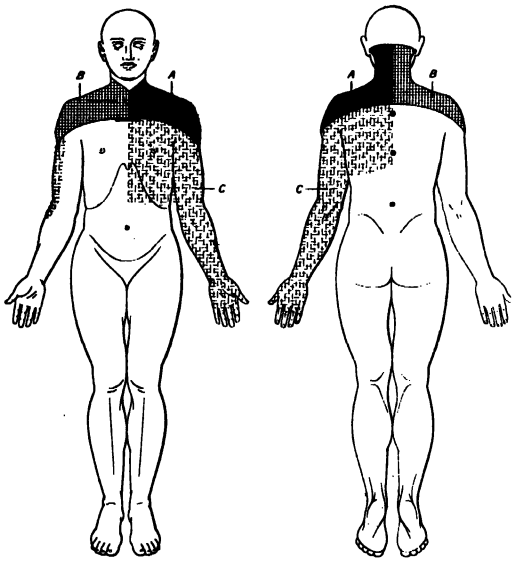


FIG. 105.—Two charts showing the sensory impairment in a case of intramedullary tumour of the cervical region. The black area (*A*) is the region of complete anæsthesia and corresponds to C 3 and 4 segments. *B* is an area of incomplete loss, and *C* a region of impaired thermal sensation.

The *motor* signs are segmental paralyses of an atrophic character, in which muscular atrophy is in excess of motor weakness.

Amongst other symptoms, the early onset of sphincter trouble, the early occurrence of an extensor plantar response, and in a general way signs of an extensive unilateral lesion, point to an intramedullary new growth.

With the extension of the growth signs of bilateral spastic paraplegia and sensory loss below the level of the lesion become apparent, and the local segmental motor and sensory

changes, which were at the outset confined to one side, become bilateral.

Diagnosis. The diagnosis has to be made mainly from syringomyelia. In this condition, as already described on p. 328, the disease runs a more prolonged course, the characteristic dissociation of sensation is well marked and is accompanied by muscular wasting. Painless whitlows, arthropathies, trophic disturbances, and kypho-scoliosis are also outstanding features of syringomyelia.

Prognosis. The disease progresses to complete paraplegia. It varies in duration, but lasts about two or three years in average cases. Death occurs from cystitis, bed-sores, or other complications.

Treatment. The treatment consists in the alleviation of symptoms as they arise, and the prevention of cystitis and bed-sores. A course of anti-syphilitic treatment may be prescribed in doubtful cases, and in some cases an exploratory laminectomy is justifiable.

4. HÆMATOMYELIA—'SPINAL APOPLEXY'

Hemorrhage into the spinal cord is rare as a primary condition. It may occur in consequence of trauma following a direct injury or a sudden jar of the vertebral column, such as results from a fall upon the feet or the lower part of the spine. It has also been observed to follow a severe exertion, such as lifting a heavy weight. We have also known it happen in consequence of diving into water from a height.

Secondary hemorrhage may take place into softened areas resulting from myelitis, or into tumours within the spinal marrow. In two cases personally examined, hemorrhage into the cord occurred in consequence of a tuberculous abscess outside the dura mater.

It is important also to note the occasional presence of spinal hemorrhage in syringomyelia, both in those cases which have presented the clinical signs of this disease, as well as in those in whom no clinical evidence of syringomyelia had been previously observed, but where an autopsy showed the existence of cavity formation. It is, therefore, not unlikely that in the cases of spinal hemorrhage attributed to exertion, the underlying cause is a pre-existing cavity. It has also

been seen in morbid blood states—such as pernicious anæmia. Spinal hemorrhage has a special tendency to affect the grey matter of the cervical and lumbar enlargements.

Symptoms. The onset is usually sudden, with a rapid development of symptoms to their maximal extent. In other cases the occurrence of spinal hemorrhage leads to a more ingravescent development of symptoms, whose maximum is not reached for a few hours. Consciousness is not lost.

FIG. 106.—Photograph showing the position of the arms in an old-standing case of hæmatomyelia of the cervical region.

Pain at the seat of the lesion, or referred to the affected root areas, is sometimes a transient symptom; and the temperature, normal at first, may be slightly raised for a few days.

The symptoms in a severe case are complete motor and sensory paralysis to the level of the lesion. The motor palsy is at first of a flaccid type, the deep reflexes are abolished, and there is retention of urine.

The first sign of improvement, which may occur in from one to three weeks, is a restitution of the tactile and pressure sensibilities, and the sense of position of the limbs. During this period the deep reflexes gradually return, the flaccid palsy by degrees gives place to spasticity, which is accompanied by

flexor or extensor spasm of the limbs, and an extensor plantar response.

Voluntary movement returns slowly, and may be first shown in flexion of the toes. The degree of motor power is impaired by the presence of spasm and the development of spastic contractures. The retention of urine gradually gives place to reflex incontinence, and constipation becomes marked. Trophic sores may develop during the first week, and frequently prove an obstinate complication.

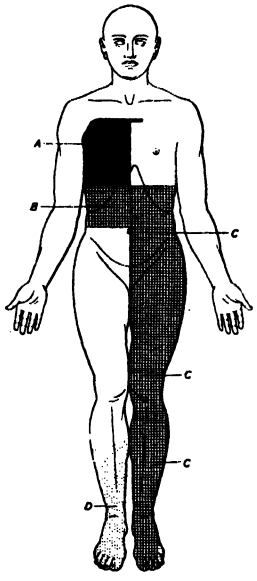


FIG. 107.—Chart showing the sensory loss in a case of unilateral hæmatomyelia. *A*, area of complete loss to all forms of sensation. *B*, region of painful and thermal anæsthesia. *C*, area of thermal and painful anæsthesia on the side opposite the lesion. *D*, impairment of the sense of passive movement of toes and foot.

In less severe cases the ultimate recovery may be nearly complete, the patient being able to walk with a spastic gait, but with good sphincter control, and perhaps some permanent loss of painful and thermal sensibilities below the level of the lesion.

Atrophic paralyses and sensory changes corresponding to the level of the lesion may become permanent.

In some cases the hemorrhage is limited to one-half of the spinal cord, and gives rise to the Brown-Séquard symptom, already described (p. 15 and Fig. 107).

The local segmental symptoms vary according to the region of the cord into which the hemorrhage has occurred. If in the lower cervical region, oculo-pupillary changes, atrophy of the intrinsic muscles of the hand, and sensory impairment along the ulnar border are observed.

If in the lumbo-sacral region, atrophic palsy of the lower limbs, loss of the deep reflexes, permanent sphincter trouble, and obstinate bed-sores are found.

Differential diagnosis. The differential diagnosis has to be made from :—

1. *Acute poliomyelitis*, by the absence of constitutional symptoms, the sudden onset following trauma or exertion, the well-marked sensory changes, and the localisation of the symptoms to a segmental level. We have seen one case in which a hemorrhage was strictly limited to the anterior horn on one side in the cervical enlargement, resulting in a flaccid palsy of the arm without sensory change.

2. *Acute myelitis*, by the suddenness of the onset, the absence of constitutional symptoms, a history of trauma, and the dissociation of sensation either in the earlier or the later stages.

FIG. 108. — Shows the 'preacher's hand' in case of hæmatomyelia.

3. *Meningeal hemorrhage*, by the lesser degree and extent of the pain, the more rapid and more complete paralysis, and the sensory dissociation. In traumatic cases the use of the X-rays may be of value as demonstrating a fracture-dislocation, and a lumbar puncture will show the presence of blood in the cerebro-spinal fluid.

Prognosis. Death is rarely the result of the primary lesion, but may ensue from septic absorption from bed-sores or cystitis and from pyelonephritis. In all but the most severe cases some degree of recovery may be anticipated. Atrophic paralysis from local destruction of nerve elements at the seat of the hemorrhage tends to remain permanent.

In forming a prognosis as to the recovery from the symptoms after lesion of the conducting tracts of the cord, the following points require consideration.

The points in favour of a satisfactory prognosis are :—

(*a*) The early return of sensation, although painful and thermal sensibilities may be permanently abolished.

(*b*) The early recovery from the flaccid palsy, with return of the deep reflexes.

(*c*) The healing of trophic sores within six weeks.

(*d*) The early return of voluntary control over the bladder.

(*e*) The return of voluntary power in the limbs within two or three months.

Of unfavourable omen are permanent loss of all forms of sensation, the retention of complete flaccidity and the development of severe spastic contractures, the continuance of bed-sores and incontinence of urine, and the absence of any voluntary power after three months.

Treatment. Rest is essential. The patient should preferably not be allowed to lie upon his back, but rather on his side upon a water-bed. The urine should be drawn off by a catheter. Great care should be taken to keep the patient dry, especially over the points of pressure.

Ergot has been recommended in the early stages.

After the acute stage has passed, passive movements and massage of the paralysed limbs may be given. Later on, the spasticity has to be combated by hot-air baths, followed by passive movements and massage, especially to the extensors. If there is a tendency to contracture extension may be applied, care being taken to avoid injury to the skin. For the atrophic paralyses, massage and galvano-faradism should be given.

If the reflex spasms are severe a combination of trional and phenacetin, or a mixture of the bromides and cannabis indica, may be prescribed.

5. CAISSON DISEASE AND DIVER'S PARALYSIS

This disease occurs in those who work under high atmospheric pressures, not during the period in which they are subjected to the pressure, but after their return to ordinary atmospheric conditions.

The factor necessary to its production is exposure to compressed air followed by a sudden diminution of atmospheric pressure. The longer the exposure and the more rapid the return to normal conditions, the more likely are nervous symptoms to ensue. It has also been noted that persons of temperate habits suffer less than those addicted to alcohol.

During the period when the worker is exposed to the high pressure, the blood becomes surcharged with gases, and if the

pressure is diminished too rapidly, bubbles of gas escape from the small blood-vessels into the nervous system and destroy the surrounding nerve tissues. Air emboli also may form in the capillaries and result in minute softenings. There has been much discussion as to the nature of the gases, some writers holding that nitrogen, others that oxygen and carbonic acid gas are the main constituents.

Pathology. A microscopic examination reveals changes in the dorsal region of the spinal cord, more especially in the white matter of the posterior and lateral columns. The changes consist of small fissures, surrounded by zones of parenchymatous myelitis, disintegration of myeline, and swelling of the axis cylinders. Small areas of localised softening may also be observed. The grey matter, nerve roots, and meninges are not affected. Hemorrhages are rare.

Symptoms. These vary considerably, both as regards intensity and character. In the main they are those of a spinal lesion, but in other cases well-marked cerebral symptoms may also be present.

The *cerebral* symptoms consist of headache, vertigo, throbbing, and deafness, often accompanied by nausea and vomiting. In some cases a transient coma occurs, more rarely monoplegic or hemiplegic symptoms persist.

The *spinal* symptoms are more general and affect chiefly the sensory system, but in severe cases the motor is also affected. In slight cases tingling, numbness in the trunk, legs, and arms, and sometimes severe pains referred to the joints are described. In association with the subjective sensory symptoms, complaint is made of heaviness and weakness of the legs. In severe cases the picture is that of a transverse lesion of the cord, with complete motor, sensory, and reflex paralyses.

On examination the objective sensory changes may be slight or wanting, but in more pronounced cases a diminution of all forms of sensibility, irregular and patchy in distribution, is observed.

In the motor system the weakness varies from slight paresis with increased tendon jerks to complete paralysis with loss of the deep reflexes. In other cases some inco-ordination of movements without much loss of power may be detected.

The **diagnosis** presents no difficulty, the symptoms coming on more or less rapidly, either immediately or within a short period after entering the normal atmosphere.

The **prognosis** is good, relatively few fatal cases being recorded. Recovery from the symptoms may be complete.

Treatment. This is mainly preventive, and consists in making the return from the highly compressed air as slow as possible. The active treatment consists in applying recompression, the patient being again put under increased pressures, which are gradually diminished. If the pain be severe, morphia may be necessary. Should permanent changes have occurred in the nervous system, the treatment is that of an acute myelitis.

Chapter III

DISEASES AND INJURIES GIVING RISE TO COMPRESSION OF THE SPINAL CORD

A well-recognised clinical picture is presented by cases in which the symptoms of progressive paraplegia are due to pressure upon the spinal cord, although both the mode of onset of the symptoms and their subsequent development and course may vary considerably. It is obvious that if the primary disease affects the structures surrounding the spinal cord before actually exerting pressure upon it, the initial symptoms may be vague and uncertain. An illustration of this is seen in the indefinite local pain and discomfort in the back in spinal caries and malignant disease of the vertebræ.

On the other hand, symptoms directly due to pressure upon the spinal cord, or its roots, tend to persist and to progress. Although, as a rule, the onset of symptoms is gradual, a more or less rapid development is not uncommon. In such cases the onset is not so rapid as in myelitis, and the early symptoms are less extensive. For example, in myelitis, paraplegia may develop within a few hours, and exhibit motor, sensory, and reflex changes below the level

of the lesion. In the compression cases, complaint may be made of sudden loss of power in one leg, with perhaps numbness in both legs; or a sudden pain is referred to a certain root area, with signs of motor weakness in the leg on the same side. In the majority of cases, however, a slow, progressively developing paraplegia is the rule.

If compression is exerted upon the anterior aspect of the cord, the primary symptoms are motor, and consist of spastic paraplegia below the level of compression, followed later by interference with sphincter control and the development of sensory phenomena. If, on the other hand, pressure is exerted on the posterior aspect, sensory symptoms (subjective and objective) accompany, and may precede, the motor symptoms. Frequently, however, the compressing body is situated laterally, and involves anterior or posterior roots. In such cases, if a posterior root, or roots, be involved, pain referred to that root, followed by anæsthesia over its distribution, is the first and most obtrusive symptom. This is followed by a motor weakness, most marked on the side of the affected posterior root, and sensory symptoms on both sides below the root level, but more intense on the opposite side. Symptoms due to pressure upon the anterior roots are of late onset, owing to the resistance offered by these roots to the effects of pressure, and to the absence of subjective signs over their distribution. In compression lesions of the cauda equina, atrophic muscular changes form early and important clinical signs.

The following are the conditions in which symptoms of compression paraplegia may be found:—

1. Tumours of the spinal membranes—
   (*a*) Extrathecal; and
   (*b*) Intrathecal.
2. Spinal caries—tuberculous disease of the vertebræ.
3. Malignant disease of the vertebral bodies.
4. Traumatic lesions of the vertebral column.

The symptoms of intramedullary tumours and of syringomyelia are elsewhere described (pp. 328 and 338).

1. TUMOURS OF THE SPINAL MEMBRANES

These tumours are divided primarily into two main groups:

(*a*) Those outside the theca spinalis—Extradural.

(*b*) Those inside the theca spinalis—Intradural.

They may arise in or grow from the nerve roots, the spinal membranes, the extradural connective tissues, the periosteum, or the bones of the vertebral column. They vary in character and position, and in the frequency of their occurrence in different localities.

The following table, founded upon the records of the Queen Square Hospital, shows the varieties of tumour found within the spinal canal and the frequency of their occurrence.

Tumour.	*Intradural.*	*Extradural.*	*Total.*
Sarcoma . .	2	5	7
Endothelioma .	5	1	6
Echinococcus (Hydatid)	0	5	5
Gumma . .	3	0	3
Fibro-myxosarcoma	3	1	4
Fibro-sarcoma .	4	0	4
Fibro-myxoma .	2	2	4
Cyst . . .	2	0	2
Myxoma . .	1	0	1
Psammoma . .	1	0	1
Fibroma . .	1	0	1
Angeioma . .	1	0	1
Cylindroma . .	1	0	1
Uncertain . .	2	0	2
	28	14	42

According to the table just given, the intradural tumours are twice as frequent as the extradural. The extradural sarcomata spring mainly from the periosteum of the vertebræ. The endotheliomata attached to the nerve roots and the membranes are chiefly intrathecal. Hydatids seem to spread from the deep muscular tissues of the back,

or the subpleural tissues, and enter the spinal canal by erosion of the laminæ. Other common forms of intradural growth are the slow-growing fibro- and myxo-sarcomata.

FIG. 109.

FIG. 110.

FIGS. 109 and 110 are photographs of extramedullary, but intrathecal, tumours pressing upon the spinal cord. Fig. 110 is a tumour involving more especially the cauda equina.

The following table shows the localities most favoured by extramedullary tumours and the frequency of their occurrence in a total of twenty-one operated cases :—

Locality.	*Intradural.*	*Extradural.*	*Total.*
Upper cervical . .	3	0	3
Cervico-dorsal . .	1	0	1
Dorsal . . .	2	7	9
Dorso-lumbar . .	2	1	3
Lumbo-sacral . .	1	2	3
Cauda equina . .	1	1	2
	10	11	21

Meningeal tumours may occur at any age, but they are rarely seen under ten or over fifty. The most common time for their occurrence is between the ages of thirty and fifty, during which period twelve out of twenty-one cases were observed and treated.

Males are slightly more affected than females, the proportion being twelve of the former to nine of the latter.

Symptoms. In the majority of cases of tumour pressing upon the spinal cord, *premonitory symptoms* are found prior to the onset of definite paralysis of the limbs. Pain in one form or another is the most common. Of the varieties described, pain and stiffness in the back, aching in the limbs, pain of a rheumatic character about the neck and shoulders, and occasionally sharp shooting pains in the leg or about the hip joints, may be mentioned. The position of pain is dependent upon the site of tumour within the spinal canal. In some cases the pain is of a dull, aching character, referred to the shoulder or the breast when the growth is in the upper dorsal region. In other cases the pain is definitely referred to the distribution of a particular root or roots, and may be accompanied by hyperæsthesia over the skin area supplied by the root.

We have found the intense, agonising, burning pains, described by some writers as characteristic of spinal tumour, to be extremely rare, such pains being present in only three out of twenty-one cases of meningeal tumour submitted to operation. The pain as above described is unassociated with any local tenderness along the nerves, but when referred to the spine may be accompanied by some tenderness on percussion, and may occasionally be increased on movements of the back or neck.

In other cases, even from the commencement, pain is entirely absent, the earliest symptom being weakness upon one side.

The premonitory symptoms may persist for an indefinite time before signs of compression of the cord make their appearance. This period may vary from a few weeks up to several years. During this period the cause of the pain is quite uncertain, and various diagnoses, such as rheumatism, intercostal neuralgia, lumbago, appendicitis, or hip-joint disease, may be made according to the seat of the pain.

Symptoms of compression of the cord, however, sooner or later make their appearance. They are seen in varying degrees of motor and sensory loss. The motor phenomena are usually those of spastic paralysis below the level of the lesion. Generally one limb, that on the side of the lesion, is affected before the other. Complaint is made of 'a giving way of the leg,' and on examination the distal portion is found more affected than the proximal. As time goes on both lower limbs become paralysed.

During the development of the motor paralysis the onset of two further symptoms is observed—flexor spasms of the legs and impairment of sphincter control. The former occur early, first in the limb homolateral to the tumour, and later in both limbs. The latter only occurs early, when the tumour presses upon the lumbo-sacral enlargement or its nerve roots. In tumours situated above the lumbar enlargement, the onset of sphincterweakness is synchronous with the development of bilateral motor weakness.

The symptoms of compression of the sensory fibres are both subjective and objective in character. The former consist mainly of numbness corresponding to the paralysed limb or limbs, and appear either with the onset of the paralysis or shortly afterwards. The objective sensory symptoms are late in onset and consist of impairment, or loss, of all forms of sensation below the level of the lesion. By this feature meningeal tumours may usually be distinguished from those within the spinal cord, in which dissociation of sensibility is an early and characteristic symptom. The delimitation of the extreme upper level of sensory change is one of the most important points in the examination of these cases, as by it the position of the tumour is definitely localised. In

lesions limited to the cauda equina the sensory loss presents a definite root distribution (p. 120).

The motor and sensory phenomena usually coincide in distribution; but not always, as one side of the cord is frequently more compressed than the other. Owing, therefore, to the greater unilateral compression, Brown-Séquard's 'symptom complex' may be observed, in which the motor palsy is more pronounced upon one side, and the sensory loss upon the other.

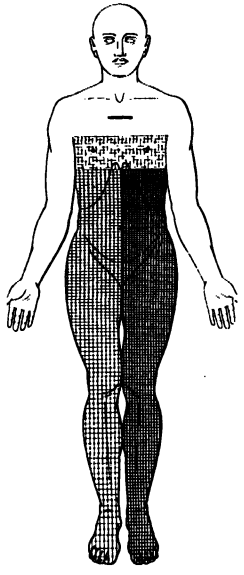


Fig. 111.—Chart showing the sensory impairme a case of tumor ressing upon the spin ord at the lev of th urth dorsal men Below the le tl ensory impair it ore marked o the left ide (p. 351).

Reflexes. The deep are exaggerated, the superficial abdominal are impaired or lost, and the plantar extensor in type. In the early stages these changes are confined to the side of the lesion.

In addition to the symptoms of compression of the cord, local symptoms due to compression of the nerve roots at the level of the lesion are found, viz. atrophic motor paralyses, hyperæsthesia or loss of sensation in the affected roots.

The spinal column. Prominence of the spines over a meningeal growth is rare, but is a common feature should the tumour originate in, or secondarily involve the vertebral bodies. Tenderness on percussion over the seat of the tumour is sometimes detected. There is rarely any limitation of movement, nor does movement usually give rise to local pain, except in cases of tumour high up in the cervical region.

Regional diagnosis. From the standpoint of symptomatology there are four chief localities in which tumours are found:—

1. Cervico-dorsal (lower cervical and upper dorsal) region.
2. Mid-dorsal region.
3. Lumbo-sacral region.
4. The cauda equina.

1. *Cervico-dorsal region.* At the level of the lesion motor weakness of the forearm and hand, either spastic in character from pressure upon the cord, or atrophic and flaccid from pressure upon the emerging roots, and symptoms of irritation or paralysis of the cervical sympathetic will be seen. Sensory impairment will be present along the ulnar side of the hand and arm as far up as a line joining the anterior and upper margins of the axillary folds.

Below the lesion paralysis with spasticity and impairment or loss of sensation are found. The deep reflexes are increased, the plantars are extensor, and the superficial abdominal diminished or lost. Sphincter impairment only occurs when motor weakness is bilateral. Priapism may occasionally be observed. These signs may not be equally pronounced on the two sides, more especially in the upper limbs, while below the lesion the motor and sensory impairment may approach the Brown-Séquard type of paralysis.

2. *The dorsal and upper lumbar regions.* The motor palsy is of the spastic paraplegic type, and the sensory loss corresponds to the level of the lesion. The reflexes below the lesion and the state of the sphincters are as described above.

3. *The lumbo-sacral region.* Here a meningeal tumour is not so likely to involve several roots as one situated within the theca. The symptoms, therefore, are those of compression paraplegia with increase in the deep reflexes of the lower limbs, and extensor plantar responses with sphincter impairment. The abdominal and epigastric reflexes are retained.

4. *The cauda equina.* For an account of the symptoms of lesion of the cauda equina, the reader is referred to p. 120.

Diagnosis. The following points would favour the diagnosis of a tumour pressing upon the spinal cord: —

(*a*) A history of pain, sometimes of a dull, aching character, at other times sharp and shooting in the distribution of particular nerve roots, for an indefinite period preceding the onset of paraplegia.

(*b*) A progressive paraplegia, usually affecting one lower limb before the other, or in cervical cases the arm and leg upon the side on which root symptoms were first present.

(*c*) Motor and sensory paralysis below a fixed segmental

level, all forms of sensation being impaired or lost, but with a tendency to the Brown-Séquard distribution.

(*d*) Absence of sphincter weakness, or only slight impairment, until the paraplegia is well developed.

(*e*) The absence of signs of caries of the spine, or of malignant disease of the vertebræ.

The *diagnosis between an extrathecal and an intrathecal tumour* is more of academic than practical interest, and in many cases is impossible, but the following are the chief indications which help in the diagnosis. If intrathecal, the tumour usually gives rise to root symptoms early, and these are followed more or less rapidly by symptoms of compression of one side of the spinal cord, some time elapsing before the establishment of signs of compression of the whole cord.

In extrathecal cases the compression of the cord takes place more gradually, as the spinal cord may be displaced before being compressed. When compression occurs, the symptoms are those of compression of the whole cord, without any definite stage in which the symptoms point to compression only of one side.

It is in cases of tumour of the lumbo-sacral region that the difference is most marked. Here an intrathecal tumour presses on several nerve roots as they pass to their point of emergence. Hence the symptomatology is that of a root lesion affecting the higher lumbar areas, viz. atrophic paralysis with root anæsthesia and signs of compression paralysis in the parts below. Thus there are loss of the knee jerks, atrophy of the thigh muscles with increase of the ankle jerks, and extensor responses and sphincter impairment. The sensory loss is that of a root lesion in the upper lumbar areas with compression anæsthesia below this level.

The differential diagnosis has to be made from

1. *Intramedullary tumour*, by the absence of root pains, the presence of muscular atrophy, and the dissociation of tactile from painful and thermal sensibility.

2. *Disease of the bones of the spine* (tuberculous and malignant).

3. *Aneurism of the aorta*, eroding the vertebral bodies and compressing the spinal cord.

Treatment. As soon as the diagnosis of meningeal tumour has been established, operation for its removal ought

to be undertaken. The spinal segments corresponding to the upper limit of the sensory impairment should be exposed, and the upper and lower limits of the tumour laid bare. Should no tumour be found, and the cord not pulsate, the laminæ higher up ought to be removed.

Pulsation in the spinal cord after removal of the tumour is a favourable prognostic sign.

Recovery after operation for removal of the growth may be interfered with by (*a*) the malignant character of the growth and the impossibility of its complete extirpation; (*b*) irreparable destruction of the spinal cord, owing to excessive and long-continued pressure; and (*c*) the formation of adhesions and cicatricial bands.

2. CARIES OF THE SPINE

Tuberculous disease of the spine may occur at any age; although more common in children, it is not at all infrequent during adult life. In most cases it is associated with tubercle elsewhere in the bones, joints, lungs, or lymphatic glands.

In a small number of cases it develops apparently as a primary condition.

The onset is usually insidious and without a definite exciting cause, but cases are met with in which the first symptoms have been noticed after an injury or strain to the spine.

Pathology. The original pathological lesion is a tuberculous osteomyelitis or periostitis. This commences either within or on the anterior, superior, or posterior surface of the body of one or more of the spinal vertebræ, most commonly of the dorsal region, and leads to the formation of a soft caseous tuberculous focus within the vertebral body. In consequence, a collapse of the affected bone occurs and the corresponding spinous process becomes prominent. The adjacent vertebræ, above and below, are therefore approximated towards each other, and their spines take part in the formation of the antero-posterior deformity, which is characteristic of this condition. The tuberculous process may, if small and localised, undergo calcareous degeneration; in other cases

the body of the vertebra may disintegrate and be partly absorbed with the formation of granulation tissue; in others, abscess formation ensues, and, more rarely, a sequestrum of bone is left with the development of a sinus. The condition known as 'caries sicca' is brought about by the absorption of the granulation tissue, leaving only carious bone. The reparative process consists of ankylosis of adjacent vertebræ.

The spinal cord may be involved in several ways, of which the following may be mentioned:—

1. Cases in which there is no obvious deformity of the spine or local tenderness on pressure, but in which the spinal marrow is compressed by a small abscess, or, more commonly, granulation tissue extending into the neural canal. In these cases the symptoms are more motor than sensory.

2. Cases in which there is no abscess within the neural canal, but, owing to the collapse of the vertebral bodies, an angular curvature is produced. This may result in the stretching and consequent compression of the spinal cord over the projecting posterior surface of the vertebral body.

3. Cases in which a combination of tuberculous tissue—abscess or granulation tissue—and deformity causes pressure upon the spinal cord. In both these groups the symptoms are largely motor in excess of sensory.

It is more probable, however, that the effect upon the spinal marrow is an indirect one, and is brought about through circulatory disturbances, arising from the pressure of tuberculous matter and thickening of the dura mater, upon the lymphatics and blood-vessels of the cord in the immediate neighbourhood of the lesion. In consequence of this a localised œdema of the cord is induced (Schmaus).

It is probable also that a state of local evascularisation, or ischæmic necrosis, may be the immediate effect of the obstruction to the blood supply of the spinal cord, and the cause of the permanent paralysis in old-standing cases of the disease.

In these cases extensive thickening of the dura mater, and adhesion between it, the pia arachnoid and the spinal cord, are observed. This pachymeningitis is not necessarily limited to the anterior surface of the cord, but is found all round it, compressing it and interfering with the vascular supply.

The microscopical appearances of the cord at the seat of

the compression are slight—the axis cylinders are swollen, and the medullated sheaths are dilated and stain badly. In some cases there are definite degenerative changes, both of the nerve fibres and cells. The white matter is more affected than the grey.

In old-standing cases secondary ascending and descending degeneration of the long fibre tracts is observed; but in many cases the secondary degeneration is slight and the axis cylinders are strikingly preserved.

Symptoms. The disease being primarily one of the bones of the spine, affecting secondarily the nerve roots and the spinal cord, the symptoms may be conveniently described according as they indicate lesion of the bone, the nerve roots, or the spinal cord.

1. *Bone symptoms.* The earliest signs of spinal caries are rigidity of the muscles on either side of the affected vertebræ and a lack of mobility in the vertebral articulations. In consequence of this a characteristic attitude is induced, which is seen in the care with which the child bends the back, as in stooping, or in other actions requiring movement of the spinal column. Pain, either spontaneous or on movement of the back, and referred to a particular region of the spine, is a later symptom. It is frequently present before any sign of vertebral prominence makes its appearance.

Percussion of the spine evokes tenderness over one or two vertebræ, or pressing the head or shoulders forcibly downwards will call forth expressions of pain in the affected area.

Later on, a local prominence or irregularity of the spinous processes, or a definite antero-lateral curvature, will indicate the nature of the disease and its position.

2. *Root symptoms.* Pains are rare in caries, but are in some cases an early symptom, and may be present before the symptoms of the bone disease are apparent. When present they are described either as dull and constant or as sharp and radiating pains referred to the root areas affected by the disease. In cervical caries headache and pain referred to the arms are often associated with sympathetic pupillary changes. In dorsal caries pain is referred to the sternum or round the body. In dorso-lumbar caries the pain is abdominal or of a girdle character. In lumbar and lumbo-sacral caries the pain is referred to the legs and genitalia.

3. *Cord symptoms.* Interference with the functions of the spinal cord is usually subsequent to the pain in the back, already described, but has been known to be the earliest indication of the disease, especially in the so-called traumatic cases, where, owing to an injury to the spine, paraplegic symptoms have suddenly developed. The onset of paraplegia is, on the other hand, usually insidious; and owing to the

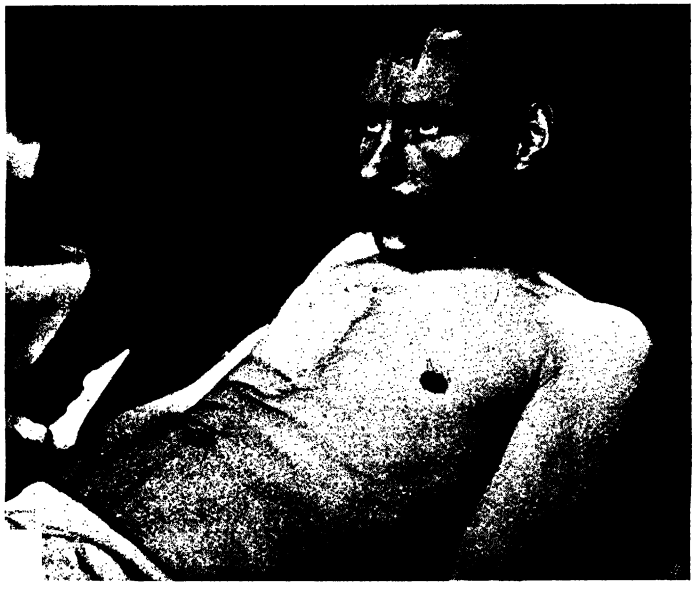


FIG. 112.—Photograph of a case of caries of the spine, with compression of the cord at the tenth dorsal segment, showing drawing upon the umbilicus by the non-paralysed portion of the recti abdominales.

pressure being primarily upon the anterior or antero-lateral aspects of the cord, motor paralysis alone, or motor weakness in excess of sensory impairment, is the first sign of pressure. If the abscess presses upon one side of the cord, the earliest symptoms are slight spastic paresis on the homolateral side, with slight, but definite, sensory impairment on the contra-lateral side. Later on, complete motor and sensory paralysis will be found below the affected segment in severe cases.

The paraplegia is of the spastic type, with exaggeration of the deep reflexes, abolition of the abdominal skin reflexes, and extensor plantar responses.

Loss of sphincter control may not ensue until a late stage, unless the caries be sacral in position, when incontinence forms one of the earliest of the pressure effects.

As already explained, when describing the localisation of function within the spinal cord, in addition to the motor and sensory symptoms below the level of compression, symptoms referred to the affected segment or segments may also be observed. These differ according to the portion of the cord affected. It will therefore be convenient to refer to them briefly in this place, mentioning at the same time certain other local symptoms of importance and diagnostic value.

Symptoms of vertebral caries in special localities

Occipito-atlo-axoid caries. The head and neck are held stiff, and pain is complained of locally, or shoots over the occiput on to the vertex. The antero-posterior and the lateral rotation movements of the head are impaired. Difficulty in swallowing may be present from the existence of a retro-pharyngeal abscess, or from prominence of the vertebral bodies.

Atrophic paralysis of the muscles supplied by the spinal accessory nerve, and sometimes of the tongue, have been described.

Caries of the upper cervical region. In addition to the motor adn sensory palsy below the level of the lesion, palsy of the diaphragm from involvement of the phrenic nerve may be observed. Pain in the neck and impairment in its movements are also present.

Cervico-dorsal caries is characterised by paralysis and wasting of certain of the arm, shoulder, and hand muscles, in addition to spastic paraplegia. The atrophic paralysis and sensory loss in the arms will depend upon the segments affected as given in the table upon p. 318 and in the figures 90 and 91.

In *lumbar caries* the fibres forming the cauda equina are in part affected, and the symptoms are those of lesion of that structure—atrophic palsy and segmental loss of sensation corresponding to the affected nerve roots (p. 318). An abscess may point locally in the back, or pus may pass along the ilio-psoas sheath and point below Poupart's ligament.

Abscess formation. In all cases of vertebral caries, the possible presence of pus should be borne in mind. The abscess may either point locally, or by passing along certain lines of least resistance appear at a considerable distance from the disease. A retro-pharyngeal abscess forms in connexion with caries of the upper cervical region; pus may invade the posterior mediastinum from caries of the lower cervical or upper dorsal regions, and a psoas abscess may point in the groin from lower dorsal or lumbar caries. Abscess is most common in association with lumbar caries.

Symptoms of tubercle elsewhere in the body should always be sought for, both in the early stages and during the prolonged treatment which these cases require. The temperature may or may not show an evening rise. Where the disease is active, general malaise, cough, and an evening rise of temperature may be present.

Diagnosis. The diagnosis of spinal caries presents no difficulty in the majority of cases. The stiffness and rigidity of the back, local tenderness on pressure, root pains, and symptoms of compression paraplegia with irregularity of the spinous processes and angular deformity are usually sufficient on which to base the diagnosis, especially when taken along with the general symptoms.

Examination by the Röntgen rays should be carried out in all cases where possible.

The malady has to be distinguished from —

1. *Hysteria.* In this condition the absence of angular deformity or of signs of organic disease, a more diffuse and extensive spinal tenderness elicited by light pressure, or not obtained by deep pressure when the patient's attention is distracted, and the presence of spinal mobility on pressure over a tender area are usually sufficient to distinguish the malady.

2. *Malignant disease of the vertebræ.* The special features of this condition are the age of the patient, the probable history of an earlier operation for tumour, the presence of malignant disease elsewhere, the diffuse character of the pain, often worse when lying recumbent, the presence of lumps on the bones, and the general cachectic condition.

Prognosis. The course of the disease is prolonged. In some cases vertebral disease may commence in childhood and

give rise to curvature, while paraplegic symptoms only ensue in adult life.

In other cases the bone disease and the secondary spinal symptoms develop within a short period of each other, or may even come on simultaneously.

In the great majority of cases, after the development of compression paraplegia, the further course of the malady is prolonged. In some, recovery results. In others, permanent paralysis results with the formation of bed-sores and cystitis; or general tuberculosis and pyæmia cause death.

Rarer complications of the disease are occasionally found in hemorrhage within the spinal cord and the formation of a central abscess (p. 326).

The prognosis is much more favourable in children than in adults.

Treatment. In all cases the general treatment of tuberculosis, in addition to the special treatment of the local condition, should be prescribed. This consists primarily in securing good hygienic conditions such as prevail in open-air sanatoria, with ample nourishment, tonics, and cod-liver oil.

Rest to the spine. Rest in the recumbent posture is essential in most cases. This may be rendered more satisfactory by the addition of extension or mechanical supports to the spine. In cervical caries the neck may be fixed by means of a collar, or by the use of sand-bags placed on each side of the head. In dorsal caries double extension may be adopted, the patient, however, being permitted the use of his arms. Rest should be persevered with for twelve months, or longer if necessary. In children this will usually suffice to relieve the cord of pressure, and procure the healing of the tuberculous lesion. In adults the results are not so favourable; but the method should be given a fair trial.

Every care should be taken to prevent the development of bed-sores, and attention should be given to the bladder. In cases with reflex spasms of the legs, the bromides, cannabis indica, phenacetin, or veronal may be prescribed.

Abscesses should be opened and washed out on the lines mentioned in the surgical text-books.

Should recovery occur, or when the recumbent posture is no longer necessary, a support may be supplied to strengthen the spine and to remove the weight of the body from the

affected part. In cervical caries a collar may be fitted; and in lower dorsal or lumbar caries, a spinal jacket fitted with arm supports resting upon the pelvis may be prescribed. By these means both the local and general condition of the patient will be improved.

The question of *laminectomy* in these cases is one of the most difficult upon which to give an opinion. It is indicated—

(*a*) In cases where the focus of the disease is pressing upon the upper cervical region and threatening life.

(*b*) In cases where, with a definite focus, the spasticity of the limbs is passing into that of flaccidity.

(*c*) In cases where there is persistent pain, associated with sensory disturbance, as in sacral caries.

(*d*) In those cases in which there is a single focus of disease, in which symptoms do not resolve with rest, and in which spastic contractures are steadily increasing.

Laminectomy is contra-indicated when there are signs of general tuberculosis, or of two or more foci of pressure upon the cord. In both these types of case operative interference, if not immediately fatal, may result in the lighting up of tuberculous meningitis. In cases complicated by lumbar or psoas abscess, this should be opened and drained.

In successful cases a more or less rapid recovery of the pressure symptoms after operation takes place. This, however, should not interfere with the carrying out of prolonged rest and open-air treatment. If the patient remains free from symptoms for two years, he may be permitted to move about a little, a suitable apparatus being supplied for the support and fixation of the spine. Special care should be taken to avoid jars, concussion, or strain upon the back. The condition of the limbs should be attended to by means of massage and passive movements.

3. MALIGNANT DISEASE OF THE VERTEBRÆ

Both benign and malignant tumours may affect the vertebral column, but the latter are the more frequent and important. Carcinoma and sarcoma are the commonest of the malignant tumours. Carcinoma is invariably secondary to cancer elsewhere, chiefly of the breast; sarcoma is usually primary, either of the vertebral bodies, their periosteum, or

the adjacent soft parts. These tumours invade the structures adjacent to their site of origin, in many cases growing into the spinal canal, and eventually pressing upon the nerve roots in the intervertebral foramina, the dura mater, and the spinal cord. The dorsal region of the spinal column is that chiefly involved in malignant disease.

Symptoms. These may be described according as they are referred to the bones of the spine, the nerve roots, and the spinal cord.

(*a*) *The bones.* Malignant disease of the bones is characterised by local pain and tenderness on pressure, by interference with the movements of the spine, arising partly from stiffness, but partly due to the fear of causing pain by movement; and by deformity, or kyphosis, of that portion of the spine in which the growth is situated.

The local pain is sometimes arrested in the recumbent posture, but readily comes on in the erect position and on movement. An eminently characteristic feature of the pain of malignant disease of the spine is a tendency to remission, sometimes for long periods, under conditions of rest.

(*b*) *Nerve roots.* These structures are affected either by pressure or from direct implication in the new growth. Root pains are of an intensely excruciating character, and radiate along the distribution of the affected root or roots. Although induced by movement, they are often entirely spontaneous in origin. They are usually unilateral, and may be associated with a definite zone of hyperæsthesia over the area of distribution of the affected nerve. If the nerve root is destroyed, a corresponding zone of anæsthesia may be detected.

In a number of cases of malignant spinal disease, the symptoms throughout the whole course of the malady are referred solely to the bodies and the nerve roots. In others, however, symptoms of pressure upon the spinal cord develop at an early stage.

(*c*) *Spinal cord.* The onset of cord symptoms is usually rapid. They consist of motor, sensory, and reflex phenomena below the level of compression.

The course and duration of these cases is variable. The interval which may elapse between the primary disease elsewhere and the appearance of symptoms of secondary

metastatic growths in the spinal column is uncertain, being in one case as long as seven years. Once the spine becomes affected the symptoms progress rapidly, but life may be prolonged for twelve or eighteen months. With the onset of paraplegic symptoms, and the development of cystitis and bed-sores, the duration of life is short, on an average of about two months.

The only **treatment** is palliative, as surgical interference is out of the question. Hypodermic injections of morphia are the best means at our disposal, but aspirin and phenacetin have proved efficacious in some cases. Complete rest in bed is essential.

4. TRAUMATIC LESIONS OF THE SPINAL COLUMN INVOLVING THE SPINAL CORD

Traumatic lesions of the spine are of several kinds. The following are the most important:—

1. *Extradural hemorrhage.* This is a complication of rare occurrence, resulting from severe strain or stress applied to the spine. The symptoms depend upon the amount of hemorrhage and the degree of pressure which the extravasated blood may exert upon the spinal cord or the emerging roots. There is a tendency for the blood to gravitate towards the lower end of the neural canal, in consequence of which the symptoms, of which pain is the chief, are mainly referred to the lumbo-sacral roots.

2. *Fracture of the spine* This may be limited to the spinous processes, the laminæ, or the transverse processes, and does not necessarily give rise to any pressure symptoms upon the spinal cord.

3. *Dislocation of the spine.* This results from subluxation of a vertebral disc, or of a vertebral body nipping the cord, and then recoiling into position.

4. *Fracture-dislocation* is a condition characterised by the permanent displacement of a vertebral body. The symptoms may arise from hemorrhage into the substance of the cord, from bone being driven into the cord, from crushing of the cord, or from a combination of these conditions.

A solution of the physiological continuity of the cord may

occur in cases in which instantaneous momentary nipping of the cord has occurred.

Incomplete lesions of the cord may result from intramedullary hemorrhage or bruising, or from extramedullary hemorrhage giving rise to compression, which occurs not immediately at the time of injury, but later during the period of recovery from shock.

The cord also may suffer from ischæmia, the result of being stretched over, or pressed upon, a displaced vertebral body or fragment of bone.

Symptoms. (*a*) *Complete anatomical severance of the cord.* This injury is followed by severe shock and collapse, and is often attended by unconsciousness. The temperature may rise to hyperpyrexia. Complete motor and sensory paralysis is found below the level of the lesion, with abolition of all the reflexes and retention of urine. At the upper limit of the lesion a zone of hyperæsthesia may be found. In a certain number of cases sugar is found in the urine. Trophic changes are especially liable to occur.

Death may occur during the period of collapse, or may follow in from one to three weeks from septic absorption from bed-sores or cystitis.

(*b*) *Incomplete anatomical, but complete physiological severance of the cord.* The immediate effects are similar to those seen in the complete cases. Should the patient recover from the effects of the injury, improvement is first seen in a return of the reflexes, the plantar returning early and of the extensor type, and followed by the restoration of the deep reflexes. After a few days, retention of urine gives place to reflex incontinence.

In upper dorsal and cervical lesions priapism may be a troublesome complication, and in cervical cases optic neuritis has been observed as a rare phenomenon.

The after-stages are characterised by the resolution of the trophic lesions, the healing of bed-sores, the onset of spasticity and reflex spasms in the paralysed limbs, which may have shown rapid wasting. The sensory symptoms also become less marked—the hyperæsthesia lessens and the sensory loss diminishes. Voluntary power returns slowly, commencing in the distal portions of the limbs.

(*c*) *Incomplete lesions with secondary hemorrhage.* In

slighter cases, a partial motor and sensory paralysis below the level of the lesion may be detected. In these cases it is important to observe the subsequent state of the patient, as in some instances more serious damage takes place later from the occurrence of hemorrhage with secondary pressure symptoms. The importance of this bears upon treatment, as in any case in which the reflexes, present after the accident, subsequently become lessened or abolished, and the motor and sensory symptoms increase, surgical interference should be at once adopted, as the recent symptoms are probably due to hemorrhage.

Symptoms pointing to an extramedullary hemorrhage are: an increase in the zone of hyperæsthesia, intensification of the local segmental symptoms, augmentation of the motor paralysis and sensory loss below the lesion, and a disappearance of the deep reflexes, which may have been increased during the initial stage subsequent to the collapse.

The symptoms of an increasing intramedullary lesion are the development of marked paralysis of the Brown-Séquard type, of dissociation of sensation, and of gradually increasing paraplegia.

(*d*) *Sypmtoms referred to a local ischæmic condition of the cord.* The early symptoms, which may have been slight, resolve to a greater or less extent; but after a few weeks, progress is arrested and an increase of the paraplegic symptoms takes place, especially motor weakness associated with increasing spasticity and reflex spasm. The important feature of this condition is the increase of spasticity and spasm, accompanied by increasing motor weakness—an indication of retrogressive action. It ought to be distinguished from an increase of spasticity associated with the increase of voluntary power—a sign of resolution and progress. Increasing weakness, therefore, indicates pressure upon the spinal cord, either from the effects of organising and contracting blood-clot, or from the long-sustained stretching of the cord over a bony prominence.

The condition closely resembles that seen in certain cases of Pott's disease of the vertebræ, in dry caries, in some meningeal lesions, and in cases of slow compression from any other cause.

The condition of the spinal column in cases of fracture-

dislocation varies. Fracture of the spines and laminæ can usually be detected by palpation, but in all cases it is advisable to make use of the X-rays, which often afford direct indications for the immediate treatment. It is important to bear in mind that there is no constant relation between the severity of the vertebral lesion and the damage to the spinal cord.

Prognosis. The complete cases end fatally, within six weeks to three months, except in those high up in the cervical region, when death may be instantaneous or occur in a few hours.

In the incomplete cases, recovery depends upon the damage to the cord and the early relief of pressure symptoms. On the one hand, recovery may be complete, or may be to the extent of a spastic paralysis, the patient being able to walk even without sticks. On the other hand, a large number of cases improve sufficiently to be able to get about with the aid of sticks or crutches. The remainder become bedridden.

The ultimate result depends especially upon the nursing and management, the prevention of septic infection from bed-sores and cystitis, the maintenance of nutrition, the prevention of contractures, and the conservation and fostering of voluntary motor power. It is important to remember that recovery may not take place until some years have elapsed after the injury.

Treatment. This depends in large measure upon the diagnosis and the appreciation of the nature and situation of the local injury. During the stage of collapse nothing ought to be done, unless the situation of the lesion is immediately dangerous to life, as in high cervical injuries, where paralysis of the phrenic nerve calls for urgent relief. After the stage of collapse in cases of severe injury, appropriate treatment such as removal of fragments of bone should be adopted. In cases of less obvious injury, where an X-ray examination may reveal fracture or dislocation, or in the cervical region blood-clot within the neural canal, operation should be carried out at once. If, on the other hand, the nervous symptoms are resolving satisfactorily, interference may be postponed, with a view to an improvement in the patient's condition. In all cases where an increase in the nervous symptoms occurs, or severe local root pains are present,

operation should be performed at once, as delay may rob the patient of any chance of complete recovery.

There remains the not inconsiderable number of cases in which no definite local evidence of fracture or dislocation is present, and in which the paraplegic symptoms are characteristic more of an intra- than an extramedullary lesion. In these cases it is wise to watch the progress of the case. In intramedullary lesions the symptoms tend to concentrate themselves and to become characteristic (Brown-Séquard's symptom and dissociation of sensibility). In extramedullary cases the tendency is towards augmentation or diffusion of symptoms, increasing root pains, and bilateral motor paralysis and sensory loss.

In all cases the patient should be placed on a water-bed, and special care taken to prevent bedsores, which are prone to occur. The catheter should be used, a low diet prescribed, and contractures minimised or prevented by massage, passive movements, and extension.

Part IX

GENERAL DISEASES OF THE NERVOUS SYSTEM

Chapter I

SYPHILITIC DISEASES

Syphilis may affect the nervous system in two ways:—

First, it causes a specific inflammatory affection of the blood-vessels and membranes of the brain and spinal cord. These changes show all the histological features exhibited by syphilis elsewhere, and bring about alterations in the nervous tissues, either by diminishing or arresting the blood supply through vascular occlusion, or by the direct effect of the pressure of new formations (gummata) upon the nervous elements—syphilitic lesions.

Secondly, it exerts a specific action upon the vital energy of various component parts of the nervous system. Although this action, as far as our present knowledge goes, is not fully understood, it would appear to be the result of a toxic agent produced within the system. The histological changes found in these cases are distinct from those of true syphilitic lesions, and consist of a primary degeneration of various cells and fibre tracts of the nervous system—parasyphilitic lesions.

Symptoms of syphilitic lesions of the nervous system may be observed at any period after infection. According to Mott there is no period at which syphilitic brain disease may not occur, but the most intractable to treatment and the most severe and generalised forms occur within two or three years after infection.

Table A shows the time of onset of symptoms after infection in 34 cases of cerebro-spinal syphilis.

Years after infection	1—2	2—4	4—5	6—8	8—10	over 10
No. of cases	7	4	3	3	3	14

In contrast to this, Table B shows the time of onset of symptoms after infection in 65 cases of tabes dorsalis.

Years after infection	1—5	5—10	10—15	15—20	over 20
No. of cases	2	8	17	23	15

The general statement may be made that true syphilitic lesions occur early, and the degenerative or parasyphilitic develop much later.

CEREBRO-SPINAL SYPHILITIC LESIONS

Etiology. In the majority of cases no immediate cause is present. In a few cases, however, an injury may favour the development of a local syphilitic lesion.

Varieties. Cerebro-spinal syphilitic lesions are of the following kinds:—

1. Vascular lesions.
2. Syphilitic meningitis—(*a*) leptomeningitis;
   (*b*) pachymeningitis.
3. Gummata.
4. Diffuse lesions—gummata, endarteritis, meningitis.

1. Vascular lesions. The vessels most commonly affected are those of the circle of Willis and the Sylvian fossa, the basilar and the lenticulo-striate arteries. The characteristic structural change consists of a proliferation of the intima, with obliteration or narrowing of the lumen. Similar changes are also seen in the middle and adventitial coats. The elastic lamina may become broken up and separated by proliferated cells. In some cases small gummata form in the vessel wall. Perivascular cellular infiltration occurs in most cases.

The effect of these vascular changes depends upon their degree and situation in the affected blood-vessel. When the lesion is localised, a thrombus may form, and give rise to softening in the distribution of the affected artery. Not infrequently, before an actual occlusion takes place, symptoms pointing to a transient functional disturbance permit of its recognition and the employment of active antisyphilitic treatment.

The fact that syphilis is the predominant cause of vascular disease in early life should always be remembered, and in any doubtful case a cytological examination of the cerebro-spinal fluid should be made.

The symptoms arising from this lesion may be referred to any part of the central nervous system.

2. Syphilitic meningitis. (*a*) Leptomeningitis. Syphilitic disease of the pia-arachnoid membrane, though frequent, is often overlooked. It may involve the cerebral or spinal membranes.

In the *brain* leptomeningitis causes an opaque induration of the membrane, often with small nodules resembling tuberculous meningitis, and invariably associated with syphilitic changes in the superficial blood-vessels. It is also found associated with more advanced changes—gummata, gummatous meningitis or vascular lesions, such conditions giving rise to epilepsy, or to Jacksonian convulsions.

The symptoms are headache, especially at night, and irritative phenomena corresponding to the locality of the lesion. If over the occipital lobe, visual spectra in the opposite half-fields are described; if over the motor region, Jacksonian attacks in the opposite limbs. Paralytic symptoms succeed the irritative, and result from the destruction of the nerve elements of the underlying cortex, by extension inwards of the gummatous process.

In the *spinal cord* its presence as a definite pathological factor, capable of giving rise to characteristic clinical symptoms, has not been fully appreciated. Certain cases of slowly progressive paraplegia with sensory disturbances to a definite segmental level, in which the diagnosis of compression paraplegia due to a tumour has been made, have been found on operation to arise from a constriction of the cord, with damming up of the cerebro-spinal fluid from a local lepto-

meningitis. That such, and not a secondary intramedullary lesion, was the cause is probable from the fact that recovery has resulted from the division and freeing of the adhesions.[1] In this connexion it is right to mention that the association of leptomeningitis with syphilitic endarteritis and spinal thrombosis is well recognised as a common cause of myelitis.

(*b*) Pachymeningitis. This is an inflammatory affection of the dura mater. It is the commonest variety of syphilitic meningitis. It may be general or circumscribed.

In the *brain* it is most frequent over the base, especially in the interpeduncular space, when it gives rise to headache, drowsiness, vomiting, and various cranial nerve affections—paralysis of the third, sixth, and optic nerves. If on the convexity, it may be primarily dural, or secondary to an extension from the bones of the skull. If circumscribed, it is not infrequently located in the region of an old injury.

In the *spinal cord* pachymeningitis affects especially the cervical and lumbo-sacral regions and the cauda equina. Clinically, this condition is characterised by severe root pains, atrophic motor paralyses, and segmental anæsthesias, corresponding to the affected roots, and when there is compression of the cord, spastic paraplegia and sensory impairment below the level of the lesion.

3. Gummata. A single gumma is rare, and only arises from the direct extension of a circumscribed gummatous pachymeningitis into the brain substance. Multiple gummata are common, and invariably arise from the pia arachnoid, or the adventitia of a cortical blood-vessel. The changes in the cerebral tissue in the neighbourhood of a gumma consist of a perivascular infiltration, an increase of the connective tissue of the pial processes, degeneration of the nerve elements, and secondary proliferation of the neuroglia with numerous spider or Deiters's cells.

Symptoms. The symptoms of single large gummata are those of intracranial tumour. When multiple, the symptoms are diffuse with headache and profound mental change. Convulsions, local or general, are present in the early stages, and are later replaced by motor and sensory paralysis.

[1] Horsley (Sir Victor), *Brit. Med. Journal*, 1907.

4. Diffuse syphilitic lesions—cerebro-spinal syphilis. In the severer forms of cerebral syphilis two or more of the above described pathological changes are combined. Basal meningitis is common, and its symptoms may either precede or follow evidence of vascular occlusion. The occurrence of fits of a general epileptic character may be ascribed to a diffuse syphilitic infection, or to an increase of intracranial pressure in association with a basal meningitis. Jacksonian attacks, or localised focal seizures, arise from a circumscribed gummatous patch.

In many cases the symptoms point to a limitation of the lesion to the intracranial contents, but not infrequently these are combined with signs of spinal meningitis. On the other hand, a small number of cases commence with spinal symptoms, which advance to a considerable severity prior to the onset of symptoms indicating intracranial disease.

The *clinical features* of the diffuse cerebral type, a condition of importance owing to its close resemblance to general paralysis, are the following: The early appearance of cranial nerve paralyses, especially of the third and sixth nerves, characteristic pupillary changes (irregularity, inequality, and impaired light-reaction), the occurrence of general or localised epileptic seizures, often followed by paralysis with definite signs of organic disease. The general symptoms of intracranial tumour (headache, vomiting, and optic neuritis) may be superadded, and marked mental impairment is present from an early stage. This form of cerebral syphilis is distinguished from progressive general paralysis of the insane by the absence of the peculiar mental state of general paralysis, the early evidence of gross intracranial disease, as shown by the paralysis of the cranial nerves and alterations in the reflexes, the absence of tremor and of slurring speech, and the favourable response to energetic antisyphilitic treatment.

Further, there is in diffuse cerebro-spinal syphilis a lesser degree of lymphocytosis in the cerebro-spinal fluid, and the Wasserman reaction is either feeble or not obtained (p. 406).

Prognosis. The prognosis of these cerebro-spinal syphilitic affections is variable, much depending upon the amount of destruction of the nerve elements which has taken

place. The most unsatisfactory type of the malady is that associated with multiple lesions (syphilitic dementia), especially when it occurs within a few years of infection. The vascular lesions are not unfavourable apart from the permanent destructive effects upon the nerve tissue. The basal lesions affecting the cranial nerves, especially if of limited extent, are relatively favourable when submitted to early and thorough treatment. The most favourable of the intracranial lesions are those arising from gummatous meningitis of the convexity.

When estimating the prognosis of cerebral syphilis, the tendency to remission and relapse of the symptoms has to be borne in mind.

SYPHILITIC MYELITIS (SOFTENING OF THE SPINAL CORD: MYELOMALACIA)

Probably the most frequent cause of so-called acute myelitis is a diseased condition of the spinal blood-vessels with thrombosis and consequent softening.

The occurrence of acute myelitis from other causes, such as bacterial infection, is relatively rare and is elsewhere described (p. 320). Spinal thrombosis therefore, giving rise to a type of acute myelitis, occurs either in association with syphilitic vascular lesions, or with atheroma.

As the clinical picture of spinal thrombosis is more or less similar whatever the cause, we propose to describe the syphilitic variety as the type.

Syphilitic myelitis may be a primary condition, or may be one of several manifestations of the action of the syphilitic virus. This form of myelitis may be associated with the pupillary changes characteristic of syphilis, and even with hemiplegia or tabes dorsalis. It is rarer than cerebral thrombosis, and although it may be unattended by clinical evidence of spinal meningitis, some degree of meningeal affection is usually present on postmortem examination.

Symptoms. The onset of paralysis may be sudden, but premonitory symptoms such as tingling in, or transient weakness of, the lower limbs are rarely absent, although in some cases they are slight and unassociated in the patient's

mind with the onset of the subsequent and graver paraplegia. Although a chill, wetting, or fatigue may be the assigned cause, a history of slight premonitory symptoms is usually obtained.

The paraplegia develops suddenly, with loss of motion and of sensation below a definite level, usually in the dorsal region of the cord, and with retention of urine. Although the symptoms are commonly bilateral, paralysis of the Brown-Séquard type is occasionally present.

If the lesion is total, the paralysis is flaccid with loss of the deep reflexes. More commonly the reflexes are not lost, and may even be increased from the onset and associated with slight spasticity. Hyperæsthesia at the upper level of the lesion may be present.

In severe cases, with complete flaccid paraplegia, bed-sores and septic infection may ensue and cause death. If this danger is averted, recovery gradually sets in, the stages of resolution being, first a return of the deep reflexes, then the substitution of incontinence for retention of urine, and later the gradual restoration of sensation. In from three to five months sensibility may be entirely restored. During this time a slow and progressive return of motor power takes place, commencing with movements of the toes, and later of the whole limb. The most troublesome of the motor symptoms is the occurrence of spasms, either extensor or flexor, more commonly the latter, along with the development of spasticity. The limbs become flexed and adducted at the hips, and the feet pointed, while sores develop from the pressure of the knees against each other and on the heels and sacrum.

The muscles show general wasting without electrical changes, and the skin becomes dry and atrophic.

With proper care and nursing contractures may be prevented or reduced to a minimum, and the patient enabled to walk with or without assistance, the gait being of the spastic type.

In the less severe cases recovery may be almost complete, the patient walking within about three months of the onset, but exhibiting in many instances slight weakness on one side.

Cystitis may arise from catheterisation, or from infection from bed-sores. This may lead to suppurative pyelitis

and death from uræmia. Pulmonary complications, such as hypostatic congestion and bronchitis, are troublesome.

The points of value in the **differential diagnosis** of this condition are: (*a*) Pupillary changes (inequality, irregularity of outline, sluggish light-reaction or reflex iridoplegia) characteristic of syphilis. (*b*) Absence of nystagmus, but impairment of the ocular movements. (*c*) Normal motion and sensation above the level of the lesion, except for a zone of hyperæsthesia. (*d*) Below the level of the lesion, motor paralysis (flaccid or spastic) and loss or impairment of all forms of sensibility, sometimes having the features of the Brown-Séquard 'symptom-complex.' (*e*) The deep reflexes are eventually exaggerated, with clonus, extensor plantar response, and absent abdominal reflexes below the level of the lesion. (*f*) At first retention of urine and involuntary evacuation of fæces, followed later by reflex incontinence and obstinate constipation.

HYPERTROPHIC CERVICAL PACHYMENINGITIS

This is a condition characterised pathologically by chronic inflammation and thickening of the inner layer of the dura mater and of the soft membranes which surround the spinal cord in the cervical region. In our experience it is a syphilitic condition, although it has been ascribed by some writers to trauma of the spine. Some cases are complicated by cavity formation within the cord.

Symptoms. The onset is usually subacute, the early symptoms being due to involvement of the nerve roots, sensory disturbances preceding the motor. These are at first unilateral, but later become bilateral. Pain in the neck extends to the occipital and interscapular regions, and is often associated with rigidity and pain on movement, and sometimes by tenderness on pressure over the cervical vertebræ.

The root pains may be associated with hyperæsthesia, and later with loss of sensation, and are referred to the upper limbs. The pain may persist for months, and is followed by atrophic paralysis, especially affecting the intrinsic muscles of the hand and the flexors of the wrist and fingers. The hand therefore assumes a position of hyperextension at the wrist,

extension of the fingers at the metacarpo-phalangeal, and flexion at the interphalangeal joints (preacher's hand).

Cervical sympathetic symptoms may ensue early, at first of an irritative character, with dilatation of the pupil, exophthalmos, widening of the palpebral fissure, and sweating over the same side of the face; later by paralytic phenomena —contraction of the pupil, ptosis, enophthalmos, and absence of sweating.

As the pressure upon the cord increases, symptoms of spastic paraplegia develop with sphincter trouble, and in extreme cases objective sensory loss.

Prognosis. Many cases tend to progress and end fatally, but a certain number become arrested with diminution of the root pains, and some recovery of power.

The **treatment** consists mainly of rest, local inunction of mercury and large doses of the iodides internally, combined with radiant heat or sulphur baths. If the disease progresses despite treatment, surgical interference may be attempted and an endeavour made to loosen and free the affected membranes. A continuance of the mercurial treatment is advisable, even after operation. Where ordinary remedies fail to relieve the pain, division of the posterior nerve roots is recommended. Atrophic and paralytic symptoms should be combated with massage, passive movements, and appropriate electrical treatment.

GUMMA OF THE CORD

Localised gumma of the spinal cord is rare, but gummatous meningitis with extension into the cord along the blood-vessels or pial processes is common.

SYPHILITIC DISEASE OF THE BONES

Syphilitic disease of the bones of the spine is rare. It occurs in the form of exostoses or periosteal nodes, but rarely gives rise to caries.

Syphilitic disease of the bones of the skull may give rise to a diffuse dense sclerosis, to localised thickenings and exostoses, to necrosis or to local thinning (cranio-tabes). Syphilitic periostitis of the other bones, especially of the forearm, may involve the nerves secondarily by pressure.

Treatment of Cerebro-Spinal Syphilitic Lesions

Syphilitic affections of the nervous system are treated on the same lines as similar lesions elsewhere in the body. The success of treatment depends to a large extent upon the carrying out of certain general principles. First, treatment should be commenced as soon as the nature of the disease is recognised; secondly, it should be of an intermittent character, the specific remedies being given for periods of about two months, and followed by tonic treatment; thirdly, treatment should be continued for a period of over two years.

The following are the drugs and their methods of administration:—

1. **Mercury.** This may be given either by the mouth, by inunction, or by intramuscular injection.

(*a*) *By the mouth.* If prescribed in this way the best preparations of mercury are corrosive sublimate (gr. $\frac{1}{12}$ to $\frac{1}{6}$) and the proto-iodide (green iodide of mercury) (gr. $\frac{1}{8}$ to 1). The disadvantages of the ingestion method are uncertainty in the action of the drugs, irregularity in their administration, and a tendency towards gastro-intestinal troubles.

(*b*) *Inunction.* This is the method used at Aachen and other spas. One to one-and-a-half drachms of blue ointment, or the ung. hydrarg. oleate, may be rubbed into the skin, preferably after a hot bath. The rubbing should take about fifteen or twenty minutes, and different portions of the body should be used on consecutive days. A course usually consists of sixty daily rubbings.

(*c*) *Intramuscular injections.* This method is strongly advocated by some authorities. Its advantages are that a known quantity of the drug is administered and absorbed, that smaller doses may be given, and that less frequent applications are necessary. Injections of the soluble salts require to be given more often than the insoluble, and their application is more painful. The soluble preparations are: hydrarg. perchlor., gr. $\frac{1}{8}$; hydrarg. succinimide, gr. $\frac{1}{6}$ to $\frac{1}{4}$; and hydrarg. cyanid., gr. $\frac{1}{4}$ to $\frac{1}{2}$.

The insoluble preparations are: metallic mercury in doses of 1 grain, prescribed according to Lambkin's[1] method;

[1] Lambkin, *System of Syphilis*, 1908.

salicylate of mercury, gr. $\frac{1}{3}$; and calomel, gr. $\frac{1}{2}$ to $\frac{3}{4}$. These may be prescribed about once weekly over a period of two months.

Mercury should be persevered with until salivation occurs, provided that the general state of the patient is maintained.

2. The iodides. These may be prescribed, either as the potassium or the sodium salt, in doses increasing from 5 to 20 or 30 grains thrice daily. When they are given by the mouth in conjunction with inunction of mercury, highly satisfactory results are obtained in the treatment of cerebro-spinal syphilitic lesions.

Iodides are of especial value in the treatment of the later manifestations of the disease.

3. Arsenical preparations. A number of new remedies, having arsenic as their chief ingredient, have been lately introduced, mainly in consequence of the researches of Neisser, Hoffmann and others, upon the treatment of experimentally induced syphilis. Atoxyl, arsacetin, and soamin may be given either by the mouth or by intramuscular injection. Serious toxic effects (*e.g.* blindness) have been recorded in consequence of their use. It is, however, too soon to express any opinion upon their action in the cure of syphilis. It is possible that they may serve as efficient tonic remedies after a course of mercurial treatment.

Local treatment. In conjunction with the general therapeutic measures just described, treatment of the paralysed limbs by aid of massage, passive movements, and the faradic current should be carried out over long periods of time.

Chapter II

PARASYPHILITIC DISEASES

1. TABES DORSALIS (LOCOMOTOR ATAXY)

Tabes dorsalis is one of the commonest chronic diseases of the nervous system. It is essentially an affection of the sensory neurones, but as such involves the functions of the nervous apparatus as a whole.

The characteristic morbid changes are degeneration of the posterior nerve roots, of the fibres which pass from them into the posterior columns of the spinal cord, and of the sensory fibres in the peripheral nerves.

The clinical manifestations of the disease consist of abnormal sensations of pain and of paræsthesia, impairment or loss of various forms of sensibility, diminution of muscular tone, loss of the deep reflexes, sphincter troubles, inco-ordination, trophic disturbances, and visceral complications.

Etiology. The disease, except for a special type occurring in the young and known as juvenile tabes, is one of early adult and middle life. About 50 per cent. of the cases commence between the ages of thirty and forty. It affects men more frequently than women, the relative proportions being about ten to one.

Syphilis is the most important, and, indeed, the fundamental factor in the production of tabes. Tabes is a degenerative process of syphilitic origin—a parasyphilitic disease. The arguments urged against the syphilitic origin of tabes are based chiefly upon the absence of a syphilitic history in a small proportion of cases, and upon the frequent failure of the antisyphilitic remedies in the treatment of the malady.

Our statistics show the large proportion of the cases of tabes and general paralysis in which a history of venereal infection was given. Out of 166 cases, 140 gave a history of previous syphilitic or gonorrhœal infection. In the cases of tabes alone 76·6 per cent. gave a history of syphilitic infection, a percentage which was raised to 89·5 per cent. if gonorrhœal infection was included. Of the paretics, 47·6 per cent. admitted to syphilitic infection, a percentage which was increased to 69 per cent. when other forms of venereal infection were included.

The cases in which venereal infection was denied formed a relatively small proportion of the total number, but it is interesting to note that in the cases examined, a cytological examination of the cerebro-spinal fluid gave the same marked lymphocytosis as was observed in the definitely syphilitic cases.

Heredity plays quite an unimportant part in the production of the disease, except in cases of juvenile tabes, where hereditary syphilis is practically universal.

Injuries, acute diseases, severe strain, exposure to cold, and alcoholic and sexual excess have all been cited as determinant factors in the production of the disease, and undoubtedly play a part by lowering the vitality of the nervous system.

Morbid anatomy. The naked-eye examination reveals some thickening of the pia arachnoid over the dorsal surface of the spinal cord, which is often somewhat flattened. The

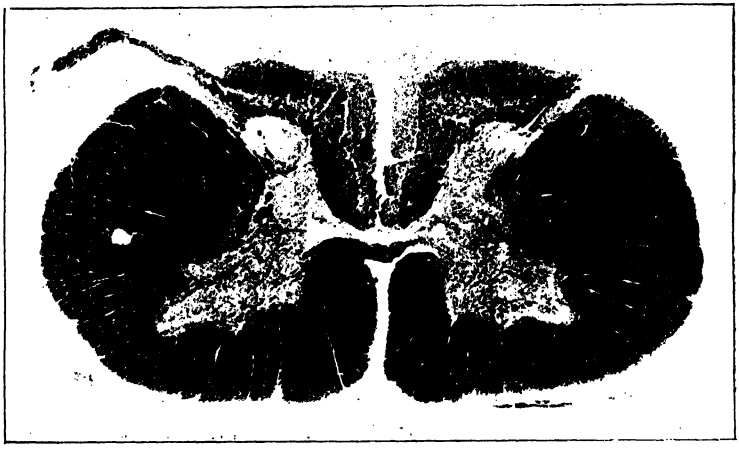


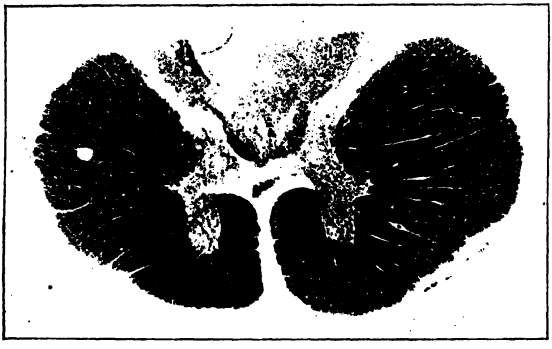
FIG. 113.—A transverse section of the spinal cord from a case of early tabes dorsalis.

posterior roots are thin, but the atrophy is not universal, though often symmetrical. On making transverse sections of the cord, the posterior columns are found to be shrunken and discoloured, their dull grey translucent appearance contrasting with the whiteness of the anterior and lateral columns. These changes are most noticeable in the lumbar region and the corresponding posterior nerve roots. Degeneration of the optic and some other cranial nerves may also be noted.

The characteristic pathological change in tabes is a degeneration of the sensory fibres, which enter the cord from the posterior roots. The posterior columns are composed of exogenous fibres which have their origin in the posterior root ganglia, and of endogenous fibres which arise from cells situated within the spinal cord. As already mentioned, the

exogenous fibres are those which are chiefly affected in tabes, and certain of them suffer more than others.

There are four distinct sets of fibres which pass into the cord from a posterior root. (1) Short fibres, which form the inner bundle of the posterior root, and, after running a short course in the cord, terminate in the posterior horn of



FIGS. 114 AND 115.—Two sections of the spinal cord from a case of well-marked tabes dorsalis.

the same side. (2) Medium fibres, which pass up for some distance in the posterior columns, and terminate by giving off collaterals to the cells of Clarke's column and to the anterior horn. (3) Long fibres, which pass up the posterior columns to the posterior nuclei of the same side. As these fibres are displaced more and more towards the mesial septum by those coming in from each posterior root, it comes about that the postero-internal column in the cervical region contains the fibres which have entered from the sacral,

lumbar, and lower dorsal roots. (4) Fine fibres, which pass into the zone of Lissauer, and form a cap for the posterior horn.

The picture of degeneration found in a case of tabes, therefore, depends upon the stage of the disease at which the cord is examined. The fibres which degenerate earliest are: (1) the medium fibres and their collaterals; and (2) the fine fibres of the zone of Lissauer.

The medium fibres are displaced towards the middle of the posterior columns, and lie in the inner part of the postero-external division; so that if the case be an early one, the degeneration will be noticed in that region, where the tract has received the name of 'Bandelette externe,' as well as in the zone of Lissauer, to which the fine fibres pass. If the case be more advanced, degeneration will be noticed in the long and short fibres as well, so that there will be complete degeneration of the postero-internal column, except for the endogenous fibres. If the disease is mainly limited to the lumbo-sacral region, degeneration will be found in the postero-external columns of the lower lumbar and sacral regions, and in the postero-internal columns of the cervical region. If the disease is chiefly in the cervical region (cervical tabes), degeneration will be noted in the postero-external column of the cervical region only.

The lesion consists of a primary degeneration of nerve fibres, the myeline sheath breaking up, and the fibre eventually being destroyed. As a secondary effect proliferation of the neuroglial tissue takes place, so that a dense network of neuroglia is formed, in which few or no nerve fibres can be distinguished.

Thickening of the pia arachnoid over the posterior aspect of the spinal cord is not uncommon, and in some cases there is a leptomeningitis.

In connexion with the *posterior horns* there is observed degeneration of the collateral reflex fibres and fibres round Clarke's column; the cells of the posterior horn show no change.

Degenerative changes in the *anterior horn* cells of varying degree and intensity may be noted, but such changes are rare, and are met with chiefly in those cases of tabes associated with muscular atrophy. The anterior horn changes

are, in all probability, secondary to changes in the motor nerves.

Posterior nerve roots. In early cases very little change may be made out in the posterior nerve roots outside the cord, although degeneration of the exogenous fibres within the cord may be well marked. In more advanced cases the myeline sheaths of the fibres swell, disintegrate, and finally disappear, especially where the roots pass through the pia-arachnoid membrane. This degeneration may extend in extreme cases into the posterior root ganglia. Secondary to the parenchymatous degeneration of the myeline sheaths, there is a proliferation of the surrounding perineural connective tissues.

Posterior root ganglia. Atrophic changes in the ganglion cells and secondary proliferation of the interstitial tissues have been described in a few cases, but such changes are rare, and are probably only secondary to the degeneration of the posterior roots. They are certainly not accountable for the degeneration of the posterior root fibres, which may be extreme in degree, without any obvious change in the ganglia.

Peripheral nerves. The changes found in the peripheral nerves are generally confined to the small sensory fibres near their peripheral distribution in the skin, and are never in any degree comparable to the degeneration found in the posterior root fibres. Degeneration of the motor nerve fibres of the spinal and cranial nerves has been described in cases with muscular atrophy.

Degeneration of the *optic nerves* is not uncommon, and commences in the peripheral portion of the nerves near the disc and extends towards the brain. Degeneration of the ganglion cells in the retina has been seen in some cases, but in others, where there was well-marked optic atrophy, no degeneration was found in the retinal cells. The degeneration is first noticed where the fibres lose the myeline sheath at their entrance into the eyeball; it is possible that this is their most vulnerable portion, but on the other hand there is some evidence to suggest that the optic nerve atrophy is not a true tabetic atrophy, but is due to an interstitial neuritis with secondary pressure and atrophy of the optic fibres (Léri).

The sensory roots of the *fifth nerve* and the *auditory nerve* have been found degenerated in some cases. In the medulla degeneration of the sensory fibres of the fifth, the solitary bundle and the posterior nucleus of the vagus have been observed.

Marina has described atrophic changes in the cells of the *ciliary ganglion* in cases with reflex iridoplegia.

The cerebro-spinal fluid. In tabes a considerable increase of the lymphocytes, up to 100 or 150 or more, is found on microscopic examination of the centrifuged deposit. This lymphocytosis is useful as a diagnostic point in cases where the diagnosis is otherwise uncertain.

The morbid changes occurring in the *tabetic arthropathies* consist chiefly of distension of the capsule of the joint, which may eventually atrophy. The synovial membrane becomes thin and shreddy, the cartilage disappears, and the bone is eroded and destroyed. In other cases a new formation of bone develops in the form of bony outgrowths, or deposits around the joint.

The bones of the limbs sometimes become rarefied and are liable to spontaneous fractures. In other cases a syphilitic osteitis or sclerosis is observed.

Blood-vessels. Although the occurrence of gummata is rare in tabes, atheroma and chronic aortitis are frequently found.

Symptoms. The clinical course of a typical case of locomotor ataxy has been divided into three stages, according to the predominance of certain symptoms. The classical division into preataxic, ataxic, and paralytic stages, although convenient for description, is far from being universally applicable. Even in the first stage some degree of instability and incoordination of movement may be detected by careful examination, and it is only when the inco-ordination becomes obtrusive that the second stage may be said to appear; and the third or so-called paralytic stage is merely one of overwhelming ataxia. It is therefore obvious that these divisions are entirely arbitrary and are based solely upon the clinical manifestations of one symptom, ataxia. Clinical experience daily demonstrates the fallacy of this classification. We have seen cases in which the initial symptom was acute ataxy without subjective sensory symptoms or sphincter disturbances;

moreover, many cases which come under treatment in the ataxic stage recover from their ataxy.

As the mode of onset and course of the disease vary so widely, the symptoms are best considered *seriatim*, as they affect the several systems.

The **mental condition** of tabetics should be carefully examined, in view of the close association which exists between tabes and general paralysis of the insane. In uncomplicated cases the mental condition remains normal, but in a few instances delusions of persecution, melancholia, and occasionally suicidal tendencies are present.

In some cases mental symptoms characteristic of general paralysis gradually develop, but in others the onset of tabes and the mental symptoms of paretic dementia occur simultaneously (tabo-paresis).

Cranial nerves. Degenerative changes in the olfactory, glossopharyngeal, and auditory nerves, with clinical evidence of loss of function, have been found in a few cases.

Vision. Progressive loss of sight, with contraction of the visual fields, occurs in about 10 per cent. of the cases of tabes. It is due to atrophy and degeneration of the optic nerves. Tabetic optic atrophy as a rule results in total failure of vision. Optic atrophy may be an early or a late symptom, and affects one eye first, but its development has no constant influence on the progress of the disease.

The pupils. Large pupils are sometimes met with, and when present are often associated with paralysis of the third nerve or with optic atrophy. Small pupils are also observed, and generally in association with cervical tabes, but medium-sized pupils are the most common.

The pupils may be equal in size or exhibit a striking inequality; as a rule careful observation will show some inequality. Sometimes the position of the pupil is eccentric. The outline of the pupil is rarely circular; it usually shows definite irregularities, which, if not due to local causes, constitute a sign characteristic of tabes, general paralysis, and cerebro-spinal syphilis. The reaction of the pupils to convergence and accommodation is generally preserved, although in a small number of cases it is impaired or lost. In marked contrast to this is the interference with the light-reaction which is so constant a feature. · Loss of the light-reflex of the

pupil, with preservation of the contraction on accommodation, is known as the Argyll-Robertson phenomenon, and is found in about two-thirds of the cases of tabes. Loss of the pupillary light-reaction may be complete or incomplete, or it may be unilateral or bilateral. The contraction may be feeble and sluggish and followed by a rapid dilatation, or the

FIG. 116.—Illustrates the characteristic appearance of the face in a case of old-standing tabes dorsalis—the tabetic face.

pupil may oscillate (hippus). Although the direct pupillary light-reaction is lost in one eye, yet the consensual light-reaction, when light is thrown upon the other eye, may be unimpaired.

The practical outcome of these observations is that any interference with the pupillary light-reflex, associated with irregularity of the contour of the pupil (not due to local causes), is as significant a sign of tabes as the complete loss of the light-reflex.

The reaction of the pupils to stimulation of the skin of the neck may also be lost or impaired.

External ocular muscles. Temporary paralysis or paresis of an external ocular muscle is not uncommon in the early stages, and a history of temporary diplopia, or ptosis, is often obtained. Permanent paralysis, or paresis, of one sixth nerve or of one third nerve is frequently observed. Temporary paralysis of the third nerve always results in a permanent loss of the pupillary light-reflex. Although these paralyses frequently occur, the most common affection of the ocular nerves is a slight drooping of both upper lids, with compensatory over-action of the frontalis muscles. This feature, in conjunction with small pupils which give the eyes a steel-like look, constitute the 'tabetic facies.' (Fig. 116.)

The *motor functions of the fifth nerve* are rarely affected, but the *sensory portion* sometimes suffers. This gives rise to lancinating pains and occasionally loss of sensibility in the distribution of the nerve.

The *seventh nerve* is not affected.

Affections of the *palate, pharynx, and larynx* are frequent. These may be either sensory, such as loss of taste, anæsthesia and paræsthesia, or motor abductor paralysis of the vocal cords. These are particularly common, occurring in about 40 per cent. of the cases. Unilateral or bilateral paralysis of the abductors of the vocal cords is chief amongst them; and although it gives rise to no phonetic loss, dyspnœa and discomfort may be present. Paralysis of the adductors of the cords and of the soft palate is rare.

Unilateral atrophy of the tongue occurs sufficiently often to be mentioned as an occasional occurrence.

Motor system. The motor system suffers indirectly as the result of the affection of the sensory apparatus. The muscles may exhibit some signs of general malnutrition and reduction in size, but as a rule local wasting does not occur. There are, however, a not inconsiderable number of cases in which muscular atrophy is associated with tabes, and in these cases changes in the motor nerves, or in the anterior horn cells, or their bulbar analogues, have been found to account for the wasting.

The muscular irritability to mechanical stimulation may

be diminished in the later stages of the disease. The power of individual muscles is well maintained, although a slight relative loss of power is common, and depends upon the degree of muscular inco-ordination.

Atonia. The most characteristic change in the motor system is the impairment or loss of tone in the muscles—hypotonia and atonia. It is due to the loss of the sensory stimuli impinging upon the motor centres in the spinal cord. It is not confined to the muscles, but involves the ligaments in and around the joints. It is shown by flaccidity of the muscles, by the diminution or loss of the deep reflexes, and in severe cases by an abnormal degree of movement at the several joints. Hyperextension of the knee joint (genu recurvatum) and abnormal flexion of the hips, whereby a patient may be enabled to place both his feet behind his head, are examples of the effect of this loss of ligamentous and muscular tone. (Figs. 117 and 118.)

FIG. 117. — Photograph showing genu recurvatum or hyperextension of the knee joint.

Ataxia. The inco-ordination of muscular action gives rise to ataxia. It may be an early, but is more usually a late, symptom. Its onset is as a rule gradual, but sometimes extremely rapid. When slight in degree the patient may obviate it by using his visual impressions to guide his erring limbs. Thus it is that the earliest symptoms are noticed when he is deprived of vision, as when in the dark or when the eyes are closed. Ataxy is due to the loss of the sense of position in the muscles and the joints. It gives rise to an inability to perform the finer movements of the fingers and the hands, such as buttoning the clothes and writing, and to an inability to walk along a straight line. In the earlier stages closure of the eyes may reveal inco-ordination in the performance of the finger-to-nose

test, or increased unsteadiness on standing with the feet together (Romberg's sign). In advanced cases the patient cannot walk, and sometimes cannot even feed himself.

Gait. In typical cases the gait is unsteady and staggering, the patient walks with his head bent down and his eyes fixed upon the ground. His feet are everted and he walks upon a wide base; the movements of his legs are excessive, the feet are thrown out too high, and the heels come down sharply on the ground. Any attempt to stop suddenly, or to

FIG. 118.—Illustrates an extreme degree of hypotonia.

turn round, results in much staggering, and perhaps a fall. Darkness increases the difficulty in walking or may render it impossible.

Sensory system. The sensory symptoms of tabes are both subjective and objective. The most characteristic of the subjective symptoms are: (*a*) Sharp, shooting, lancinating pains, which occur in paroxysms commencing suddenly and terminating abruptly. The pains are felt in the fleshy parts immediately under the surface of the skin, and are specially frequent in the legs and ulnar side of the forearm. Similar pains often radiate from and to the joints. After a bout of such pains there is often extreme hyperæsthesia and tenderness to tactile impressions on the skin over the affected area. (*b*) Deep pains of a gnawing and burning character, which may last for days or weeks, and are usually referred to the bones or deep muscles. (*c*) Superficial tenderness and feelings of constriction by hot or painful bands, of which the girdle sensation is the most common example. Besides these, a sense of compression, numbness, and tingling are commonly described.

Such subjective phenomena are usually early symptoms

of the disease and are often attributed to rheumatism or gout.

The objective examination of the several forms of cutaneous and deep sensibility reveal many characteristic abnormalities.

1. The *loss of sense of position* is seen in the inco-ordination and unsteadiness to which it gives rise. As a rule, the distal parts of the limbs suffer before the proximal. Thus, a patient may be unable to tell in what position his toes, feet, or legs are placed, and when asked to point to his foot will take as a guide the impressions received from the hip joint. Similarly in the upper limb he may not know the position of his fingers, but be able to tell the position of his hand and forearm.

2. The *loss of deep pain* (painful pressure) is an early symptom, especially in the muscles of the legs, when painful pressure on the calf muscles is not appreciated as such. The loss of deep pain is a valuable sign in the differential diagnosis of tabes from alcoholic neuritis.

3. *Cutaneous pain* is usually impaired out of proportion to the loss of tactile sensibility. In the early stages it may be diminished or delayed, in advanced cases it is lost.

4. *Extremes of heat and cold* are impaired to much the same degree as cutaneous pain, and both are usually affected over the same areas.

5. *Discrimination and vibration* are often impaired, but as a rule later in the course of the disease and less completely than those already mentioned.

6. *The sensibility to moderate degrees of temperature* and *light tactile sensibility* are frequently found to be slightly defective, but such changes are late in appearing. Its preservation is in marked contrast to the loss of painful sensibility.

7. *Contact and pressure* are preserved in almost all cases, and are the last to disappear.

The distribution of the sensory changes depends entirely on the situation of the disease. In cases where the lumbo-sacral roots are chiefly involved, the sensory loss is confined to the perinæum and lower limbs. In cervical and dorsal tabes the ulnar border of the arm and the upper thoracic root areas are chiefly affected. (Fig. 119.)

But as in most cases of tabes both the cervical and lumbo-sacral regions of the spinal cord are the seat of degeneration, characteristic sensory disturbances may be demonstrated over both the upper and lower limbs and the trunk. In rare cases the sensory loss is found in the distribution of the fifth nerve.

Reflexes. Interference with the plantar, bulbo-cavernous, cremasteric, and anal reflexes is common. These reflexes are affected *pari passu* with the involvement of the lumbo-sacral cord. The epigastric and abdominal reflexes are rarely lost, as the dorsal roots are often not involved in the tabetic process. In cases of gastric crises the superficial epigastric reflexes may be hypersensitive.

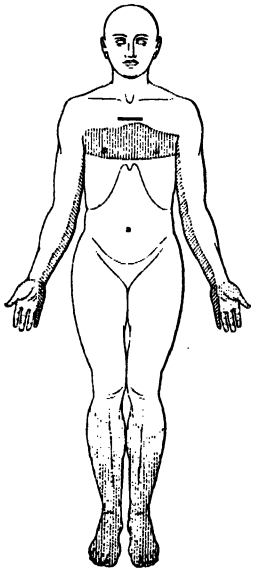


FIG. 119.—Chart showing a common distribution of e sensory impairment tabes. Dots signify bjective, lines objec-ve phenomena.

Deep reflexes. These are at first diminished and ultimately lost. If the disease commences in the lumbo-sacral region the ankle and knee jerks disappear, as a rule the ankle before the knee jerk. The deep reflexes of the upper limbs are not lost unless the upper cord is affected. In advanced cases all the deep reflexes, including the jaw jerk, may be abolished.

Organic reflexes. Interference with micturition may be an early sign. At first there is 'stammering' micturition, by which is meant that the patient feels an urgent call to pass water, but when he tries to do so, fails, but having failed he may shortly after have precipitant micturition, which he is unable to control. Later there may be loss of control at night, and when excited during the day. The final stage is over-distension of the bladder, leading to dilatation of the ureters and pelvis of the kidney.

Distension of the bladder may occur quite unknown to the patient, as owing to the loss of sensibility he has no feeling of pain or discomfort. The liability of the residual urine to become septic and give rise to cystitis and pyonephrosis con-

stitutes one of the most serious complications of the disease, and is in many cases the ultimate cause of death. Constipation is common, but in some cases attacks of diarrhœa with loss of control over the anal sphincter occur.

Generative functions. In the early stage the patient may complain of pains in the genital organs with an increase of the sexual desire. Erections and nocturnal emissions are frequent. In the later stages sexual power and desire are lost and erections cannot be obtained. This is often associated with loss of testicular sensation and atrophy of the testicles.

Visceral crises. The commonest is the gastric crisis. This consists of attacks of nausea and vomiting, of sudden onset, accompanied, or preceded, by severe pains in the region of the stomach passing round the body or through to the back. The skin over the epigastric region may become hyperæsthetic. Vomiting is persistent, and the patient brings up what food he may have in his stomach, then bile and blood. The attacks may last for a few hours or several days. They occur during the early stages of the disease, and as a rule tend to become less severe and less frequent as years go on. The ingestion of food is not an exciting cause in the majority of cases, but in some a disordered stomach undoubtedly gives rise to an attack.

Laryngeal crises are of two types. The mild are characterised by an obstinate dry cough, occurring at frequent intervals, and ending by the expectoration of a quantity of viscid mucus; the severe by pain, stridor, and a feeling of suffocation and impending death, the lips become livid and the skin pale, and there is indrawing of the chest and substernal region. These attacks pass off as suddenly as they come on.

Laryngeal crises may be present when there is no paralysis of the vocal cords, or both may be combined. Paralysis may exist without laryngeal crises, but often follows such attacks.

Among the rarer forms of visceral crises are rectal, nephritic, intestinal, cardiac, and respiratory crises.

Trophic changes. The more serious trophic changes which occur in tabes are (1) arthropathies, and (2) trophic ulcers and sores. The changes in the joints are as a rule of gradual onset, and are often preceded by pains in and around the affected joint. The first stage in the process consists of a

relaxation of the ligaments of the joint; later, the synovial membranes become thickened and ossification takes place in the fringes. The bones may either hypertrophy or atrophy, and the joint becomes distended with synovial fluid. This results in the total disorganisation of the joint. Various complications may arise, such as a serous or hemorrhagic effusion into the joint, or the limb below the joint may become

FIG. 120.—Illustrates arthropathy (Charcot's joints) of the right knee and ankle in the case of tabes.

the seat of a hard and tense œdema. Secondary to the joint changes the surrounding muscles undergo atrophy, and in some cases the peripheral nerves may be pressed upon with resulting paralysis. These changes do not cause the patient any pain, and suppuration or tuberculous infection rarely occur. The affection is usually limited to one joint. The knee joint is that most commonly affected, and then, in order of frequency, the hip, shoulder, ankle, and elbow. Hypertrophy is common in the knee and elbow joints, and

atrophy when the hip joint is the seat of the lesion. (Fig. 120.)

Commonly the bones become rarefied with loss of osseous tissue, and in such cases spontaneous fractures are liable to occur. These are of sudden onset without any exciting cause, or are due to a trivial injury. There is no pain, and crepitus can be produced without the patient's knowledge. The union is often delayed and an abnormal amount of callus is thrown out. The bones of the lower limbs appear to be most liable to spontaneous fractures.

Fig. 121.—Illustrates the characteristic perforating ulcers of tabes.

The 'tabetic foot' is the result of trophic changes in the ligaments, whereby the arch of the foot sinks, the foot is everted and notched, so that the patient walks upon its inner side and the metatarso-phalangeal joint of the big toe forms a prominence.

Perforating ulcers. These ulcers form one of the special signs of tabes. They most frequently arise on the plantar surface at the base of the great and little toes. They commence usually as a corn, round which the patient complains of sharp shooting pains. The corn becomes excoriated, and a small circular ulcer with irregular edges is formed. From the ulcer a serous or sero-purulent discharge exudes. The ulcer extends deeply so as to expose the subjacent bone, of which a sequestrum is formed. The ulcer itself is painless. (Fig. 121.)

Similar trophic changes may occur in the nose, palate, and jaws, in the last instance resulting in the loosening and dropping out of the teeth.

Rarer changes are spontaneous rupture of tendons, herpes of the skin, subungual hemorrhages, purpura, and œdema. Falling out of the hair and brittleness of the nails are other common trophic manifestations of the disease.

Emaciation, sometimes extreme, is an almost constant accompaniment of locomotor ataxy. It occurs in all types of the disease, independently of the amount of nourishment which the patient is taking.

Clinical types. In the early stages the disease may present several types.

1. A type commencing with slight limb pains, which are usually attributed to rheumatism, and which may persist for many years before the onset of ataxia. This may be described as a type with a prolonged first stage. As a rule, the usual physical signs in the pupils and deep reflexes are definite even in the early stages.

2. The *neuralgic* type. This is a rare variety characterised by the existence of severe and long-continued pains, often with little or no ataxy or sphincter trouble, and with preservation of the deep reflexes. The pupil changes may be quite definite.

3. The *gastric* type is characterised by the frequent recurrence of severe gastric crises. Sensory changes and ataxy may be absent or slight, but the other signs of the disease are usually present.

4. The *trophic* type. In the cases of this type joint changes and perforating ulcers are the early symptoms, and sometimes persist for long periods before any other symptoms develop. This type is often associated with visceral crises.

5. The *ataxic* type. This is characterised by the sudden onset of severe ataxy, often preceding the advent of pains and other symptoms.

6. The *perinæal* type. In this the action of the vesical and rectal sphincters is impaired and the sexual functions are abolished. These disturbances may precede, or be accompanied by limb pains, but are rarely associated with ataxy in the early stages.

7. The *amblyopic* type. In this type loss of vision and

optic atrophy may be found in association with loss of the deep reflexes, but without the other symptoms of the disease.

Prognosis. The prognosis as regards life is good, though the disease may persist for many years. Its average duration is about twelve or fifteen years. Although the malady is known as progressive locomotor ataxy, it is a matter of practical experience that care and judicious treatment may do much to mitigate the symptoms and prolong life. In many cases, also, it would appear as if the disease became arrested.

The following complications are unfavourable: (*a*) Cases which develop symptoms of general paralysis. These do not survive the onset of the mental symptoms more than two or three years. (*b*) Cases complicated with vascular lesions, either general arterial degeneration, or aortic insufficiency with aortitis and angina pectoris. (*c*) Cases with pronounced bladder complications, especially retention of urine, owing to the risk of infection of the urinary passages.

Cases characterised by the rapid onset of ataxia, even when most severe, yield in a remarkable manner to the influence of rest and perseverance with co-ordinating, or Frænkel's exercises.

The pains are difficult to allay, but even their continuance in a severe degree over prolonged periods, although temporarily exhausting, do not interfere with the duration of life, or modify the course of the disease.

Treatment. Much may be done in this disease by judicious care and treatment. The disease would appear to become arrested in some cases, and troublesome complications, terminating in death, may be averted or controlled. It is therefore essential that the malady should be taken in hand as soon as its nature has been recognised, and the patient encouraged by every available means to persevere with treatment.

The first question which demands consideration is whether *antisyphilitic treatment* should be adopted, and if so, what is the most suitable form in which it should be given. To prescribe a course of this treatment in every case of tabes is not good practice, although many will derive benefit from it. Those most likely to be improved are cases in which the tabetic symptoms have arisen within a comparatively short time (five years) after infection, and those who have not previously had an energetic course of treatment for the original

syphilitic infection. Although these may be laid down as guiding principles, cases of tabes which do not fall into either category may with advantage undergo a course of mercury and the iodides. In cases with much debility, and in old-standing cases, general tonic treatment will be found of greater service.

Antisyphilitic treatment may be given in the form of mercurial inunction, or intramuscular injections in combination with the iodides by the mouth (p. 378).

General treatment. The patient's life should be so regulated that all excesses are avoided, both as regards work and physical exercise, and the use of alcohol. Exposure to cold and wet is especially detrimental, so that residence in an equable and genial climate is to be prescribed. Equability of climate is not always to be found by going abroad, and we have known tabetics whose pains have been less and whose general comfort has been greatest when resident in London. An occasional course of massage, especially if given in combination with mild faradism, and for a time rest in bed, are usually methods which may be adopted when there is any temporary increase in the symptoms, such as pains, marked debility, or rapidly progressing ataxia.

Symptomatic treatment. Pains. These are best treated by analgesics, of which a large selection is at the physician's disposal—such are antipyrin, acetanilide, phenacetin, aspirin, phenalgin, exalgine, pyramidon, and aluminium chloride. They may be prescribed alone, in combination, or with iodides or salicylate of soda. Morphia is now rarely needful, but if prescribed should be given hypodermically.

Gastric crises. In severe attacks no drug other than morphia will give relief. In milder attacks, codeia, cocain, or belladonna, if given early, may sometimes arrest or modify a crisis. A useful remedy is 1 minim of tr. iodi in a wine-glass of water frequently administered. Small quantities of ice to suck and local fomentations will soothe the discomfort and pain in the epigastric region. Occasionally drinking large quantities of water is successful. Rest in bed is usually necessary, as the subsequent prostration is often severe.

Bladder weakness. The catheter should be used in all cases of retention, or when residual urine is shown to be present. Cystitis may be relieved either by urotropin, or

preferably by washing out the bladder. The patient should be instructed to pass his urine at regular intervals.

Ataxia. The most satisfactory method of treating this symptom, is that introduced by Frænkel. The principle of his method lies in the precise performance of various movements. This precision is only obtained by careful repetition, the patient devoting his whole attention to mastering the exercises. At first progress is slow as mental and bodily fatigue are induced. As benefit only results from the proper appreciation of the sensations evoked by the correct execution of the movements, the exercises should not be continued for longer than the patient is able to sustain his attention. By this repetition and by aid of what sensation remains, the patient learns to effect his movements more or less correctly. In cases where vision is retained, he is enabled to correct his errors and to attain to greater proficiency; but at the same time he must note the sensations coming from the limb which is being exercised. By the full appreciation of these sensations he is eventually able to dispense with the aid of vision.

In severe cases the patient is kept in bed and prescribed exercises, which he can carry out in the recumbent posture. As he improves he is taught exercises, first in a sitting position, then when standing, and later on when walking on the level, and finally up and down stairs. For the hands and arms exercises at a dummy keyboard, or a 'solitaire' board, or writing are useful.

The treatment should be continued for twelve months or more, according to the progress made by each case. The duration of the exercises should at first be short—about ten minutes at a time thrice daily; but later on, as the patient becomes stronger and more expert, the sittings may be prolonged from a half to one hour three times daily. They should be stopped short of the onset of fatigue.

The general results are most gratifying, especially in young tabetics, and in cases of pronounced and overwhelming ataxy.

Juvenile tabes dorsalis

This is a rare condition, and affords a striking example of the association between tabes and syphilis. The majority of the cases are due to inherited syphilis, although a small

proportion would appear to arise from syphilis acquired during the early years of life. In some cases, although no signs of congenital syphilis may be present in the patient, a parental history of syphilis, or of a syphilitic malady, may be obtained.

The *symptoms* of the disease are essentially similar to those seen in the adult forms. The symptoms make their appearance shortly before or at puberty, and in contradistinction to the greater preponderance of the adult affection in the male, the juvenile form is more common in girls.

There are two chief types: one in which the symptoms commence with pains in the limbs and ataxia; the other in which optic atrophy is an early and outstanding feature. Argyll-Robertson pupils, sensory disturbances, and loss or diminution of the deep reflexes are invariably present. In many instances ataxia is absent or of late development. In the latter type evidences of congenital syphilis are common.

The *diagnosis* has to be made mainly from Friedreich's ataxy (p. 474).

The *prognosis* is favourable as regards the life of the patient, which may be prolonged for many years, up to seventeen years in one case.

2. PROGRESSIVE GENERAL PARALYSIS PARALYTIC DEMENTIA

General paralysis is a progressive degenerative disease due to decay and death of the cortical association neurones (Mott) characterised by mental, motor, and sensory symptoms.

Etiology. Statistics show that from 75 to 85 per cent. of general paralytics have suffered from syphilis. The constant relation between the juvenile form of general paralysis and congenital syphilis is also strong proof of the causal connexion between the two maladies. The not infrequent relation and coexistence of tabes and general paralysis in the same person is a forcible argument not only in favour of the essential unity of the two diseases, but also of their common origin from previous syphilitic infection. In this relation, also, a point has been made of the inability to infect sufferers from general paralysis with the syphilitic poison

(Krafft-Ebing). A further interesting fact in favour of the syphilitic causation and identity of tabes and general paralysis is seen in the cases of so-called conjugal tabes and general paralysis.

Although syphilis is the predisposing cause of the malady other factors play a not unimportant part in its production: these are alcoholic and venereal excess, prolonged mental strain, worry, anxiety, and overwork. These factors may induce symptoms of neurasthenia in healthy individuals in middle life as well as in those who in youth have been infected by syphilis. The onset of neurasthenic symptoms after thirty-five in the latter should not be treated lightly, and should call for a careful examination. Injury to the head in cases of syphilis is only a rare cause of general paralysis.

Ford Robertson[1] and others have stated that the rôle of syphilis, in the causation of general paralysis, is to reduce the immunity of the nervous system to bacterial invasion from other parts of the body.

General paralysis is more common in men than in women; in the latter it is more frequently found in the lower social ranks. It is more common also in urban than in rural districts. It arises more often between thirty-five and fifty than either before or after these ages. A juvenile form occurs in young persons during puberty or early adolescent life. It is rarely associated with any family predisposition to mental or nervous disease.

Pathology. To the naked eye the brain of an advanced case of general paralysis is wasted and shrunken; the convolutions – particularly of the frontal and Rolandic areas—are atrophied, and the pia arachnoid only strips off with difficulty: portions of the subjacent grey cortex coming away with it. In the subdural space blood-clot may be found in various stages of organisation or cystic formation—the condition known as pachymeningitis hemorrhagica. There is usually an excess of cerebro-spinal fluid both in the subdural space and in the ventricles, the ependyma of which presents a granular appearance. The weight of the brain is considerably diminished.

Microscopical examination of the cerebral cortex shows

[1] Ford Robertson, *Review of Neurology*, 1905

degenerative changes in both the vascular and nervous elements. The cortical arterioles present thickening of the intima, hyaline, and fatty degeneration of the muscular coat, and proliferation of the perivascular nuclei. The glial cells are large, and show a characteristic 'spider' appearance; while the pyramidal cells show various stages of chromatolysis, degeneration, and atrophy. Importance has been placed upon the degeneration of the tangential and association fibres of the cortex, more particularly of the frontal lobe. Similar vascular and nervous lesions are found in other parts—such as the basal ganglia, the pons, medulla, and spinal cord.

In the spinal cord secondary degeneration, both in the crossed pyramidal tracts and the posterior columnar fibres, has been found. The optic nerves and tracts may also show a degenerative atrophy similar to that seen in tabes dorsalis.

The fibres of the peripheral nerves have also been found degenerated in this condition.

The *cerebro-spinal fluid.* The lymphocytes are greatly increased in number – p to as many as 150 or 200 or more c.mm. of fluid—and the Wasserman reaction is present in 90 per cent. or more of the cases (Mott).[1]

Symptomatology. The symptomatology of general paralysis is both definite and characteristic. Its manifestations consist of mental and motor phenomena. The earlier and wider recognition of its many types has to some extent modified the general conception of its early stages: which was at one time based upon the study of cases seen in asylum practice, at a period distinctly more advanced than that at which the disease is now recognised in the out-patient department of hospitals and in private practice.

The clinical picture of the fully developed disease is characterised by mental exaltation with grandiose ideas, slurring speech, tremulous lips and tongue, and pupillary changes.

Mental symptoms. The psychical changes consist of: loss of memory for recent events, inability to concentrate attention, impairment of the finer mental faculties, delusions, and a tendency towards progressive dementia. This dementia may commence in various ways: (*a*) with mild

[1] Mott, *Lancet*, 1909.

exaltation, grandiose conceptions, and an excessive feeling of well-being, which is often a source of self-satisfaction; (*b*) with an alteration of the character, so that the patient commits gross acts of immorality entirely foreign to his nature; (*c*) with symptoms of neurasthenia, associated with insomnia, mild depression, indecision of action, and sometimes irritability and headache; (*d*) with melancholia, which presents the usual features of depression, delusions as to the poisoning of food, and sometimes suicidal feelings and homicidal tendencies.

In whichever way the dementia starts, the tendency is towards a progressive increase in the symptoms, until eventually a condition of profound dementia results, in which memory is entirely in abeyance, and the patient pays no attention to his surroundings, nor to the care of his person.

An inability to fix the attention is a remarkable feature of most of these cases during examination.

Cranial nerves. Primary optic atrophy, similar to that found in tabes, may be seen. Oculo-motor palsies may also occur, but are probably due to coexistent intracranial syphilitic lesions.

Pupillary changes occur early: are constant and characteristic, provided the significance of irregularity in outline, inequality in size and shape, and impaired light-reaction are fully recognised. In many cases the complete Argyll-Robertson reaction is present, but the above-mentioned changes are equally significant. The fifth nerve may show some sensory anæsthesia, especially in the tabo-paretic variety.

The other motor cranial nerves show no paralyses, bu the muscles supplied by them present the characteristic tremor of this disease, which will be presently described.

The *motor system.* A characteristic tremor is the most constant early symptom of interference with the motor functions. It is seen first in the circum-oral facial muscles, lips, and tongue, and may be observed at rest; but is accentuated by emotion, attempts to speak, and on protrusion of the tongue. It consists of irregular, inconstant, more or less rapid, quivering movements; rarely confined to one muscle, but playing over groups of muscles. The same kind of tremor is seen in the outstretched hands and

arms, and some degree of inco-ordination of movement is frequently combined with it.

The articulation is slurring and tremulous: the commencement of a sentence being relatively distinct, but terminating in an inarticulate drawl. Individual syllables may be repeated, or, on the other hand, they may be glossed over. The most difficult phrases to pronounce are those in which the tongue and lips are specially brought into action—for example, 'Territorial Artillery,' and 'West Register Street.' In addition to the articulatory defect the patient is often unable to repeat sentences, owing to mental impairment, and he may also be temporarily aphasic. In the later periods he becomes inarticulate.

Voluntary power is unaffected in the early stages; but, as the disease progresses, a gradual diminution and eventually complete loss of motor power occurs. The finer movements of the hands—as in writing—become impaired by a combination of tremor and inco-ordination.

The muscular tone is at first hypertonic; but in cases of the tabo-paretic variety, hypotonia is observed. The latter is also characterised by considerable instability and ataxia.

The *reflexes*. The tendon jerks are usually exaggerated, but may be lost in the tabo-paretic cases. The superficial reflexes vary, but are as a rule present; the plantars are usually flexor, except after severe epileptiform seizures, when they show the extensor response.

The *sphincters*, especially that of the bladder, are affected early. In the later stages of dementia, incontinence and involuntary evacuations occur. Sexual power is often lost in the early stages, but in other cases is increased almost to the extent of satyriasis. Temporary return of sexual power has been known to occur after previous abolition.

Epileptiform seizures in this disease are of three kinds: (*a*) temporary losses of consciousness, followed by transient aphasia; (*b*) limited Jacksonian seizures, followed by transient and sometimes permanent loss of power; and (*c*) general epileptic seizures. During the severer attacks the temperature is increased sometimes to hyperpyrexia.

The effect of such attacks is to aggravate the symptoms of mental decrepitude, and to hasten the onward progress of the disease.

Sensory symptoms. Headache and cephalic sensations are frequent, but not constant symptoms. In the tabo-paretic cases lightning pains and the objective sensory changes characteristic of tabes are present.

Clinical types. (1) *Exalted* type. This is a form presenting exaltation and grandiose ideas, with incoherence of speech. In many respects it resembles an attack of acute maniacal excitement.

2. A *melancholic* type, rarer than the preceding, and presenting a very definite clinical picture. It commonly commences with neurasthenic symptoms, which precede for a longer or shorter time the onset of obvious delusional melancholia.

3. A type, which may be called the *tabo-paretic* type, where the disease commences with the symptoms of tabes dorsalis, but eventually develops mental symptoms of general paralysis.

4. A *convulsive* type, in which the disease commences with a series of one or more general epileptic fits. Upon recovery, some mental or motor impairment may be detected. The patient remains in this state until again prostrated by a further series of convulsions.

The **differential diagnosis** has to be made in the early stage from neurasthenia (p. 545), and in the later from diffuse cerebral syphilitic lesions (p. 373) and tumour of the frontal lobe (p. 243). In all doubtful cases of neurasthenia with a history of syphilitic infection, a cytological examination of the cerebro-spinal fluid should be made. An excess of lymphocytes is diagnostic of syphilitic affections.

From diffuse cerebral syphilitic disease the diagnosis is difficult, but in this the main points are: the occurrence of paralyses of the cranial nerves and signs of gross organic disease, the absence of grandiose ideas, and of the characteristic tremors and slurring speech (p. 373).

From chronic alcoholism, a knowledge of the habits of the patient, and the retention of the pupillary light-reflex, are points which may guide the observer.

The examination of the cerebro-spinal fluid by the Wassermann reaction is more likely to throw light upon the differential diagnosis than any other method. This is a complicated process, but in the hands of competent persons is of assured

value. Mott[1] has stated that a positive reaction to the Wasserman test, in the cerebro-spinal fluid, was present in 89 per cent. of his cases; that the reaction was less constant in cases of tabes, and that it was often negative in syphilitic meningitis and diffuse cerebral syphilitic lesions. In all the reaction of the blood serum, may be positive. The reaction is probably not present in any other diseases.

Prognosis. The average duration of the disease is about three years; rarely do these patients live more than five. Many general paralytics however die, in the ordinary course of their malady, well under three years. Their tenure of life is, moreover, very uncertain: as death may result in consequence of an epileptiform seizure, and there is great liability to intercurrent complications. On the other hand, there are cases which run a rapid course of a few months, and die either from the intensity of the mental symptoms, or from intercurrent pulmonary complications. In the ordinary course death takes place from inanition, bedsores, cystitis, pulmonary troubles, and sometimes from status epilepticus.

Treatment. In the early stages an energetic course of antisyphilitic treatment, followed by general tonic remedies, is important in cases which have not been previously treated for syphilis.

The patient should be removed from work and advised to live a quiet life in the country. He should be protected from the consequences of ill-balanced action or erratic procedures.

In many cases it is necessary to certify and send him to an asylum; or, at all events, to have him under care and supervision in a sanatorium.

Juvenile general paralysis

This condition is intimately associated with inherited syphilis, and occurs with equal frequency in both sexes. It commences between the ages of eight and twenty years, but not infrequently engrafts itself upon an already defective nervous system.

The *morbid anatomy* is similar to that described in the adult form, with, in addition, signs of congenital syphilis in other parts of the body.

[1] Mott, *Lancet*, 1909.

As a rule the *symptoms* develop gradually. The mental changes commence with depression, exaltation, or perversion, and tend to progress to a state of complete dementia. Grandiose ideas, delusions, and outbursts of acute excitement may occur, but are less common than in the adult form. On examination the patients invariably present the features of congenital syphilis. They are small and poorly developed, with absence of pubic hair, and ill-defined breasts and testicles; scarring about the angles of the mouth, peg-shaped teeth, interstitial keratitis, and choroidal atrophy. The pupillary light-reaction is absent, and the deep reflexes are in some brisk; in others, abolished. They present the characteristic tremor of the face, tongue, and limbs already described, and the articulation is slurring. As in juvenile tabes, optic atrophy is a common and sometimes an early sign.

The *prognosis* is bad, as the disease usually terminates fatally in two or three years; but in some cases life may be prolonged for five or six years. There is progressive mental and physical weakness, which renders the patient liable to any intercurrent affection.

Chapter III

CHRONIC ATROPHIC PARALYSES OF SPINAL AND BULBAR ORIGIN

Under this title are grouped and described three closely allied disorders—progressive muscular atrophy, amyotrophic lateral sclerosis, and bulbar paralysis. The difference in their clinical features depends chiefly upon the seat of the lesion.

1. *Progressive muscular atrophy* is a malady characterised by progressive wasting of certain muscles, or groups of muscles, unaccompanied by sensory changes, and without obvious evidence of lesion of the upper motor neurone. It is probable, however, that few pure cases of this type exist, as almost invariably some coexistent alterations in the reflexes, and slight spasticity, may be found associated pathologically with early degenerative changes in the pyramidal system.

2. *Amyotrophic lateral sclerosis.* The symptoms consist primarily of muscular wasting—commonly of the distal portions of the upper limbs—without sensory impairment, and progressive spastic paraplegia, in which the upper limbs also participate. They are due to degenerative atrophy of the

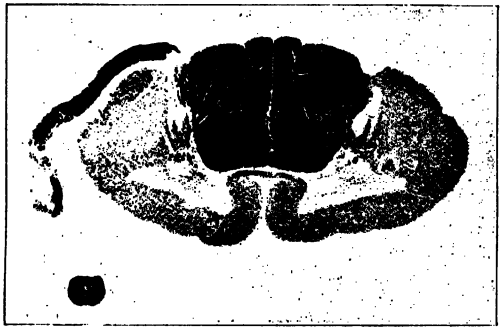


FIG. 122.

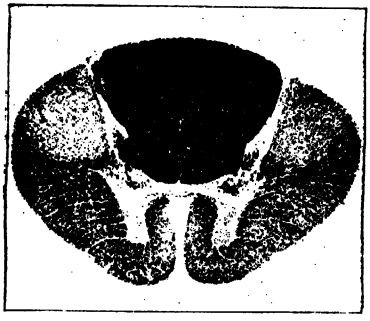


FIG. 123.

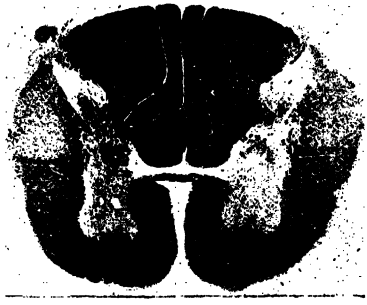


FIG. 124.

FIGS. 122-124.—These figures illustrate the pathological changes in the spinal cord from a case of amyotrophic lateral sclerosis. The posterior columns are normal; an extensive degeneration is seen in the antero-lateral columns, and especially in the crossed pyramidal tracts. The anterior horns are wasted.

ganglion cells of the anterior horns, along with well-marked sclerotic changes of the pyramidal fibres. Most of the cases of amyotrophic lateral sclerosis develop bulbar symptoms, as the result of similar atrophic changes in the ganglion cells of the bulbar nuclei.

3. *Bulbar paralysis* is characterised by muscular wasting,

commencing primarily in the muscles supplied from the bulbar motor nuclei. In this type the atrophic changes go on to affect the anterior cornual regions of the spinal cord, and the pyramidal implication is early and well marked.

A family form occurring in children (Werdnig-Hoffmann) is described on p. 471.

PROGRESSIVE MUSCULAR ATROPHY

Pure cases of this form of chronic atrophic paralysis are rare. Males are affected more frequently than females, and in both sexes the age at onset is usually after thirty.

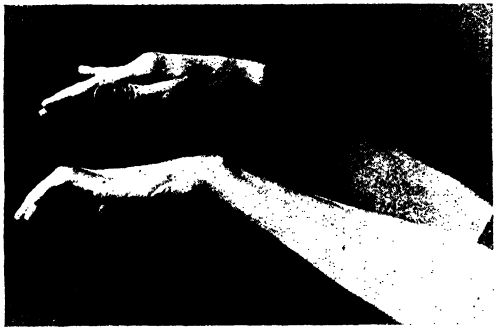


FIG. 125.—Showing atrophy of the small muscles of the hands in an early case of progressive muscular atrophy.

Symptoms. The onset of the disease is insidious: the patient's attention being first drawn to the condition by the atrophy of the small muscles of the hands. In the early stages the patient notices that his hands are getting thinner before he is conscious of any loss of power. As the atrophy progresses, weakness of the hands develops—especially for holding objects with the thumb and fingers. The thenar and hypothenar eminences waste, and the metacarpal bones stand out prominently owing to the atrophy of the lumbricales and interossei. Fibrillary tremor may be observed by the patient, or may give rise to a sensation of 'fluttering under the skin.' As time goes on the muscles of the forearm are involved, and later those of the shoulder, so that the patient is unable to raise his arms above his head. If the disease progresses, the neck, muscles, and those of the lower limbs,

may atrophy and the patient become bedridden in from nine to eighteen months. In pure cases, however, the extension of the disease is slow, and the atrophy may remain limited to the small muscles of the hand alone. In cases where the atrophy is extensive, complaint may be made of dull aching pain in the limbs, due to the extra strain on the weakened muscles. This pain is not attended by any muscular hyperæsthesia nor by objective sensory changes. Some idea of the rate of progress may be obtained by observing the distribution of the fibrillary tremor which, as a rule, precedes the onset of the atrophy; thus in any case where such tremor is widespread the prognosis is most unfavourable.

FIG. 126.—Illustrates the appearance of the shoulders and arms in a case of progressive muscular atrophy.

The *mental condition* is unimpaired.

The cranial nerves. These are not affected except in the advanced cases when the bulbar nuclei have become involved, and atrophy of the muscles supplied by the spinal accessory nerve — trapezii and sterno-mastoids — and later of the tongue and palatal muscles ensues.

The motor system. The muscles most commonly affected are the small muscles of the hand, the muscles of the shoulder and upper extremities, neck, lower limbs, and trunk. Fibrillary tremor is a constant and characteristic feature, and the electrical reactions show all stages of the reaction of degeneration, from mere diminution of faradic excitability to the fully developed reaction of degeneration. On striking the muscle a slow and prolonged contraction takes place. It is characteristic of the malady that in the early stages the muscular atrophy is out of all proportion to the loss of power, which is not complained of until the wasting of the muscles is well advanced. The loss of power

is always less than the appearance of the affected muscles would suggest.

Sensory system. Subjective sensations of aching in the limbs, and of 'fluttering' under the skin, are sometimes complained of; but these sensations are due to the weakness of the muscles and the presence of fibrillary tremor. There is no objective loss of sensibility.

Reflexes. The deep reflexes disappear concurrently with the muscular atrophy, but may remain unaffected in quality, though reduced in quantity, as long as normal muscle remains. Where the muscles are not wasted the deep reflexes remain normal or are slightly increased. Exaggeration of the deep reflexes and clonus only occur when the pyramidal tracts are involved—that is, in cases of amyotrophic lateral sclerosis.

The superficial reflexes remain normal in all pure cases of progressive muscular atrophy; but where pyramidal affection develops, the abdominal and epigastric reflexes become diminished, and the plantar reflexes become extensor in type.

Sphincters. There is no affection of the sphincters in the pure cases.

Clinical types. (1) The common type. The wasting commences in the small muscles of the hands, and may either extend to and involve other muscles, or remain more or less limited to the small muscles.

(2) A type in which the atrophy first affects the muscles of the shoulder girdle and upper arm. This is comparatively rare, and is seen generally in persons who have been exposed to some chronic intoxication—such as lead. (Fig. 126.)

(3) A type in which the muscles of the lower extremities are first affected.

Differential diagnosis. Progressive muscular atrophy has to be distinguished from—

1. The *muscular dystrophies* (p. 45) by:
   - (*a*) The absence of any family history.
   - (*b*) The age at onset.
   - (*c*) The distribution of the atrophy which is not that of any of the characteristic types of myopathy.
   - (*d*) The presence of fibrillary tremor.

(*c*) The presence of the reaction of degeneration on electrical examination.

2. *Peroneal muscular atrophy* (p. 472) by:
   (*a*) The absence of any family history of the disease.
   (*b*) The age at onset.
   (*c*) The distribution of the atrophy which tends to involve groups of muscles other than those affected in peroneal muscular atrophy.
   (*d*) The absence of sensory changes.
   (*e*) The progress and extension of the disease.

3. The *muscular wasting associated with cervical ribs* (p. 110) by:
   (*a*) The bilateral distribution of the atrophy.
   (*b*) The absence of sensory changes.
   (*c*) The progressive extension of the atrophy.
   (*d*) The negative result of an X-ray examination.

4. *Lesions of the peripheral nerves* (p. 98) by:
   (*a*) The absence of a local or general cause.
   (*b*) The absence of pain or sensory loss.
   (*c*) The distribution of the paralysis, which is not according to root or nerve lesion.
   (*d*) The absence of trophic changes other than muscular.

4. *Arthritic muscular atrophy* (p. 99) by:
   (*a*) The absence of arthritic changes.
   (*b*) The distribution of the atrophy which is not related to any joint, and the presence of R.D.
   (*c*) The evidence of intramedullary changes.

5. *Craft palsies* (p. 513) by:
   (*a*) The absence of a history of employment in an occupation likely to cause a toxic or pressure paralysis.
   (*b*) The absence of paræsthesia.
   (*c*) The absence of evidence of lesion of a peripheral nerve.

Prognosis. This depends on the rate of development of the atrophy. If after six months the atrophy is still limited to the muscles originally affected, the progress of the disease is likely to be slow, although no recovery of the wasted muscles will occur.

The prognosis is bad if there be along with the muscular atrophy much fibrillation in other muscles.

In the rapid cases death usually occurs in from twelve

months to two years; but in the more chronic cases life may be prolonged for five or fifteen years, provided that bulbar symptoms do not supervene and that serious pulmonary complications are avoided.

Treatment. The most valuable medicinal remedy is the hypodermic injection of strychnine ($\frac{1}{50}$ grain to $\frac{1}{15}$ grain). This should be supplemented by general tonic treatment, fresh air, and the avoidance of fatigue—more especially of the

FIG. 127.—Illustrates the appearance of the hands in an early stage of amyotrophic lateral sclerosis.

affected limbs. Treatment should also be directed to any conditions which may be possible causal factors. The local treatment consists of galvano-faradism and massage of the atrophied muscles. This ought to be applied short of fatigue. Although most cases are progressive, yet the employment of treatment, as above described, may lead to an arrest of the disease.

AMYOTROPHIC LATERAL SCLEROSIS

In this condition there is, in addition to the degeneration of the anterior horn cells, a sclerosis of the pyramidal system extending up to the cortex cerebri.

Symptoms. The symptoms vary at the outset according

to whether the anterior horns or the pyramidal system are first affected. Generally both are involved simultaneously, so that a condition of atrophic paralysis in the small muscles of the hands is present coincidently with a spastic paresis of the lower limbs. As the disease progresses, and it is essentially a progressive disorder, the spasticity of the lower limbs increases and the wasting extends to other muscles

FIG. 128.—Illustrates the appearance of the hands in an advanced case of amyotrophic lateral sclerosis.

of the upper extremities. At this stage the arms are wasted and useless, and walking is difficult owing to the weakness and spasticity of the legs. Still later, bulbar symptoms arise and the patient has difficulty in swallowing and in articulation, and the muscles of the lower limbs commence to atrophy, so that the spastic paralysis gives place to atrophic flaccid paralysis.

The condition of the atrophied muscles is similar to that seen in progressive muscular atrophy; but weakness and spasticity are found in those which are not atrophied.

The atrophy involves specially the small muscles of the hand and those of the arm, shoulder, and neck; then the bulbar muscles and those of the lower limbs.

Sensory system. There is no disturbance of the sensory functions beyond painful reflex spasms in the legs, aching

pain in the upper limbs due to the muscular atrophy, and the sensation due to the fibrillary tremor.

Reflexes. The deep reflexes are increased, and clonus always develops in the later stages.

The superficial, epigastric, and abdominal reflexes are abolished, and the plantar reflexes are extensor in type.

The sphincters are slightly affected; some delay in micturition may take place, especially when the patient is

FIG. 129.

FIG. 130.

FIGS. 129 AND 130.—Two photographs showing the facial appearance on laughing, and on attempts to protrude the tongue, in bulbar paralysis.

bedridden; but there is never incontinence of urine. Constipation may become obstinate.

Clinical types. In some cases the development of the disease is slow, and the patient may live from three to five years. It may commence with atrophy of the small muscles of the hand or with spastic weakness of the legs, but eventually both these conditions are found in association with each other.

In other cases the progress of the disease is rapid with widespread muscular wasting and bulbar paralysis. The cases in which the bulbar nuclei are involved end fatally in from ten to eighteen months.

Bulbar Paralysis and Chronic Ophthalmoplegia

The pathological basis of both these conditions is similar to that of progressive muscular atrophy and amyotrophic lateral sclerosis, and differs only in the early implication of the cranial nerve nuclei.

In the bulbar cases it is rare for the disease to remain limited to the bulbar nuclei, as it tends to involve the

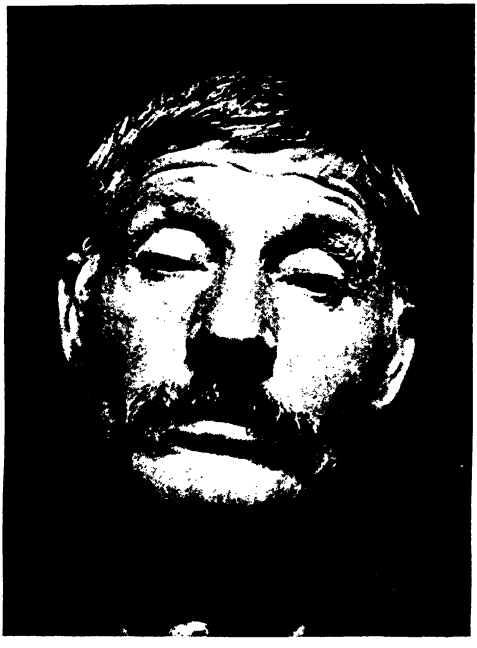


Fig. 131. The face in a case of complete nuclear ophthalmoplegia. Observe especially the partial double ptosis, the overaction of the frontalis, and tilting back of the head.

pyramidal system and the anterior horn cells in the spinal cord.

Symptoms. These are wasting and degeneration of the muscles supplied from the bulbar nuclei, wasting and atrophy of the tongue, which cannot be protruded, atrophy of the palatal and pharyngeal muscles, and often of the upper part of the trapezius and sterno-mastoid muscles, and sometimes of the face.

In some cases the disease affects the upper cranial nerve

nuclei, giving rise to a chronic ophthalmoplegia, often limited to the external muscles of the eyes, but sometimes affecting the pupillary muscles as well. The paralysis may remain limited to the ocular muscles, or may extend to the bulbar and spinal muscles. In rare cases the ocular muscles are involved secondarily to bulbar or spinal paralysis.

Differential diagnosis. Amyotrophic lateral sclerosis and bulbar paralysis have to be distinguished from—

1. *Intramedullary lesions* (tumour, syringomyelia) (p. 328), by :
   - (*a*) The absence of pain.
   - (*b*) The bilateral and symmetrical progress and distribution of the atrophy.
   - (*c*) The absence of sensory loss.
   - (*d*) The absence of trophic changes other than muscular.
   - (*e*) The non-involvement of the sphincters.
2. *Extramedullary lesions affecting the cord* or spinal roots (p. 350), by :
   - (*a*) The absence of root pains.
   - (*b*) The distribution of the paralysis which is not that of root paralysis.
   - (*c*) The absence of sensory loss.
   - (*d*) The absence of a definite segmental level of paralysis, sensory or motor.
   - (*e*) The course and spread of the disease.
   - (*f*) The non-involvement of the sphincters.

Bulbar palsy and chronic ophthalmoplegia have to be distinguished from—

1. *Myasthenia gravis* (p. 485), by :
   - (*a*) The absence of remission and relapse of symptoms.
   - (*b*) The presence of atrophy with relatively slight loss of motor power, whereas in myasthenia the loss of power is great with little or no atrophy.
   - (*c*) The absence of the myasthenic reaction and the presence of the reaction of degeneration.
   - (*d*) The presence of signs of lesion of the pyramidal tracts.
2. *Double hemiplegia* (p. 188), by :
   - (*a*) The absence of a history of a stroke or strokes.
   - (*b*) The presence of atrophy with the reaction of degeneration.

The **prognosis** in cases of bulbar paralysis is unfavourable, some cases ending fatally in six months from failure of the respiratory and cardiac centres. Few cases survive more than three years. Where the paralysis is limited to the ocular muscles, the prognosis as regards life is not so unfavourable, many cases living for ten to fifteen years after the onset of the symptoms.

Treatment. This is similar to that described under progressive muscular atrophy; but in addition means ought to be taken to allay spasticity and to prevent contractures. These objects are best attained by the employment of local hot-air baths, followed by massage and passive movements. Walking should be permitted for periods well within the limits of fatigue. The chances of arrest of the disease are less hopeful.

In the bulbar forms special attention has to be given to feeding, semi-solid food being swallowed more easily than liquid or solid food. Care should be taken in the later stages to prevent the passage of food into the larynx, nasal feeding sometimes being necessary. Pulmonary and cardiac complications need careful attention.

Chapter IV

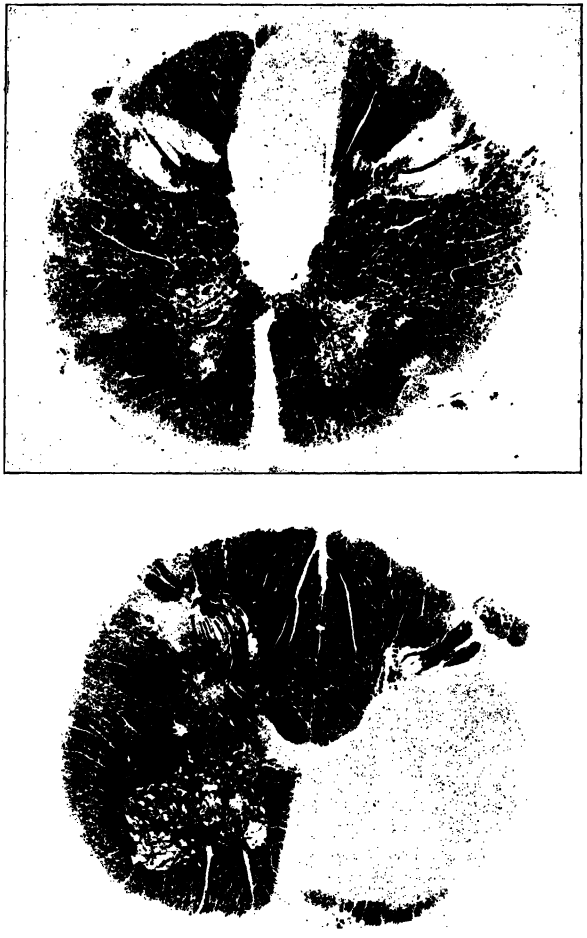
DISSEMINATED SCLEROSIS

(SYN.: SCLÉROSE EN PLAQUES—MULTIPLE CEREBRO-SPINAL SCLEROSIS)

This is a chronic progressive disease, characterised pathologically by the presence of patches of sclerosis situated at random throughout the central nervous system, and clinically by paræsthesia, spastic paralysis, interference with the action of the sphincters, tremors on volitional effort, articulatory disability, and sometimes by optic atrophy and other cranial nerve palsies.

Etiology. This disease commonly commences in early adult life, but may arise at any age from twenty to forty-five. Its occurrence in children has been described, but it is doubtful whether these have been cases of the classical disease as seen

in young adults. Out of three hundred cases which we have personally observed there were only two in which the symptoms



FIGS. 132 and 133 are two sections of the spinal cord from a case of disseminated sclerosis. In Fig. 132 the patch of sclerosis occupies the posterior columns; in Fig. 133 the patch is in the antero-lateral portion of the cord on the right-hand side, involving both white and grey matter.

commenced before the sixteenth year, one case at fourteen and one at fifteen. Its onset after fifty is rare.

It occurs rather more frequently in males than in females.

There is little that is certain known about its causation. It has no relation to syphilis.

Certain conditions, however, such as pregnancy, fatigue, and acute pyrexial disorders, have a deleterious influence upon the developed malady.

Pathological anatomy. On palpation of the cord, the surface presents irregular hard lumps which may be felt by the finger. To the naked eye, when sections are made through the brain or spinal cord, irregularly shaped and diffusely scattered patches of various sizes of a pinkish and semi-translucent appearance are seen. These are well defined, present a sharp edge, and stand out clearly from the adjacent nerve tissue. They are most common in the spinal cord, the mid and hind brain, and the centrum ovale, but may be found in any part of the central nervous system. They affect indiscriminately both the grey and the white matter. Similar patches have been observed in the optic and spinal nerves.

The chief changes noticed on microscopical examination of a sclerotic patch are: the absence of the myeline sheaths of the nerves, the retention of normal or perhaps somewhat atrophied axis-cylinder processes, and the presence of a dense network of neuroglial fibrils with spider-cells and nuclear infiltration. The retention of the axis-cylinder processes and their normal reaction to the usual staining reagents are the characteristic pathological features of the disease. There may be no signs of Wallerian degeneration of the tracts either above or below the sclerotic areas, a fact which is probably explained by the retention of the axis-cylinders of the nerve fibres. Should a patch of sclerosis implicate the grey matter, the ganglion cells may undergo some degenerative alteration, but such changes are of slow development.

In the advanced stages of the disease large areas of sclerosis are observed, in some places throughout the whole cross-section of the cord; in other places small islets only of apparently normal nerve tissue may be detected within the areas of sclerosis. In the vast majority of cases the transition between the normal tissue and the sclerosed area is quite abrupt. Vascular changes are not characteristic, but sometimes hyaline degeneration of the vessel walls is present. At the borders of recent patches compound granular cells are sometimes found. (Figs. 132 and 133.)

No satisfactory explanation to account for the formation of the disseminated patches has yet been given. Three theories have been advanced: first, that the condition is due to a congenital defect in the development of the myeline sheaths in certain localities; secondly, that the sclerosis is primarily of vascular origin; and, thirdly, that the disease is an inflammation of the neuroglial tissues, which destroys the myeline sheaths around the axis-cylinders.

Symptoms. The symptoms necessarily depend, in part upon the situation of the lesions, and in part upon the number of the islets of sclerosis. In some cases the spinal cord may be the primary seat of the disease, when the early symptoms are more or less paraplegic in type; in others the lesions may primarily be limited to the brain, with monoplegic or hemiplegic symptoms; in others again the cranial nerves may be affected, the early symptoms being diplopia from oculo-motor paralysis or loss of sight from optic nerve atrophy. It is obvious, therefore, that great variability and irregularity may characterise the onset of this disease; but in the later and terminal stages the clinical picture is much the same, in whatever way the disease commences.

A tendency to remission and relapse of symptoms is a highly characteristic feature of this malady, the relapse not necessarily occurring in the same part of the body as that originally affected. Thus the first symptom may be an oculo-motor palsy, which disappears, and is followed later on by paraplegia or sphincter trouble.

The mental condition. Most cases, especially those originating in early adult life, exhibit a peculiar and more or less characteristic mental attitude. There is some mental dullness, the temperament is peculiarly cheerful, combined with a tendency to slight childishness, and an abnormal optimism as to the progress of the malady. The facial expression is vacant or blank, but may be broken in upon by smiles and laughter aroused by trivial circumstances.

Special senses. Smell, taste, and hearing are rarely affected. Impairment of vision (amblyopia) may be an early symptom. The following varieties are found: (1) amblyopia over the whole field with contraction of the visual area; (2) central scotoma. Both of these conditions may, or may not, be associated with optic atrophy, and may be confined to

one eye. The onset of these symptoms may be sudden or gradual. In cases where the onset is sudden, remissions frequently take place. (3) Hemianopsia and quadrantic loss of vision may be observed in other cases, as a result of sclerotic patches in the primary or higher visual tracts. Vision is rarely completely lost.

Optic atrophy. Pallor of one or both optic discs is present in about 50 per cent. of the cases when the disease is fully developed.[1] The colour of the disc is usually a papery white, the pallor being more pronounced upon the temporal side of the disc. The atrophied disc never shows the grey tint which is so characteristic of tabetic atrophy, and the blood-vessels of the fundus stand out clearly, and rarely present signs of inflammatory or vascular lesion.

Papillitis. In some of the more acute cases, especially those with sudden onset of impairment of vision, a mild degree of papillitis may be observed suggestive of a retrobulbar neuritis. There is no evidence that it is an inflammatory lesion. It is stated by Uhthoff to occur in 5 per cent. of the cases. The condition is transitory and gives place to optic atrophy, with temporary or permanent loss of central vision.

Oculo-motor nerves. The pupils are either of medium size or large. Their reaction to light is not impaired, except in those cases in which optic atrophy is present.

Diplopia, most commonly resulting from paralysis of the sixth nerve, is sometimes seen, and is often an early and transient symptom. Inquiry should always be directed as to antecedent diplopia. Occasionally the third and fourth nerves may be affected.

Nystagmus, especially on lateral movement, is a very characteristic sign. Although commonly bilateral, it may be present on one side only. The movements may be fine or coarse, and are often quite irregular. In some cases nystagmus is absent, but in other cases it is spontaneous, especially when there is optic atrophy.

Affections of the other cranial nerves are rare, but spastic weakness and inco-ordination of the muscles supplied by the seventh, ninth, tenth, and twelfth nerves give rise to defects of articulation, and are not uncommon in the fully developed

[1] Buzzard (Thomas), *Simulation of Hysteria by Organic Disease*, 1891.

disease. 'Nystagmus' of the palate and of the vocal cords has been observed.

Speech defects are entirely articulatory, and give rise to the 'staccato,' scanning or syllabic utterance, which, though a classical sign, is of rare occurrence. Minor degrees of articulatory impediment are, however, not uncommon, especially indistinct pronunciation of the explosives, b, d, g, and a monotonous voice, in which the syllables are uttered hesitatingly and slowly.

Motor system. The motor affections vary in degree and extent, are often transitory, but tend to progress. The paresis, or paralysis, is as a rule one of movements rather than of muscles, and of the upper neurone type, namely spasticity without muscular atrophy or changes in the electrical reactions.

In the early stages and in slight cases spasticity may be almost negligible; but in the severe cases may be profound, and lead to flexor and adductor contractures of the lower limbs. Another feature of the motor affection is the readiness with which fatigue sets in.

Muscular wasting from affection of the anterior cornual region occurs in a very small percentage of cases.

The characteristic 'intention' or 'action' tremor, which is one of the classical signs of the malady, is almost invariably present in greater or less degree; and as a rule the resulting unsteadiness is the chief disablement of the upper limbs. It is static in character, and may also be seen in the head and trunk as well as in the limbs. It may, indeed, be present under all circumstances, except that of complete rest in bed. In its minor degrees it may be demonstrated by asking the patient to approximate the points of his fingers or to touch the point of his nose; and in contradistinction to cerebellar ataxy, the unsteadiness and movements are most marked when the ultimate object is achieved. The cause of this tremor is quite obscure, but there is no evidence to support the view that the motor impulses, which are directed along fibres whose axis-cylinders are denuded of their myeline sheaths, are retarded in their passage through the cord. It is more likely that it is due to interference with the efferent cerebellar tracts and their connexions.

The gait may be spastic-paretic in character, the toes

being scraped along the ground in walking. On the other hand, an element of instability is frequently superadded, giving rise to the spastic-ataxic gait, which is so commonly seen in this disease.

Sensory system. Subjective symptoms are rarely absent at one period or another. Giddiness, paræsthesia, numbness over the limbs, body or face, feelings of heaviness and weight in the limbs are common. None of the above are painful. The only painful symptoms to which such patients are liable are those arising from muscular spasms of the limbs. Girdle and lancinating pains, such as are common in tabes, are strikingly absent in disseminated sclerosis. Headache is rare, except in the terminal stages, when it may be associated with obstinate constipation, vomiting, and sometimes hæmatemesis.

Objective sensory signs are slight and rare compared with the frequency of the subjective symptoms. Careful examination, however, will seldom fail to detect slight relative losses of cutaneous sensibility in the paræsthetic areas, more especially to cotton wool, the finer degrees of heat and cold and tactile discrimination, although all forms of sensibility may be slightly impaired. In rare cases hemianæsthesia with hemiplegia may be present. Segmental losses of sensation are practically unknown.

Reflexes. The deep reflexes are never absent; on the contrary they are as a rule increased, and where motor weakness is present, they may be greatly exaggerated, and clonus easily obtained.

The superficial reflexes are lost or diminished over the epigastrium and abdomen. This is one of the most characteristic and definite signs of the malady. As their loss depends upon an interference with the fibres of the pyramidal system above the corresponding segmental levels, they will still be elicited until such interference has occurred. Hence, although their presence does not exclude the existence of disseminated patches elsewhere, their absence indicates involvement of the pyramidal tracts.

The plantar reflex is extensor in type in all cases where spastic weakness is present. As a rule it is an early sign, and may persist during a period of remission, when other symptoms have disappeared. Occasionally in the early stages the

response may be flexor. If the response is indefinite on one side, and the corresponding abdominal reflex is absent, the significance is that of an extensor response.

Sphincters. Some degree of hesitancy in passing water is an early symptom, especially in males; in females the difficulty is in retaining it. It is not a troublesome symptom until the later stages. Constipation is very common; in the

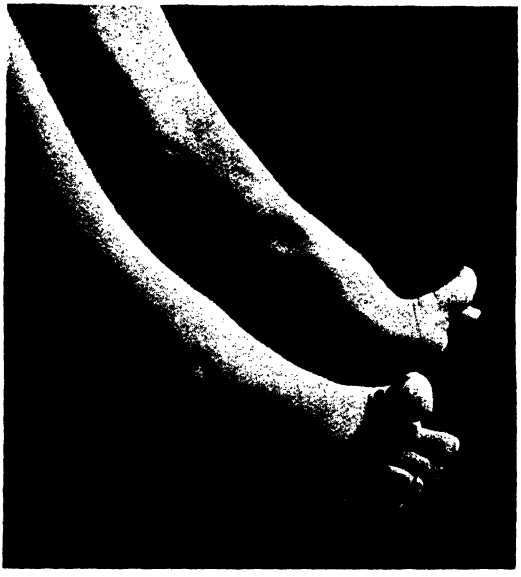


FIG. 134.—Photograph illustrating permanent hyperextension of the great toes in a case of disseminated sclerosis.

later stages this may give rise to copræmia, which may be the cause of death.

The *sexual* functions are usually impaired.

Trophic disorders are not obtrusive, but vaso-motor disturbances, such as sweating and œdema, occasionally occur. Bed-sores may develop in the later stages. The nails may become brittle.

Clinical types. 1. *Remittent type.* This type is characterised by the onset of slight symptoms, such as numbness, motor weakness, diplopia, temporary loss of vision, and sphincter trouble. Often the onset may be determined by a shock, illness, or accident, and, as the patient recovers

apparently completely, the symptoms are attributed to hysteria. The recurrence of symptoms may be postponed for an indefinite period, even up to five or ten years. As a rule the remission period becomes shortened, the attacks last longer, and eventually permanent paralysis develops, and the typical manifestations of the disease ensue.

2. *Paraplegic type.* In this variety the early symptoms are referred to the lower limbs, complaint being made of heaviness, numbness, and weakness of the legs. The toes tend to scrape the floor, flexor spasms of the legs give discomfort at night, and there is difficulty in passing water. Gradually the symptoms increase, and the typical form evolves with symptoms referred to implication of the higher portions of the nervous system. In such cases inquiry should be made as to a previous history of diplopia and transitory paresis, which are not connected in the patient's mind with his illness. The existence of nystagmus in these cases is highly suggestive of disseminated sclerosis.

Although the motor and sphincter paralysis may be well marked, segmental loss of sensation, as in compression paraplegia, is rare.

3. *Hemi- and monoplegic types.* In these the symptoms are of sudden onset and affect one limb, a portion of a limb, or one side of the body. They are frequently transient, and, as in the remittent type, may be suspected of being hysterical.

4. *Cerebellar type.* The early occurrence of patches in the cerebellum, or its peduncles, may give rise to cerebellar symptoms without any affection of the motor or sensory paths. The symptoms are mainly hemiataxy and tremor, without characteristic changes in the reflexes. The recognition of this type can only be made by the further progress of the disease.

In whichever of these ways the disease may commence, *the terminal stages* present similar features: nystagmus, pallor of the discs, articulatory speech defects, spastic paresis or paralysis with 'action' tremor, either universal or confined to certain portions of the body, some degree of ataxia and unsteadiness, loss of sphincter control, incontinence of urine and obstinate constipation, increased tendon reflexes with clonus, absence of the abdominal and epigastric reflexes, and

slow typical extensor plantar responses, and subjective sensory symptoms and diminution of all or some forms of cutaneous sensibility in irregular areas. In this condition the patient may remain bedridden for a number of years, and in the absence of bedsores or intercurrent maladies the general health may be well sustained.

Differential diagnosis. It is only in the early stages of the disease that a difficulty is likely to arise in diagnosis, and more particularly in the differential diagnosis from *Hysteria*. The onset of both maladies during early adult life, the tendency shown by both to remission and relapse of symptoms, and the existence in both, even at the outset, of paraplegic symptoms, often accompanied by tremor on volitional effort, present an ample field for error.

Difficulty may also be experienced and the possibility of error considerable in the diagnosis of disseminated sclerosis from *Intracranial Tumour*, situated beneath the tentorium. In all these disorders nystagmus, inco-ordination of movement, and instability may be early and well-marked symptoms.

It is also important to distinguish the paraplegic type of disseminated sclerosis from *Paraplegia*, which is a symptom of many other conditions, such as that arising from the pressure of tumours within the spinal canal, the so-called primary lateral sclerosis, and the 'ataxic paraplegia' of subacute combined sclerosis.

The chief points in the differential diagnosis are given in the table on p. 428.

Prognosis. Disseminated sclerosis is a progressive disease, terminating in paraplegia. Death in the majority of cases supervenes upon complicating conditions, such as cystitis and pyelonephritis, bedsores, copræmia, and pulmonary disorders. The duration of life varies within considerable limits, even after the disease has been thoroughly established.

Cases in which sphincter paralyses are absent or only slight have been known to last for many years.

The marked tendency towards a remission of symptoms, particularly in the early stages, enables a more favourable outlook to be given in those cases in which a more or less complete disappearance of the symptoms takes place. In the remitting type of the malady, the intervals of remission diminish in duration with each relapse, until the time comes

TABLE GIVING POINTS IN DIFFERENTIAL DIAGNOSIS BETWEEN DISSEMINATED SCLEROSIS, HYSTERIA, EXTRA-CEREBELLAR TUMOURS, COMPRESSION PARAPLEGIAS, SUBACUTE COMBINED SCLEROSIS, AND CEREBRO-SPINAL SYPHILIS

	DISSEMINATED SCLEROSIS.	HYSTERIA.	EXTRA-CEREBELLAR TUMOURS.	COMPRESSION PARAPLEGIAS.	SUBACUTE COMBINED SCLEROSIS.	CEREBRO-SPINAL SYPHILIS.
Age at onset	Early adult life	Variable	Adult life	At any age	From 45 to 65	Adult life, history of syphilis
Course of disease	Remitting and relapsing tends to progress	Remitting and relapsing	Slow and progressive	Slow and progressive May be sudden	Progressive	Remitting or progressive
Motor symptoms and gait	Paraplegic, monoplegic, and hemiplegic Temporary ocular palsies Gait frequently unsteady Spastic or ataxic	Paraplegic, monoplegic, and hemiplegic Gait not that of organic disease	Paralysis of limbs rare; and if present, late Ocular palsies rare Gait unsteady and of 'drunken' reeling type	Paralysis below level of compression	Ocular palsies rare Gait spastic and sometimes unsteady Paralysis bilateral (legs more than arms)	Paraplegia, hemiplegia, monoplegia
Sensory symptoms	Subjective common Objective rare and usually slight	Subjective rare Objective well marked, and characteristic, hemianæsthesias, or stocking and glove type	Sensation unaffected, except slight over face on one side: deafness present on side of lesion	Sensory loss below level of compression	Subjective well marked Objective at first in hands and feet, later of segmental type	Brown-Séquard symptoms Segmental below lesion
Vision and optic discs	Amblyopia, contracted fields, central scotoma Pallor of optic discs	Amblyopia, blindness, marked contraction of visual fields Discs never affected	If vision affected due to optic neuritis or secondary optic atrophy	Vision not affected Discs normal	Vision not affected Discs normal	Not usually affected Pupils may be inactive to light
Tremors	Intention tremors in head, body, and limbs	Intention, vibratory, and rhythmic tremors	On side of lesion ataxy without spasticity; on side opposite lesion spasticity	Tremor not present	May be some instability of arm movements, but no intention tremor	Tremor may be present
Nystagmus	Nystagmus equal on both sides	Never nystagmus	Nystagmus coarse to side of lesion, fine to the other	No nystagmus	Nystagmus not present	Nystagmus not present
Reflexes	Deep reflexes exaggerated Plantars extensor Abdominals absent True ankle clonus	Deep reflexes exaggerated Plantars absent or flexor Abdominals may be absent Pseudo-clonus of foot	Deep usually exaggerated Plantars may be extensor Abdominals may be absent Clonus may be present	Deep exaggerated Plantars extensor Abdominals absent below level of lesion Clonus usually present	Deep exaggerated in early, lost in late stages Plantars extensor Abdominals absent Clonus present in spastic stage	Deep exaggerated Plantars extensor Abdominals absent Clonus present
phi nc ters	Affected early	Never affected, but retention of urine may occur	Never affected until late stages	Affected with onset of paraplegia	Affected in middle and late stages	Affected early or late

when the phenomena of the malady persist. Periods of freedom from symptoms have been known to last for five or ten years. The prognosis, therefore, in any given case has to be based, partly upon the type of the malady and partly upon the absence of serious complicating features.

Treatment. Little, if any, benefit is obtained by medicinal remedies in this disease, although general and nerve tonics, such as strychnine, arsenic, and iron, may be prescribed with advantage. For the sphincter weakness, ergot and belladonna often prove of great value.

The most satisfactory means of treating the disease is by massage, passive movements of the limbs, and faradism. In the early stages, these in association with as much rest as possible and the avoidance of undue fatigue, are usually sufficient to materially relieve the symptoms. In the later stages, when tremor and instability are present in addition to motor weakness, rest in bed should be enjoined. Complications should be treated as they arise, and great care taken to avoid the occurrence of cystitis, bed-sores, or troublesome constipation. Ataxia may be treated by Frænkel's exercises.

Some types of the disease respond very satisfactorily to massage and electrical treatment. In these cases an occasional course of treatment is often of great value in delaying the onset of complications, or diminishing their intensity should they be present.

Pregnancy should be avoided, as the disease is almost invariably aggravated during the puerperal period.

Chapter V

SUBACUTE COMBINED DEGENERATION OF THE SPINAL CORD

This disease is characterised clinically by the onset of subjective and objective affections of sensation referred to the distal portions of the limbs, followed by signs of interference with the motor functions, segmental anæsthesias and sphincter paralyses, and terminating in flaccid paraplegia, loss of the

tendon reflexes, muscular atrophy, mental impairment, and death.

Etiology. It occurs both in men and women, but rather more frequently in the latter. It commences usually between the ages of forty-four and sixty-five, and is not infrequently associated with anæmia, either of the simple or pernicious

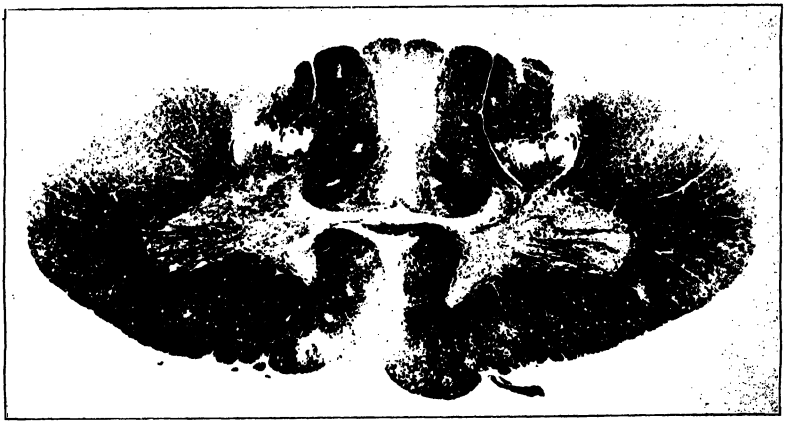


FIG. 135.—Section of the cervical region of the spinal cord from a case of subacute combined degeneration.

type. It is, however, far from clear that the anæmic state of the blood, to which attention has been drawn, is the direct causal factor. More probably the condition is due to some toxic influence, which acts both upon the blood and upon the nervous system, the latter suffering not only directly, but also from the impoverished condition of the blood.

Pathology. Few alterations are visible in the nervous system to the naked eye. In some cases there is an excess of cerebro-spinal fluid, associated with slight atrophy of the cerebral convolutions. On section of the spinal cord, areas of degeneration are seen especially in the posterior columns, around the periphery, and in the pyramidal tracts. The white matter around the central grey horns stands out clearly in contrast to the surrounding degeneration. These changes may be slight, but in severe cases are well marked in some segments and encroach more closely upon the grey matter. In most cases the spinal cord is pale and slightly atrophic.

Microscopically, the degeneration is usually of some standing; and although some nerve fibres stain with Marchi's osmium bichromate method, the degenerated areas are more clearly seen after staining with the Weigert-Pal hæmatoxylin stain.

The degeneration is always most marked in the posterior

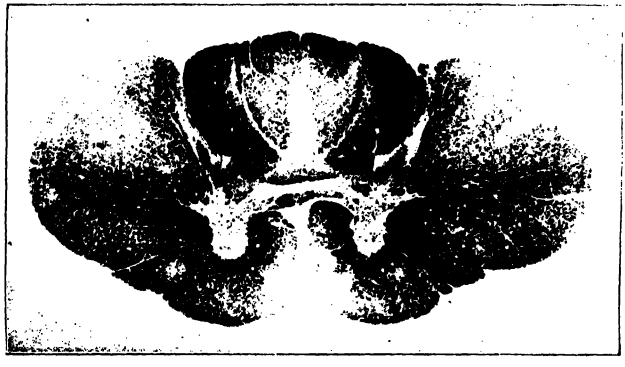


FIG. 136.

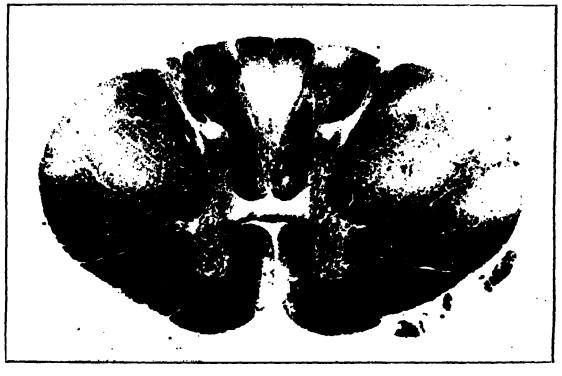


FIG. 137. See page 432.

columns and around the periphery of the cord. In old-standing cases the pyramidal tracts may be as much degenerated as the posterior columns. Stained by hæmatoxylin, fuchsin, and picric acid (Van Gieson) sections show an absence of inflammatory changes. The blood-vessel walls are healthy, but in some parts the arteries are engorged with blood, and in other parts they are empty; perivascular cell infiltration is also

absent. In the more acute cases, rounded hyaline-degenerated bodies, staining a purple colour, are seen in the periphery of the cord and along the course of the vessels, especially in the posterior columns. The neuroglial cells in these cases are swollen, often vacuolated, and occasionally degenerated; and as a rule there is proliferation of the neuroglial nuclei with

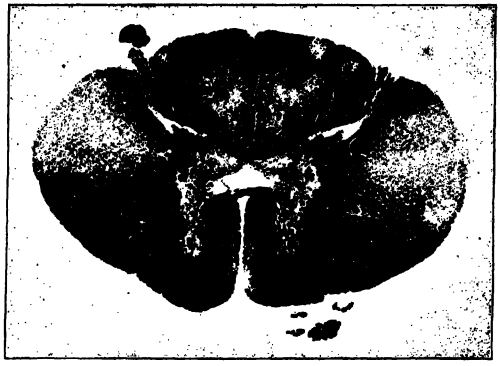


FIG. 138.

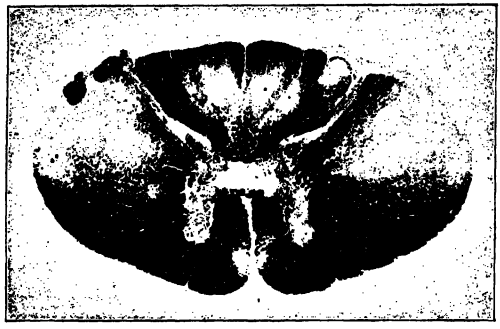


FIG. 139.

FIGS. 136 AND 139.—These sections illustrate the characteristic changes in the spinal cord from a case of subacute combined degeneration.

some swelling of the surrounding protoplasm. In more chronic types an over-growth of the neuroglia may be observed.

In the degenerated portions of the cord the myeline sheath of the nerve fibres is broken up into characteristic globules. In places the sheath has entirely disappeared and the axis

cylinder is exposed (Bielschovsky). The nerve roots are similarly degenerated, but to a less extent.

Alterations are also detected in the nerve cells, more especially in the cells of Clarke's column, which show a chromatolytic formation of the protoplasm. In the anterior cornual cells the changes are only slight, and when present,

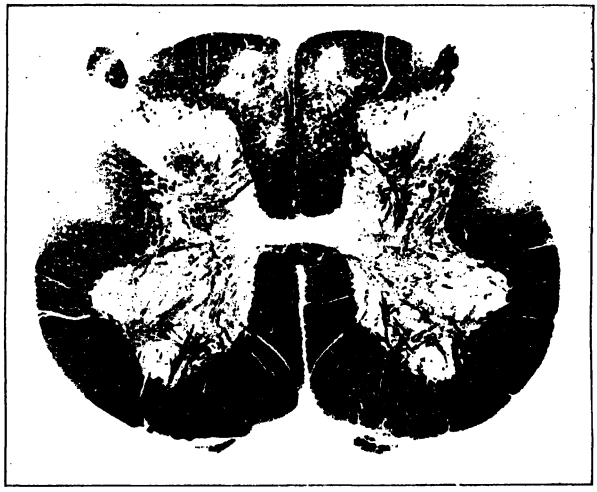


FIG. 140.—Section of the cord in the lumbar region from a case of subacute combined degeneration.

consist of an aggregation of yellow pigment around the periphery of the cell; and similar changes may be seen in the Betz cells of the cerebral cortex and in the Purkinjé cells of the cerebellum. These cell changes are always slight and by no means universal. On the other hand, the changes in the nerve fibres are out of all proportion to those seen in the nerve cells.

From the microscopical examination it would appear that the changes are neither inflammatory nor vascular in character, nor does any evidence support the view that the alteration is due to a primary affection of the nerve cells. It would seem rather as if a toxic influence was at work, first upon the neurone as a whole, resulting in degeneration of those portions of the fibre farthest removed from the trophic influence of the cell; and secondly upon the blood, which

becomes impoverished and leads to malnutrition of the nerve fibres and their cells.

Symptoms. Although the term 'subacute' was applied to this disease by those who originally defined its anatomy and its symptoms, further observation and the study of a larger number of cases have tended to broaden its conception.

The earliest symptoms are referred to the sensory system, and consist of paræsthetic sensations of numbness and coldness in the distal parts of the limbs. As a rule these symptoms affect the feet earlier and more severely than the hands, and are unaccompanied by any obvious circulatory derangement. At this period a slight diminution to all forms of sensibility is found when tested objectively.

The first stage may be prolonged for several years, or may temporarily pass away, to recur within a period of a few months to two years. More commonly, after lasting from three to eighteen months, it gradually merges into the second stage.

The patient now begins to feel, and to complain of, a sensation of heaviness and weakness of the legs and arms; and his friends may notice that his general condition is impoverished and his appearance is white and anæmic. Spasticity of the lower limbs gradually develops, its onset being heralded by the presence of flexor spasms and drawing up of the legs in bed, with stiffness and cramps in the muscles. This spastic state may increase slowly or rapidly, but usually within a few weeks or months paraplegia is so fully developed that walking is impossible.

During this (second) stage the general condition is one of ataxic paraplegia, not limited to the legs alone, but affecting also the hands and arms. Weakness of sphincter action is usually present some time during this period. The deep reflexes are exaggerated, and extensor plantar responses with absence of abdominal reflexes are always noticed.

Of the organic signs, the state of the abdominal reflexes and the plantar response may afford the earliest evidence of the affection of the motor system, and definitely precede the onset of obvious spasticity.

During this period also the objective sensory symptoms undergo a marked change. An examination reveals a loss of sensation up to a definite segmental level. Attendant on

the development of this segmental anæsthesia, paræsthetic symptoms of tightness, coldness, or numbness may be present. It is of interest to note that at the autopsy a greater local incidence of the pathological changes is found to exist in the segment corresponding to the upper limit of the anæsthesia. The development of these segmental symptoms is not

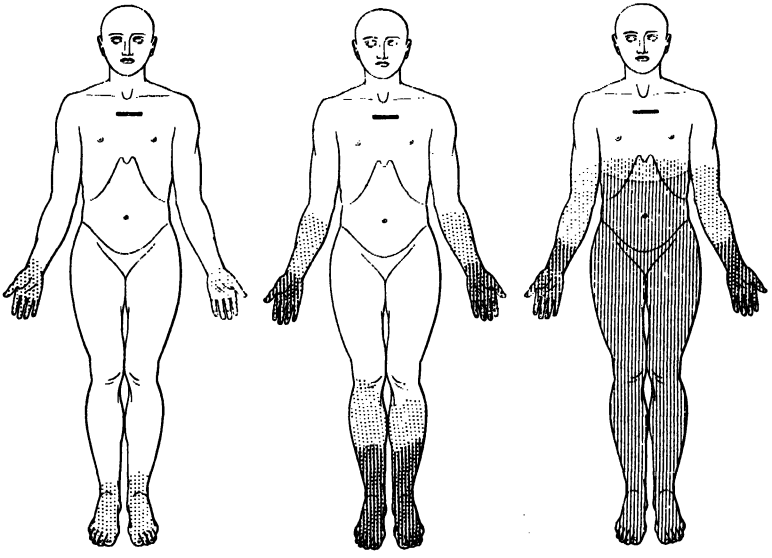


FIG. 141. FIG. 142. FIG. 143.

Three charts illustrating the sensory symptoms in subacute combined degeneration of the cord. Fig. 141 shows the distribution of the paræsthesia in an early stage of the disease (dotted areas). Fig. 142 shows the sensory loss confined to the distal portions of the limbs (shaded areas). Fig. 143 shows the sensory loss below a segmental level in the late stages.

infrequently attended by a rise of temperature of one or two degrees, and a decided deterioration in the general condition.

The occurrence of such indications of segmental lesion ushers in the final stage of the malady. Not uncommonly these exacerbations recur at increasingly higher levels; and the muscles below the level of the lesion begin to waste rapidly. In a few weeks the spastic paraplegia gives place to flaccidity with loss of all the reflexes, the extensor responses being retained when all the others have disappeared.

Loss of sphincter control becomes complete, and hemorrhage

from the bladder is not infrequent, and may be a terminal complication. Bedsores develop, the mental condition deteriorates rapidly, drowsiness, torpor, or mild delirium supervene, and death results from pulmonary, bladder, or renal complications, and general exhaustion.

Such in brief is the clinical history of a disease which has a very definite clinical and pathological basis, and by its recognition there has been brought into the clear light of knowledge one more of the ill-defined diseases which have sheltered under the clinical covering of 'ataxic paraplegia.'

Aberrant types of this condition are:—

1. Cases in which the first stage exists for over three years without any obvious evidence of organic change.

2. Cases in which the sensory symptoms are early and severe, and are accompanied by pains in the limbs. In these cases the spasticity may be overshadowed or absent, and the deep reflexes abolished in an early stage.

3. Cases in which spasticity may coexist with only slight subjective sensory symptoms for a long period. This is an especially frequent type in older people.

Diagnosis. This malady presents so many phases that, if seen for the first time in any one of its stages, it may readily be mistaken for any form of nervous disorder, organic or functional.

The first stage has to be distinguished from—

1. *Hysteria* (p. 522) by:
   (*a*) Its onset over the age of thirty-five years and the absence of any stigmata of hysteria.
   (*b*) The existence of anæmia of recent origin.
   (*c*) The development of an extensor plantar response.

2. *Acroparæsthesia* associated with atheroma, rheumatoid arthritis and the climacteric period, by the definite signs of organic disease, especially the extensor plantar response.

The second stage has to be distinguished from—

1. *Disseminated sclerosis* (p. 418) by:
   (*a*) The absence of nystagmus, or optic atrophy.
   (*b*) The age of the patient and a history of freedom from previous symptoms of nervous disease.
   (*c*) The course of the disease, the limitation of the paræsthesia to the distal extremities, and the development of segmental anæsthesia.

(*d*) The coincidental occurrence of severe anæmia.

2. *Syphilitic meningo-myelitis* (p. 374) by:
   (*a*) Absence of a history and the signs of syphilis.
   (*b*) The retention of the pupillary light-reflex, absence of cranial nerve palsies, and the occurrence of paræsthesia in the hands, above a segmental anæsthesia.
   (*c*) The absence of lymphocytosis in the cerebro-spinal fluid.

3. *Tabes dorsalis* (p. 379). The only likelihood of difficulty in diagnosis arises in the sensory form or in the terminal stages, when motor symptoms are marked by flaccid paraplegia and the deep reflexes are absent. The points of difference are:
   (*a*) Absence of pupillary and cranial nerve changes.
   (*b*) Coincidental loss of all forms of sensation without special affection of cutaneous or muscle pain sensibilities.
   (*c*) Presence of an extensor plantar response.
   (*d*) The absence of lymphocytosis in the cerebro-spinal fluid.

4. *Peripheral neuritis* (p. 137) by:
   (*a*) Absence of any toxic cause.
   (*b*) Absence of tenderness on pressure along nerves.
   (*c*) Presence of extensor response.
   (*d*) Absence of trophic changes and drop foot or wrist.
   (*e*) Absence of sphincter trouble.

Prognosis. The prognosis is most unfavourable. In the senile cases the duration of life may extend for several years; but in all other cases, and as soon as the segmental anæsthesia develops, the duration seldom exceeds two or three months, but in each case the condition of the patient and the rapidity of the onset of symptoms must be considered in forming an opinion.

Treatment. Little, if anything, can be done to arrest the progress of the disease, but general tonic treatment, an open-air life, attention to the mouth and teeth, and iron, arsenic, and cod-liver oil, may delay the progress of the early stage. Those cases in which a remission of symptoms occurred were treated in this manner.

Chapter VI

ACUTE POLIOMYELITIS

(SYN.: INFANTILE PARALYSIS, ACUTE ATROPHIC PALSY OF ADULTS, ACUTE ANTERIOR POLIOMYELITIS)

This is an acute specific disorder, characterised by the onset of constitutional symptoms and the sudden development of paralysis, resulting either in recovery or in permanent atrophic paralysis.

Etiology. This disease is as common in males as in females, and affects young adults as well as children, although it is more frequent amongst the latter. It is by no means so rare in adults as was at one time supposed.

It has a definitely seasonal incidence, occurring more frequently in the late summer and early autumn months, statistics from several different countries showing that it is most common during the months of June, July, August, and September. That it is an infective disorder is shown by its tendency to attack several members of the same family about the same time, or several persons working in the same locality. It is, moreover, known to occur in epidemic form, several well-authenticated instances of which have been recorded.

The clinical association which has been sometimes found to exist between poliomyelitis and encephalitis, and the similarity of the pathological changes, would point to an identity between these conditions.

Pathology. The pathological changes found in the central nervous system in cases which have died within a brief period of the onset are characteristic. They are not confined to any particular segment or portion of the cord, but are more intense in the grey matter, and implicate more or less the whole of the bulbo-spinal centres from the oculo-motor nuclei to the conus medullaris.

The changes characteristic of the disease at an early period are a well-marked and widespread proliferation of small cells in and around the veins and capillaries. These cells, which seem to resemble lymphocytes in appearance and structure, infiltrate the walls and adventitial coat of the blood-

vessels. The grey matter of the spinal cord is mainly the seat of this perivascular infiltration, but not solely, for it is also observed around the vessels in the pia arachnoid and those radiating into the white matter, mainly upon the antero-lateral aspect of the cord.

In addition to these perivascular changes, a well-marked infiltration of cells occurs in the grey matter, more particularly of the anterior cornual region, but not exclusively limited to it. According to Farquhar Buzzard,[1] they are both neuroglial cells in various stages of proliferation and disintegration, and lymphocytes similar to those seen in the perivascular infiltrations. In the neighbourhood of the most pronounced infiltrations, the ganglion cells of the anterior horns, as well as those of Clarke's column and the posterior horns, are found in various stages of disintegration. All grades of cell change are present, from mere chromatolysis to conditions in which it is difficult to discern even the outline of the cell.

The cerebro-spinal fluid, withdrawn during life, frequently presents a large lymphocytosis; and although it is not uncommon to detect in it organisms resembling the pneumococcus, there is no clear evidence that such are the causal factors in the production of this malady.

In consequence of the destruction of the ganglion cells and the neuroglial tissues, the appearance of the spinal cord in old-standing cases of acute poliomyelitis is characteristic. A marked shrinkage of the anterior horns and of the antero-lateral region of the cord, and an almost complete disappearance of ganglion cells in certain limited localities, are seen. The fibres of the corresponding anterior nerve roots are atrophied as a result of the destruction of their ganglion cells.

Symptoms. The onset of the disease, whether in children or adults, is ushered in by pyrexia, malaise, and pains in the head, limbs, and body. Sometimes it is accompanied by vomiting, convulsions, and delirium. The temperature may rise to 102° F. or 103° F. at the onset, remain at 100° F. or 101° F. for a few days, and then gradually subside to normal in about a week. The pains are frequently severe, are generally a prominent symptom, and are occasionally accompanied by stiffness and rigidity of the muscles of the back, suggesting

[1] Farquhar Buzzard, *Brain*, 1907.

the onset of meningitis. The muscles are tender to pressure, and the patient complains of pain on passive movements and handling the limbs. This may persist for some weeks after all constitutional symptoms have disappeared.

Within a day or two of the onset of the constitutional symptoms, a flaccid paralysis is observed, either generally distributed or confined to one or other part of the body, such as an arm or leg, the back and abdomen, and in rare cases the eyes and eyelids. During this early period a temporary retention of urine is not uncommon.

FIG. 144.—A case of extensive acute poliomyelitis.

After the first week, by which time the pyrexia and constitutional phenomena have disappeared, the appearance of the patient is usually sufficiently characteristic of the malady. One limb may be completely paralysed, and the others escape or be partially affected, or there may be weakness of the spinal, abdominal, and intercostal muscles. In the rarer cases of superior poliomyelitis, ptosis, and ophthalmoplegia externa may be observed, with or without difficulty in swallowing, anarthria and facial palsy.

In all cases of acute poliomyelitis, whatever may be the part of the central nervous system permanently affected, the initial paralysis is always more extensive and more complete than the residual or permanent.

Motor system. On examination of the motor system, paralysis of wide or limited extent may be found. The incidence of greatest paralysis may be in the limbs, or the

trunk, more rarely in the facial and ocular muscles. One feature is characteristic of the disease, namely, that at the points of greatest paralysis the distribution of the paralysis is according to the nuclear grouping of the muscles in the spinal cord. Thus, it is common to find extensive paralysis with atrophy affecting one group of muscles, the neighbouring muscles remaining intact. For example, in the upper limb, paralysis of the shoulder muscles may be present without any affection of the forearm or hand muscles; in the lower

FIG. 145.– Photograph of a child with extensive acute poliomyelitis involving the abdominal muscles. The illustration shows the 'ballooning' of the belly on attempts to raise the head.

extremity paralysis of the anterior tibial group may exist without affection of the sural and posterior tibial muscles.

But whatever the distribution or extent of the paralysis, the maximum loss is seen immediately after the onset. Both the extent and severity of the palsy diminish in course of time, at all events up to about two years.

The palsy is of the flaccid atrophic type.

In fat children the atrophy may not be apparent, owing to the thickness of the subcutaneous tissue, but on feeling the limb the true condition is revealed.

The effects of the atrophic palsy are shown in the early stages by a loss of movement and a flail-like condition of the limbs with relaxation of the joints.

Electrical reactions. All degrees of the reaction of degeneration may be seen, from the fully developed reaction of degeneration to a mere diminution of faradic excitability. These changes begin in from four to ten days after the onset of paralysis.

As time goes on, *deformities* may develop. They arise in two ways: either by contraction of the non-paralysed muscles owing to the absence of the opposition of the paralysed muscles, or by subsequent contracture of the paralysed muscles. Spinal curvature may develop in consequence of weakness of the spinal muscles, or from a habitual postural attitude adopted by the patient to obviate a locomotory difficulty.

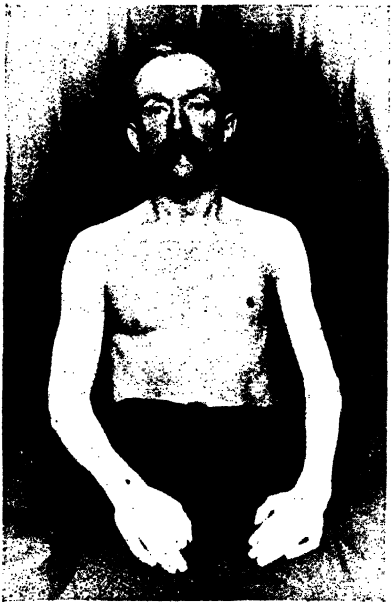


FIG. 146.—Shows wasting of the left arm in an old case of acute poliomyelitis.

Sensory system. Pain is induced on handling the muscles or moving the limb. This may be a prominent symptom, and may in children prevent the discovery of the paralysis. Objective loss is exceptional, but slight degrees of impairment of cutaneous sensibility have been detected in a few cases during the early stages and are always transient.

Reflexes. The tendon reflexes are abolished in the paralysed muscles. During the earlier stages an increase of the deep reflexes below the affected segments may be observed. Thus, in one case in which there was extensive paralysis of the upper extremity, the deep reflexes on the corresponding leg were definitely increased, in association with an extensor plantar response. These signs usually disappear, and are to be attributed to slight interference with the pyramidal tract during the initial widespread inflammatory condition of the cord.

The integrity of the superficial reflexes depends upon the state of the reflex arc and muscles.

Sphincters. Retention of urine is common in the early stages, and weakness of the abdominal muscles may cause a slight difficulty both of micturition and defæcation. Where

the lesion has affected the lumbo-sacral enlargement, dribbling of urine may persist for a time.

Trophic changes may be confined to the muscles. In severe cases the growth of the limb may be impaired.

Diagnosis. The points of differential diagnosis between acute poliomyelitis, Landry's paralysis, and toxic polyneuritis are given on p. 448.

Prognosis. The prognosis of this malady has to take into consideration two eventualities: first, the likelihood of recovery from the intensity of the initial symptoms; and, secondly, the degree and extent of the permanent paralysis and deformity.

In the first place, there is a severe and fulminating type of the disease, in which paralysis is extensive in distribution and of rapid onset, leading to death within a few days. This is a rare type, but one which requires recognition, as it has not uncommonly been mistaken for Landry's, or acute ascending paralysis.

FIG. 147.—Shows eversion of the foot, in an old standing case of acute poliomyelitis, from paralysis of the posterior tibial muscle (talipes valgus).

In the majority of the cases of acute poliomyelitis the constitutional symptoms subside satisfactorily, and the patient enters upon a long period of slow recovery, with more or less paralysis and disablement. On the other hand, he may almost completely recover.

What amount of paralysis is likely to remain may to some extent be based upon the electrical reactions. Those muscles which show the complete reaction of degeneration from three to six months after the onset remain paralysed. The degree of atrophy also is of value in gauging to what extent paralysis of individual muscles may recover.

Treatment. The treatment varies according to the stage

at which the patient comes under observation. In the acute or pyrexial period treatment should be upon general lines, rest in bed, light diet, and sedative draughts for the relief of the pains, restlessness, and malaise.

After the first week, when the paralytic symptoms have become apparent, hypodermic injections of strychnine, galvano-faradism to the paralysed muscles, and massage ought to be

FIG. 148. Talipes calcaneus in a case of acute poliomyelitis from paralysis of the calf muscles.

commenced and continued for several months. It is most important that the paralysed limb or limbs should be kept warm; hence the constant use of warm woollen stockings or bed slippers is desirable.

The method of applying the electrical treatment is important. In all cases an electrical examination of the muscles ought to be made, with a view to ascertain the degree and extent of the degenerative atrophy. To those muscles which fail to respond to the faradic current, galvanism ought to be applied, while the faradic current should be used to those in which the faradic excitability is still present. The combined current is best and most satisfactory for this purpose. As soon as voluntary power begins

to return, the patient should be encouraged in the use of exercises and of movements against slight and graduated resistance.

It not infrequently happens that during the early stages non-paralysed muscles are used in place of those that are paralysed, and in the later stages, when recovery has taken place, the child neglects to bring them back into use. This may be overcome by encouraging the patient in the physiological use of the previously paralysed muscles, or by means of an apparatus.

Deformities should be prevented by judicious manipulations or by the application of apparatus at those joints where the over-action of unopposed muscles is likely to cause distortion. If, in the later stages, deformities have already developed, special appliances may be used, or the question of tenotomy may require consideration. Special supports, such as a poroplastic jacket, are necessary in those cases in which the trunk muscles are permanently paralysed. (Fig 149.)

FIG. 149.—Photograph of a case of acute poliomyelitis, affecting the muscles of the back, and showing a resulting spinal curvature.

Two operative procedures have been recommended with a view to overcome or minimise the effects of paralysis when limited to a particular group of muscles—tendon transplantation and nerve anastomoses. The one method brings the paralysed muscle back into use by attaching to it a portion of the tendon of an adjoining healthy muscle, the other by anastomosing its nerve to an adjacent healthy nerve trunk.

Progress is often slow, little improvement being observed even from month to month. Treatment, more especially by means of massage, movements against resistance and exercises, should be persevered with for at least eighteen months.

Chapter VII

LANDRY'S PARALYSIS

(SYN.: ACUTE ASCENDING PARALYSIS)

This is an acute or subacute disease, characterised by rapid and progressive motor paralysis of the flaccid type, without muscular atrophy or electrical changes, often commencing in the lower limbs and extending upwards to involve the trunk, arms, diaphragm, and cranial nerves, sometimes resulting in recovery, but usually terminating in death from respiratory paralysis.

It is more common in males than in females, and occurs mainly between the ages of twenty and forty.

Pathology. The morbid changes are, according to Farquhar Buzzard, almost entirely confined to the ganglion cells of the spinal cord. On examination by Nissl's method, early chromatolysis with eccentration of the nucleus of the cells of the anterior horns and of Clarke's column is observed. By aid of Marchi's osmium bichromate method, small fat droplets are found in the fibres of the spinal cord and peripheral nerves. These changes are considered by Buzzard to be due to a toxic action, as they are common to other toxic conditions. Vascular and neuroglial changes are absent, and beyond general vascular engorgement no macroscopic changes are noted.

Various micro-organisms have been found, but it is not yet possible to ascribe the disease to any particular form.

Symptoms. The onset is insidious, and may be attended by general malaise, headache, a feeling of fatigue, and by cramps, pains, and tingling in the legs and back. Occasionally there is a slight rise of temperature, rarely exceeding 100° F., sometimes accompanied by anorexia. These premonitory symptoms may be slight, and the patient's attention is usually first attracted by a feeling of tiredness and heaviness in the legs, which rapidly become weakened and eventually paralysed. One leg may be affected before the other, but generally the weakness is present in both, although predominating in one. The paralysis does not invariably

commence in the legs, and we have seen cases in which it originated in the arms. Cases also are on record in which paralysis of the motor cranial nerves was the first symptom.

The motor weakness may affect the proximal portions of the limbs, such as the hip and shoulder muscles, before it affects the distal portions. But in whatever manner it commences, the condition rapidly spreads, and in the common or leg type the paralysis of the lower limbs is such, that within six to twenty-four hours the patient is unable to walk, and the legs are in a condition of extreme flaccid paralysis with loss of the deep reflexes. The trunk and thoracic muscles gradually become weak, so that the patient is unable to sit up and the respiration becomes diaphragmatic. In a few hours, or in three or four days, the palsy may be widespread. The patient now lies propped up in bed, with the arms and legs in a state of complete flaccid palsy. He is unable to raise his head from the pillow. Respiration is rapid and laboured, the intercostal muscles are paralysed, the diaphragm acts feebly, and the accessory muscles are brought into action. As he is unable to cough, mucus accumulates in the air passages. Phonation is weak, articulation and mastication feeble, but swallowing may still be possible. The face is cyanosed, sweating is profuse, and the pulse rapid (120–130 per minute).

The *mental condition* remains clear throughout; the patient is unable to realise the seriousness of his condition; he feels well and can give an accurate account of his symptoms.

Cranial nerves. The motor nerves alone suffer, chiefly the fifth, seventh, ninth, tenth, eleventh, and twelfth; less commonly the oculo-motor nerves are affected. Complete palsy of the cranial nerves is never observed, and the pupils respond to light.

Motor system. The motor weakness is more or less general, the distal parts of the limbs not suffering more than the proximal, as in toxic polyneuritis. When the paralysis commences in the lower limbs, these may be in a state of profound flaccidity before the higher parts of the body are completely involved. The muscles are not atrophic, and do not show any atrophy even after recovery. Myotatic irritability is diminished, but beyond a slight sluggishness to faradism, no changes in the electrical reactions are observed.

Sensory system. Numbness and tingling may be complained of in the early stages, but are rarely severe. Pain in the back and cramps in the legs, with slight muscular hyperæsthesia on pressure, are more common, but never approach the acuteness found in peripheral neuritis. Slight objective blunting of cutaneous sensibility has been described, but as far as personal observation goes, its presence is exceptional.

Reflexes. The deep reflexes are abolished, the superficial absent, and the plantars are either absent or flexor in type.

The *sphincters* are not affected, except in so far as the patient, partly from the position in bed, and partly from palsy of the abdominal muscles, has a difficulty in passing urine.

Course and prognosis. In acute cases death may occur in from two to six days, but the disease may still prove fatal even after three or four weeks. If the paralysis ceases to increase during a space of twenty-four hours, the outlook as regards life is materially improved, but the majority of cases end fatally. If death does not ensue, the prognosis as regards recovery of motor power is quite good, and there is no liability to relapse.

Treatment. The patient should be placed at rest upon a water-bed. Diaphoresis should be encouraged. The withdrawal of cerebro-spinal fluid by lumbar puncture is advantageous. Oxygen relieves the distress of the later stages, and strychnine and alcohol may be given.

DIFFERENTIAL DIAGNOSIS BETWEEN POLIOMYELITIS, LANDRY'S PARALYSIS, AND ACUTE POLYNEURITIS

	POLIOMYELITIS.	LANDRY'S PARALYSIS.	ACUTE POLYNEURITIS
Age at onset	Childhood	Adults	Adults
General symptoms	Well-marked	Slight	Often absent
Onset of palsy	Sudden	Gradual	Gradual
Distribution	General or according to nuclear grouping	Lower limbs, extending upwards	Peripheral
Effects of pressure	Muscles tender	No tenderness	Nerves tender
Sensory symptoms	Rare	None	Of peripheral type

PART X

VASO-MOTOR AND TROPHIC DISEASES

RAYNAUD'S DISEASE. SYMMETRICAL GANGRENE

This disorder is characterised by paroxysmal and recurrent attacks of transitory interference with the blood supply, resulting in local syncope, asphyxia, or gangrene, and affecting as a rule the extremities, usually bilateral and symmetrical in distribution, and always associated with spasm of the arterioles.

Etiology. It is more common in women than in men, and more frequent in young adults than in the old. It is more often seen in persons of nervous disposition, especially when run down or enfeebled. Exposure to cold, especially when produced by a sudden drop of temperature, is the most important of the exciting causes. The disease also shows a certain periodicity, and in women is worse during the menstrual period.

Pathology. This is not definitely known, but the symptoms appear to have, as an underlying factor, disturbance of the vaso-motor system, manifested by arterial spasm, which may be induced reflexly by cold or emotional shock. There is no constant or well-recognised morbid anatomy, but changes in the peripheral nerves, and in the later stages of some cases obliterative arteritis have been found.

Symptoms. Three degrees or phases of the disease have been described.

(*a*) A mild form, or *local syncope*. In this variety complaint is made of a feeling of deadness and numbness of the fingers, which rapidly become pale and blanched, with

lowering of the surface temperature. The condition is usually symmetrical, and affects the upper and lower extremities. It may be limited to the fingers and toes, or may involve the whole of the hand or foot.

Stiffness of the fingers and toes, with some disturbance of sensibility, are observed. The objective sensory loss consists of impairment of tactile sensibility and a more marked degree of analgesia. There is never, however, any complete dissociation of sensation. Deep sensibility is not impaired. Owing to the stiffness and sensory defects, the patient is unable to execute fine movements of the fingers, but the grasp may be relatively unimpaired.

During an attack, which may last from a few minutes to one or two hours, the pulse is small, but is never obliterated. In the mildest forms a rapid return to the normal takes place without much reaction; but in the graver forms the return is marked by burning pain, tenderness on pressure, flushing, and sometimes local perspiration.

(*b*) A moderate form, or *local asphyxia*. The initial stage of this variety consists of a slight blanching of the extremities, which rapidly gives place to local asphyxia, commencing in the tips of the fingers or toes, and spreading upwards to involve the hands and feet, but rarely extending above the elbows or knees. This condition may also affect the point of the nose and the lobes of the ears. The colour of the parts varies from a purple-red to a slate or blue-green. The nails become dark, the veins swollen, and the surface temperature is much reduced. A slight œdema is usually present, and on pressure a white spot is left, which persists for a considerable time. The stiffness and the sensory changes are more pronounced than in the syncopal form.

The duration of these attacks varies from a few hours to two or three days, and the reaction is more pronounced, being attended by pain, local perspiration, tenderness, and occasionally by puffiness or powdery desquamation.

(*c*) A severe form, or *local gangrene*. In this form trophic changes of a permanent character take place, resulting from an increase either of the local syncope or of the asphyxia. The pain is severe, and small vesicles, which burst and discharge blood-stained fluid, form over a limited area. The underlying skin dies, and a small sequestrum of necrosed

tissue is formed, bounded by a zone of ulceration. The bone is rarely affected.

Complications. Cerebral complications of the nature of epileptiform fits are not uncommon, and slight degrees of the disease are often associated with epilepsy. Mental changes, especially acute mania, have been recorded in cases of Raynaud's disease. Temporary amblyopia, coexistent with vascular changes in the fundus oculi, have also been noted in rare cases.

Hæmoglobinuria or hæmaturia may be observed either during an attack, or alternating with the attacks in which the extremities are affected. This complication is probably more frequent than is generally realised.

Diagnosis. The paroxysmal character of the attacks, and the absence of permanent sensory changes, suffice to distinguish this disease from congenital cardiac disease, syringomyelia, and senile gangrene.

It may be distinguished from the gangrene associated with endarteritis obliterans, by its symmetrical distribution, its superficial extent, the absence of arterial degeneration, and the preservation of the pulse in the affected limbs.

Somewhat similar conditions are seen in poisoning by ergot of rye, quinine, and the coal-tar products.

Prognosis. In the milder forms appropriate treatment will cut short an attack, and care and attention to diet, with avoidance of exposure to cold, are markedly beneficial.

In the gangrenous form the prognosis is favourable, so far as the possible spread is concerned, and even if amputation is necessary the result is not unsatisfactory.

Treatment. Attention should be paid to the avoidance of cold and fatigue. Cod-liver oil and general tonics, regulation of the bowels, attention to the state of the mouth and digestion are important in lessening the tendency to the attacks. Physical exercise should be encouraged.

Galvanism and galvano-faradism to the affected limbs, applied by means of a foot or hand bath, for periods of fifteen to forty minutes, may check or cut short an attack. Massage of the limbs is also important and useful for this purpose. Submerging the hands in warm or hot water during an attack will relieve the pain and discomfort.

The constant use of warm woollen gloves and stockings by

those who are subject to this malady is recommended, and the avoidance of sudden exposure to a lowered temperature.

INTERMITTENT CLAUDICATION. INTERMITTENT LIMPING

This is a condition characterised by the occurrence of weakness, paræsthesia, and cramps, mainly in the legs, which come on in certain persons during muscular exercise, and is associated with angio-sclerosis and vaso-motor disturbance.

Etiology. It is more common in men than in women, and in those of a nervous disposition. Alcohol, syphilis, gout, and the excessive use of tobacco and tea have a predisposing influence, in so far as they act as causes of vascular degeneration. Aneurism of the femoral artery, pressure upon the arteries from a truss, or during pregnancy, have been found as coexistent features in several cases. The association of flat foot has been noted in a number of instances.

Pathology. There are two distinct factors in the production of the disease: (1) an angio-sclerosis affecting both the arteries and the veins of the limb, and (2) disturbance of the vaso-motor mechanism. The latter factor alone distinguishes the cases from ordinary senile obliterative endarteritis with gangrene.

The vascular changes consist in thickening of the intima of the arteries and the veins; these changes are constant in the small vessels, and are sometimes associated with a like process in those of larger calibre.

Slight general wasting of the muscles has been observed, and degeneration of the peripheral nerves, apparently as a secondary result of the sclerotic changes of the vasa nervorum, has been described.

Symptoms. These depend upon the cutting off of the blood supply to the muscles and nerves. During rest no discomfort is present, but after walking for a short time the leg begins to feel heavy, fatigued, and painful. If a pause is made, the symptoms pass away. If, on the other hand, the patient persists in his efforts, the leg begins to feel cold and a pricking sensation comes on, which increases to a feeling of numbness and weakness, with sometimes a sensation as if the leg is

going to burst. The muscles become stiff, and may be the seat of painful cramp, which eventually prevents walking. The foot and leg may appear congested and swollen, are usually cold, and have a mottled, cyanotic look.

On examination of the limb, the most important feature of the disease is detected, namely, obliteration of the pulse in the dorsalis pedis and posterior tibial arteries. This has been observed in all genuine cases. Although the condition is usually unilateral, symptoms may affect the other leg to a less extent.

Some cases progress to gangrene of the foot. In this way it comes about that clinically this affection links simple obliterative gangrene with symmetrical gangrene or Raynaud's disease.

The **prognosis** is bad, but by prolonged rest, and care in avoiding that amount of exercise which will induce the pain, the symptoms may be alleviated and the progress of the malady delayed.

The **treatment** consists in avoiding all preventable causes of vascular degeneration. The administration of iodides and of nitro-glycerine are of little value. Locally, warmth obtained by the use of woollen stockings and warm baths may relieve the symptoms when present; but the essential feature is the curtailing of exercise well within the point at which the symptoms are brought on, and occasional spells of rest in bed. We have found considerable relief obtained by the use of the high frequency currents, and gentle massage may be applied with advantage.

In the event of gangrene supervening, amputation of the limb is necessary.

ERYTHROMELALGIA

This is a chronic affection characterised by the occurrence of severe pain usually in one extremity, followed by local redness and elevation of temperature over a limited area of skin, and aggravated by a dependent position of the limb and muscular exertion.

Etiology. It occurs in persons of middle life, and in men more commonly than in women. It has been observed most frequently amongst those of Jewish descent. A

predisposing cause is excessive work, with long periods of standing; shock, injury, and acute diseases have been cited. It has also been observed in association with some forms of organic nervous disease.

Pathology. It depends upon disordered vaso-motor functions, combined with a morbid condition of the blood-vessels, consisting of thickening of the middle and inner coats of the arteries, and in some cases with an associated degeneration of the peripheral nerves.

Symptoms. It is most common in the lower extremities, but may occur elsewhere; as a rule, one foot only is affected. The first indication is pain of a burning character coming on towards the end of the day in the sole of the foot.

Rest and elevation of the limb may bring temporary relief. In course of time the pain becomes more frequent, and is more easily induced. Sooner or later a rosy redness appears in patches, especially after the limb has been dependent for a time. The affected area is hot, the veins engorged, and the arterial pulse may be exaggerated. The patch becomes extremely hypersensitive to deep and superficial pressure, and slight swelling may be seen. The pain and redness are increased by heat and pressure, and relief is brought about by rest, cold, and elevation of the limb. Slight general muscular wasting may develop later.

The **prognosis** is bad, the affection tending to become worse, although in some cases it has disappeared spontaneously.

Treatment. Gouty or syphilitic taints should be treated by appropriate remedies. Rest, elevation of the limb, and the application of cold, afford temporary relief; if the pain is severe, the bedclothes should be kept off the feet by means of a cradle. Section of a nerve, or amputation of a limb, has not proved successful.

FACIAL HEMIATROPHY

This is a rare condition, characterised by atrophic changes in the skin, subcutaneous tissues, muscles, and bones of the face upon one side in the distribution of the trigeminal nerve.

Etiology. The disease usually arises before puberty, but only rarely, if ever, after thirty years of age. It is more common in women than in men. In a few cases a direct

heredity has been traced. It may in some instances be ascribed to acute infective diseases, in others to trauma, but in the majority no obvious cause is found.

The **morbid anatomy** consists in a proliferating interstitial neuritis of the trigeminal nerve (Mendel), but it is debatable how far the cutaneous and osseous changes are directly and solely the result of the nerve lesion. If this were

FIG. 150 is a photograph of a case of left-sided facial hemiatrophy.

so, it is difficult to understand why there are no symptoms of lesion of the sensory fibres, which form the bulk of the fibres of the fifth nerve. It is more probable that the nervous and cutaneous lesions arise from one and the same cause, and are not cause and effect. The disease would appear to be due to an arrest of development during the growing period, arising from a morphœa of the fifth nerve (Hutchinson).

Symptoms. The skin, usually over the cheek, chin, or forehead, becomes white or whitish yellow. The patch thus formed spreads, sometimes by fusion with adjacent patches. When the disease is well marked, there is a characteristic

depression under the malar bone, arising from atrophy of the subcutaneous tissue in this locality. If the change involves the hairy parts of the face, alterations in colour may be detected, or the hair thins and falls out. The facial muscles in old-standing cases are thinned and wasted, but do not show the degenerative changes characteristic of nerve lesion. The facial bones—frontal, malar, and maxillary—also become shrunken; the nasal cartilages share in the atrophic change, but the ear is less often involved. The tongue also sometimes shows atrophic changes. Sensation is unaffected.

The distribution of the atrophic change on the face varies, so that certain varieties have been described: (*a*) complete unilateral facial atrophy, (*b*) incomplete unilateral atrophy, (*c*) bilateral atrophy, and (*d*) cases with similar changes in other parts of the body.

Prognosis. The malady is progressive and has no tendency to shorten life.

The **treatment** consists in the administration of general and nerve tonics, such as quinine, iron, arsenic, and strychnine. Massage of the facial muscles and faradic stimulation may lessen or hinder the effects of the atrophic process.

ANGIO-NEUROTIC ŒDEMA

This name has been given to a number of conditions characterised by localised, or general, œdema which is unassociated with inflammatory or other gross lesion, but generally coexisting with some impairment or alteration of the nervous functions.

Pathology. It belongs to the same class of angio-neuroses as Raynaud's disease. It is probably dependent upon a local disturbance of innervation of the blood-vessels of the affected area.

Symptoms. The symptoms consist of a circumscribed swelling and œdema of the skin and subcutaneous tissues in various parts of the body, chiefly of the face, arm, and hand. The swelling appears suddenly without obvious cause, and disappears in the course of a few hours or days. It is unattended by pain or itching, but may give rise to a tingling or burning sensation. The local cutaneous attacks may be accompanied by signs of similar affection of the mucous

membranes, such as vomiting, diarrhœa, dyspnœa, and even asphyxia from œdema of the larynx and epiglottis. Hæmoglobinuria has been found in rare instances. It has a special tendency to appear in certain families.

A condition known as *hereditary œdema*, which probably stands in close relation to the angio-neurotic form, has also been described.

Treatment consists mainly in the administration of general and nervine tonics. A calomel purge at the onset of an attack is of value, and nitro-glycerine has been recommended should the seizures be frequent or persistent.

SCLERODERMA

This is a localised or diffuse hardening of the skin, affecting females more frequently than males, and occurring during the middle period of life.

Symptoms. The *circumscribed form* is characterised by the appearance of hard, brawny patches of a waxy colour, developing with great rapidity and disappearing after a few weeks, or persisting for months or years. It is most common about the neck and chest. In the *diffuse form*, a brawny hardness appears in the skin of the face or limbs, and gradually affects the whole of the limb, face, or trunk. Occasionally it is universal and gives rise to a characteristic appearance. The face is expressionless, eating is carried out with difficulty, and the limbs are fixed and immobile.

The secretion of sweat is abolished, the skin is dry, glossy, and smooth, and sometimes the extremities of the limbs present a cyanosed appearance.

Prognosis. The disease is chronic and may persist for many years. Death occurs from pulmonary or renal complications.

Treatment consists in maintaining the body temperature by warm clothing. Warm baths and massage are of much use. Rubbing the skin with oil is strongly recommended.

Part XI

FAMILIAL DISEASES

THE MUSCULAR DYSTROPHIES

Grouped under the title of Muscular Dystrophies are several varieties of muscular atrophy in which the voluntary musculature is primarily involved, and the nervous system is only secondarily, if at all, affected.

The close relationship which exists between the nervous and the muscular apparatus renders it imperative to consider the muscular dystrophies along with the diseases of the nervous system, as they give rise to weakness and impairment of movement, and have to be distinguished from the myelopathic muscular affections, or those secondary to disease of the nerve elements.

Although muscular dystrophy is a congenital condition, its manifestation may be delayed until the patient has advanced to puberty, adolescence, or later life. The changes in the muscles are both quantitative and qualitative. The former consist of defective development or absence of certain muscles, or portions of muscles; the latter of a potential weakness of the muscle, whereby it is unable to develop properly, and sooner or later becomes the seat of pathological changes. These alterations may give rise to an increase in the bulk of the muscle from an over-growth of the interstitial tissues—pseudo-hypertrophy, or to a diminution and atrophy of the muscle. In all cases the tendency is eventually towards muscular atrophy.

The muscular dystrophies have been divided by some writers into three clinical subgroups, according to the character and situation of the muscular changes; but, although many

cases fall naturally into one or other group, there are some which conform to none, or have features common to all.

1. Pseudo-hypertrophic muscular paralysis

Etiology. This type occurs more frequently in males than in females, in the proportion of five to one. When present in females it is often of later development and slighter degree. Isolated cases are rare, the disease having a familial distribution. It is always transmitted through the mother, who is herself not affected. It manifests itself during the period of development, either in late infancy or in mid or later childhood. The commonest time of onset is between the ages of four and six, and three-fourths of the cases occur before the age of ten.

Pathology. In the pseudo-hypertrophic forms *the muscles* are pale and fatty-looking, and on microscopic examination an increase in the interstitial substance is noted, either of fibrous tissue, of fat, or of both. The muscle fibres are thinned and irregular in shape, large in one portion, small in others. Striation is often indistinct, and the fibres may be seen undergoing granular degeneration, hyaline change, and vacuolation. The nuclei may be increased in number. In some cases of pseudo-hypertrophy the muscle fibres are enlarged and show large central nuclei. As a rule, the fibres are best preserved where there is an increase in the fatty rather than in the fibrous interstitial substance. Bundles of apparently normal muscle fibres may be scattered throughout. The muscle spindles are intact.

Nervous system. Atrophic changes in the anterior nerve roots, consisting in a loss of fibres and increase of fibrous tissue, have been described.

In the spinal cord itself a reduction in the size and number of the anterior cornual cells has been observed, in some associated with an increase of the neuroglial matrix. In no case has any degenerative alteration, either of nerve cells or fibres, been found in other portions of the central nervous system.

Symptoms. The first feature which calls attention to the condition is usually instability in walking, the child being easily knocked over, having difficulty in going up stairs, and showing inability to play games like other children. Later on, the gait may be noticed to be peculiar.

This disability comes on gradually, and it is a not uncommon cause of wonderment to the parents why a child with such well-developed legs is unable to walk properly. In some cases the child has never walked well, but as a rule it has been able to get about in a normal way for some months or a year or two before the weakness is noticed.

The child is usually fat and full-faced, though anæmic looking. In some cases the muscular hypertrophy may not be at first apparent, but even then the affected muscles can be felt to have a hard, 'putty' feeling. In the majority of cases, on the other hand, an increase of size in certain muscles is at once obvious. The following are the muscles most commonly increased in size :—

Lower limbs : Calf muscles ; extensors of knee—rectus femoris and vastus externus ; glutei ; anterior tibial muscles (rarely).

Trunk : Lumbar muscles.

Upper limbs : Supra- and infra-spinati ; deltoid ; triceps and biceps.

Face : Masseters (rarely).

The following muscles are frequently diminished in size, or absent : Lower portion of the pectoralis major; latissimus dorsi ; teres major ; clavicular part of the sterno-mastoid.

The flexors of the hip are also usually affected, but cannot be objectively examined.

The muscles of the face and tongue, forearms and hands, and the flexors of the knee are not affected. As the disease progresses the implicated muscles gradually atrophy, so that those originally enlarged tend after a time to become small.

Owing to the bilateral weakness or absence of the lower part of the pectoralis major, latissimus dorsi, and teres major, the axillary folds are practically absent. The child is unable to depress the arms against resistance, and it is impossible to lift the child up by the arms. Owing to the weakness of the latissimus dorsi, coughing is very feebly performed.

The attitude and gait are characteristic. The child stands on a wide base with the shoulders and upper part of the body held well back, and showing a marked anterior convex curve in the lumbar region. This lordosis is only present when the patient is standing erect, and is the device made use of to compensate for the weakness of the extensors of the hips,

as by its aid the centre of gravity is thrown further back and the strain upon the extensors relieved. In walking, the feet are lifted off the ground with some difficulty, and the pelvis is tilted downwards to the side opposite to the leg which is about to be advanced. As the advanced leg reaches the ground the centre of gravity is transferred to it by tilting the pelvis to the same side. This is the basis of the waddling

FIG. 151.

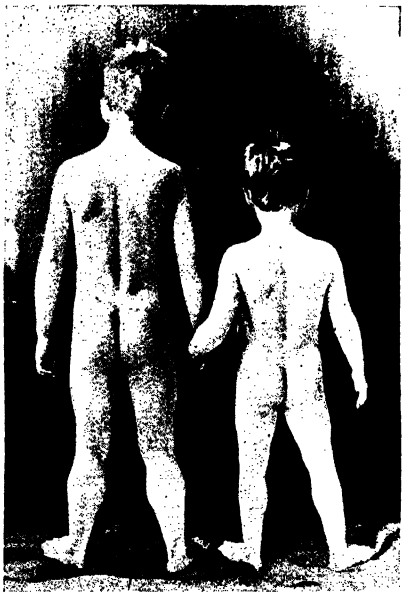


FIG. 152.

FIGS. 151 AND 152.—Illustrate the general appearances of pseudo-hypertrophic muscular paralysis.

or oscillating gait (alderman's gait) characteristic of pseudo-hypertrophic paralysis.

The full demonstration of the defect is best seen when the patient is asked to rise from the floor. The patient, resting upon his hands and knees, raises the lower portion of the back so that he rests upon his hands and toes, placing them as far apart as possible. He next supports some of the weight of his body with one hand on the floor, while the other hand is placed upon the front of the corresponding thigh, by this means aiding the weakened extensors of the knee. He now

depresses the sole of the foot on to the ground, and by gradually working the one hand up the thigh, at the same time pressing off the ground with the other, he lessens the strain on the weakened extensors of the hip. The hand which still remains upon the floor is now placed upon the thigh, and by gradually working both hands up the thighs the body is brought more and more to an upright position, until by a final effort the shoulders are thrown back, the spine curved forwards, and the erect attitude attained. In more advanced cases the patient is usually unable to rise at all from the floor.

In the early stages the lordosis, present when standing, is absent in the sitting posture; but in advanced cases weakness of the erectores spinæ results in curvature when sitting.

Talipes equinus, due to the fibrous shortening of the calf muscles, is one of the earlier deformities, and the flexor contractures at the knees and elbows, resulting from the unopposed action of the flexors of the knee and elbow respectively, are among the later.

In rare cases the tongue has been affected, but the cranial nerves and the sensory system remain intact.

The electrical examination shows a quantitative decrease to faradic and galvanic stimulation corresponding to the degree of muscular change.

The deep reflexes are never exaggerated, but become impaired as the various muscles atrophy, and are finally lost. Sphincter trouble is rare.

These patients are as a rule placid, intelligent, and good tempered. Mental defect is not common, and if present ought to be regarded as a complication.

2. Cases in which the original situation of the muscular affection is similar to that seen in pseudo-hypertrophy, but in which no hypertrophy of the muscles is detected.

3. Cases in which a general affection of the muscular system is present.

4. Juvenile muscular atrophy

This is an affection of the muscles of the shoulder girdle and upper arm, usually atrophic, but frequently with a combination of atrophy and pseudo-hypertrophy (Erb's type of juvenile muscular atrophy). The typical features are

the onset of the malady some time during puberty and early adolescent life, usually before the age of twenty years. The muscles of the shoulder girdle primarily implicated are the lower part of the pectoralis major and the latissimus dorsi, less commonly the upper part of the pectoralis major, trapezius, serratus magnus, and rhomboids. In the upper arm, the biceps, triceps, and supinator longus are usually involved, while the deltoid, supra- and infra-spinati as a rule

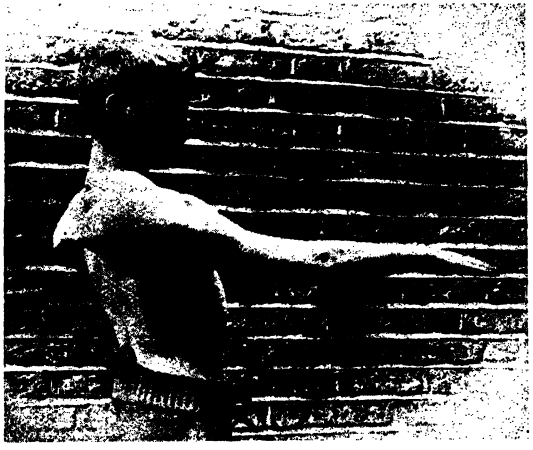


FIG. 153.—A case of Erb's juvenile muscular atrophy showing weakness of the serratus magnus muscles and winging of the scapulæ.

escape, but in some instances are found hypertrophied. In the pelvic girdle the muscles usually affected are the flexors of the hip (ilio-psoas), glutei, and the extensors of the knee. In the later stages the trunk muscles, erectores spinæ, intercostals, diaphragm, and abdominals become involved.

5. Cases in which the face is primarily affected. The face may be affected alone in infancy, but most cases develop later into the facio-scapulo-humeral type.

6. The facio-scapulo-humeral type

This is a type in which the face is primarily involved, and later the shoulder and upper-arm muscles—the facio-scapulo-humeral or Landouzy-Déjèrine type. In the facio-scapulo-humeral type (Landouzy-Déjèrine) males and females are

affected more equally than in the pseudo-hypertrophic form. It is more constantly present in successive generations. Usually the onset is between the ages of fifteen and twenty-five. The muscular changes may be limited to the original situation for a number of years, and life may be prolonged up to fifty years from the onset.

It is characterised by an affection commencing in the facial muscles—zygomatics, orbicularis oris, risorii, and

FIG. 154.—Illustrates the appearance of the face and mouth in the facio-scapulo-humeral form or Landouzy-Déjèrine type of muscular dystrophy.

levatores menti. Occasionally the orbicularis palpebrarum is also affected. The expression of the face is more or less characteristic, the lips are everted, and may remain apart, the lower lip protruding and giving the so-called 'tapir mouth.' The lips cannot be pursed, or applied to a glass in drinking, whistling and blowing out the cheeks are impossible. Articulation is affected owing to an inability to pronounce labials. The naso-labial fold is lost, and in smiling, instead of the angles of the mouth being drawn upwards or outwards, the upper lip is elevated and the mouth forms a straight line.

The shoulders and arms are affected similarly to that already described under the Erb or juvenile type.

7. The pelvic type

In the pelvic type (Buzzard) the paralysis commences with weakness of the ilio-psoas group, so that a difficulty is experienced in going up stairs. Owing, however, to the situation of the muscles, no objective examination can be made. In some of these cases, as the malady progresses, other muscles of the pelvic girdle become affected; in other cases, pseudo-hypertrophy may be noted in some of the muscles, such as the infra-spinatus. The affection as a rule originates in early adult or later life, affects females more commonly

FIG. 155. Illustrates the 'myopathic facies'; observe the straight line of the mouth and the obliteration of the nasolabial folds.

than males, and is slowly progressive, so that the fully developed cases present the features already described under the other varieties.

8. The distal type

In the distal type the small muscles of the extremities are primarily affected. In this type, in addition to the affection of the small muscles, the face is frequently implicated. Although this presents some resemblance to the peroneal type of muscular atrophy, the two conditions are quite distinct, the affection of the face and the absence of sensory changes serving to distinguish the two diseases.

In whatever position the muscular dystrophic condition commences, there is a tendency, as the malady progresses, for it to involve other muscular groups, so that eventually the characteristic attitude, gait, and postures already described under pseudo-hypertrophic muscular paralysis are assumed. On the other hand, cases persist for a number of years without such extension; in these instances the type observed is that shown in the early stages only.

Diagnosis. The chief points of distinction between muscular dystrophies and the atrophic muscular palsies of nervous origin are seen in the following table :—

	Muscular Dystrophy.	Nervous Amyotrophy.
Age at onset	Childhood, puberty, adolescence	Adult or later life
Heredity	Well marked	Usually none
Muscles affected	Facio-scapulo-humeral, pelvic girdle, &c., i.e. not according to nuclear grouping in spinal cord	According to nuclear grouping and arrangement in spinal segments
Bulb	No bulbar symptoms	Frequently bulbar symptoms
State of muscles	Pseudo-hypertrophy of some, atrophy of others Congenital atrophy or absence of some muscles No fibrillary twitchings	Degenerative atrophy only occurs; no hypertrophy Fibrillary twitchings
Reaction	No typical R.D., but reactions diminished in proportion to atrophy	In acute cases the R.D. is well marked
Reflexes	Tendon reflexes never increased. Disappear in correspondence with the muscular affection Superficial not affected	Tendon jerks exaggerated; ankle clonus may be present Superficial usually lost Extensor plantar response
Sensation	Never affected	Rarely affected
Sphincters	Rarely affected	Rarely affected

From the atrophies dependent upon lesions of the peripheral nerves, the dystrophies may be distinguished by the absence of sensory symptoms (objective and subjective) and the other points mentioned in the above table.

Prognosis. In all the types the prognosis as regards recovery is bad. The pseudo-hypertrophic variety tends to progress, and in from eight to ten years the patient is unable to stand or walk. In cases which arise in later years (the juvenile forms) the progress is usually slower; and in some cases the affection remains limited to the parts originally involved. The cause of death is an intercurrent affection, especially of the respiratory system, such as acute pneumonia, bronchitis, broncho-pneumonia, or pulmonary phthisis. Any illness, which obliges the patient to take to bed, is followed by a rapid increase of paralysis, and patients able to walk before may be unable to do so afterwards. Once a patient is unable to get about, deformities rapidly develop.

FIG. 156 is a photograph of a case of Erb's juvenile muscular atrophy.

Treatment. These patients should be encouraged to move about and use the muscles as far as possible short of fatigue. They should never be kept in bed if this can be avoided.

Massage, passive movements, and faradism help to maintain and improve the muscular condition.

General tonic treatment should be prescribed, and care taken to prevent respiratory complications.

AMYOTONIA CONGENITA

This is a rare condition characterised by a congenital affection of the muscles. In typical cases, it is noticed that shortly after birth the child's limbs are unnaturally flaccid and hypotonic. It is also observed that sucking is impossible,

and as the child grows, that it cannot sit, and that the limbs may assume any position in which they are placed. Later on the child is unable to walk, the movements are feeble, the limbs are flail-like at all joints, and a tendency to shorten-

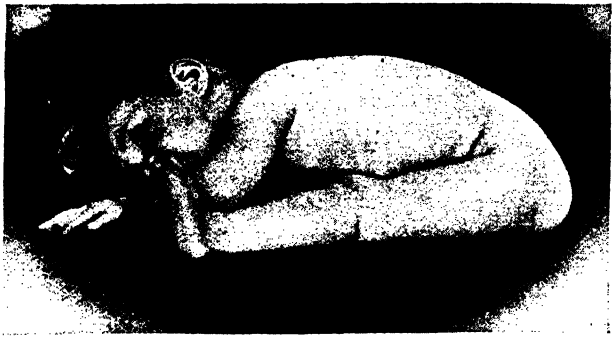


FIG. 157.

FIG. 158.

FIGS. 157 AND 158.—Illustrate the condition described as amyotonia. Owing to the muscular atonia the limbs may be placed in various abnormal positions.

ing of muscles ensues, which may limit the amount of passive movement. The electrical reactions are quantitatively diminished and the deep reflexes are abolished. There are no sensory changes. The mental condition is unaffected.

The tendency of the malady is towards improvement, but as none of the cases so far recorded have been observed over a sufficient period of time, it is impossible to say how much

improvement may take place, or what may be the duration of life.

In two cases examined pathologically, changes in the muscles and in the anterior roots and cornua of the spinal cord, similar to those found in some cases of muscular dystrophy, have been described.

It is impossible to exactly classify these cases, but from the point of view of morbid histology they are myopathic, although clinically distinct from the muscular dystrophies just described.

MYOTONIA CONGENITA. THOMSEN'S DISEASE

This rare condition occurs in families and appears to be more or less hereditary. Its onset is usually in early childhood. It is characterised by delayed relaxation of the voluntary muscles after contraction, owing to the presence of tonic spasm. The difficulty may exist on the initial movement, or on attempts at movement after a period of rest; but after repeated efforts the spasm becomes progressively less, an improvement which is maintained during continuance of the effort.

In its slighter forms the disease is manifested by stiffness and awkwardness of movement, but in the more severe forms it may completely disable. For example, the patient may be unable to rise from bed or from a chair, but after repeated efforts the spasm becomes less and finally disappears, so that he can walk for miles without fatigue. In the most severe instances the spasm may affect not only the limbs, but may extend over the whole body, rendering it rigid and immobile.

The limbs, especially the legs, are mainly affected; respiration, micturition, and defæcation are unaffected. In rare cases mastication and swallowing have been embarrassed.

The muscles are large and firm, but their power and strength are not in proportion to their size. They possess an increased excitability both to mechanical and electrical stimulation; and the contraction, thus excited, persists for a considerable period. The anodal closing contraction is as easily obtained as the cathodal. A continuous galvanic

current causes a series of wave-like contractions from the cathode to the anode (Erb).

The reflexes are unaltered.

The disease persists throughout life with little change. A life of activity tends, on the whole, towards amelioration of the symptoms.

The condition is characterised pathologically by a hypertrophy of the muscular fibres and a proliferation of their nuclei.

MYOTONIA ATROPHICA

This is a very rare condition, which never arises before puberty. It is characterised by a myotonic state of the muscles of the extremities, especially of the hands. Owing to this the patient is unable to relax his grasp suddenly. In addition to the muscular tonicity, atrophy may be present. The muscles usually affected are: the facial muscles, the sterno-mastoids, vasti femoris, and dorsiflexors of the feet.

FAMILY PERIODIC PARALYSIS

This is a rare disease, characterised by recurring attacks of paralysis of the voluntary muscles, with temporary loss of electrical excitability.

It is essentially a family disorder, and has in one instance been traced through five generations. It is commonest in children and young adults. Fatigue would appear to be its chief exciting cause.

Pathology. The disease appears to be of a toxic nature. Slight degrees of lymphocytosis have been observed on examination of the blood, and portions of muscles excised during life have shown marked vacuolation and fissuring. A hypertoxicity of the urine has been observed after an attack of paralysis. It has been suggested by Buzzard that the action of the toxic substance is upon the muscular rather than upon the nervous tissues.

There is so far no record of any post-mortem examination.

Symptoms. The attacks of paralysis follow upon physical exertion, some hours intervening between exercise and the onset of the paralytic symptoms.

The attacks consist of motor paralysis of a flaccid type,

commencing in the proximal muscles of the limbs and then invading the whole limb, the muscles of the trunk, and the respiratory muscles. The diaphragm is rarely affected, but the area of cardiac dullness is increased and a systolic bruit may develop at the mitral orifice. The cranial nerves are rarely paralysed, although ptosis and bulbar symptoms have been occasionally observed.

The palsy is of the flaccid type, the patient lying helpless in bed, breathing only with difficulty. The deep reflexes are in abeyance, and no response is obtained to either galvanic or faradic stimulation. This is a characteristic feature.

The duration of the attacks varies from six or eight to sixty or more hours. Their disappearance, like their onset, is gradual. The intervals between the attacks also vary from a few hours to several months. There would seem to be a tendency in the course of time for the intervals to lengthen and the attacks to shorten.

Prognosis. The outlook as regards life is favourable. No patient so far has died of the disease.

Treatment. The most successful procedure has been the administration of diuretics and copious libations of soda water. The elimination of toxic substances is in this way, encouraged. Tonic treatment, moderate exercise, and simple easily digested food may also be prescribed with advantage.

THE FAMILY FORM OF MUSCULAR ATROPHY OF SPINAL TYPE IN CHILDREN (WERDNIG-HOFFMANN)

This disease commences during the latter half of the first year of life, and is characterised by the development of weakness and atrophy of the muscles. It occurs in more than one member of the family, and runs a similar course in each case The weakness is first seen in the muscles of the back and hips, and gradually spreads to involve the limbs, including the small muscles of the hands. Later, bulbar weakness is noticed. The onset of the paralysis is followed by atrophy, more or less rapid and extensive, with occasionally fibrillary tremor. The reaction of degeneration is present. Sensation is unimpaired, the sphincters are normal, and the deep reflexes are lost.

As the malady progresses, the child becomes helpless and

paralysed. Death frequently results from respiratory complications, but may not take place for three or four years.

Pathology. Atrophy of the muscular fibres is associated with atrophy of the nerves, anterior roots, and anterior horn cells of the cord.

PERONEAL MUSCULAR ATROPHY

This disease commences between the ages of fifteen and forty years, but usually before twenty-five. One or more members of a family are affected, the males more commonly than the females. It may be directly hereditary, or may miss a generation.

FIG. 159.—Illustrates the wasting of the outer aspect of the right leg in a case of right-sided peroneal muscular atrophy.

Pathology. The changes found in the nervous system consist of muscular atrophy of neuritic type, degeneration of the mixed nerves, of the anterior nerve roots and anterior horn cells, and also in lesser degree of the posterior roots and posterior columns. It is therefore quite distinct from the myopathies, and must be placed amongst the muscular atrophies of nervous origin.

Symptoms. It runs a slow course, extending over many years. It is characterised by the development of atrophy of the small muscles of the feet, the peronei muscles, the extensor hallucis, extensor communis digitorum, and later the gastrocnemii.

A similar condition affecting the small hand muscles and those of the thenar and hypothenar groups, with consequent deformity, develops later in the upper limbs. The appearance of the leg is typical. In the majority of cases the foot is lengthened and inverted, the toes pointed, the leg below the knee wasted, especially on the peroneal aspect. In some cases the ankle joint is loose and flaccid, in others it is ankylosed. Deformities in the shape of talipes varus and equino varus are sometimes present. Fibrillary twitchings and an incomplete reaction of degeneration are seen in the muscles.

FIG. 160.—Appearance of the legs and feet in a case of peroneal muscular atrophy.

Impaired electrical irritability to faradism alone, or in association with muscular atrophy, may be observed over a wider distribution.

The ability displayed in walking is surprising, considering the atrophic state of the limbs and weakness of the muscles.

Sensory symptoms such as pain are occasionally present, and slight but definite disturbance of sensibility may be found on the outer side of the leg. There is no tenderness on pressure along the nerves.

The deep reflexes diminish and disappear according to the muscles affected. The superficial remain normal. The sphincters are unaffected.

Course and prognosis. Life is not shortened by the disease, which may run a course of many years. A certain number of cases appear to become arrested, though the greater number develop atrophy in the distal segments of the lower and upper limbs.

The **treatment** is conducted along the lines for atrophic palsies in general. Orthopædic appliances, or surgical measures, may be adopted for individual cases with great benefit.

FRIEDREICH'S DISEASE

This is a progressive malady, often occurring in families, characterised clinically by the gradual onset of inco-ordination of movement, impairment of articulation, and the development of deformities of the back, feet, and hands. The pathological changes consist of a system-degeneration of the spino-cerebellar tracts, and of sclerosis of the posterior and lateral columns of the spinal cord.

Etiology. It commences during the early years of life, most commonly between the seventh and seventeenth years. It affects males rather more often than females. It can scarcely be called an hereditary disease, as it is rarely seen in parent and offspring. It is more strictly a familial disorder, affecting several members of the same family. It would therefore appear as if there was a congenital tendency rather than a direct inheritance to the malady. Consanguinity, alcoholism, and syphilis have been noted in the parents in several cases; but no constant causal factor has been ascertained. It is rare to find any exciting cause for the disease.

Pathological anatomy. Degeneration is found in the posterior columns, the spino-cerebellar, and the pyramidal tracts. Both sides of the cord are symmetrically degenerated, and the dorsal region is often more affected than the other portions.

In the posterior columns, the postero-internal tract is more sclerosed than the posterior root zone. The cells and fibre network of Clarke's column are degenerated, a condition which is associated with sclerosis of the direct cerebellar tract and of Gowers's tract, the latter in more advanced cases.

The cerebellum only exceptionally shows atrophy of the Purkinjé cells, probably secondary to sclerosis of the spino-cerebellar tracts.

Three explanations have been given to account for the changes observed in the spinal cord in this disease. First, a tendency for degenerative changes to occur in the peripheral parts of the cord, which are less freely supplied with blood than the central areas (Williamson). Secondly, an inherited tendency towards general early vascular deterioration (Pitt).

Thirdly, abiotrophy, or an inherent tendency towards early death of the nerve fibres (Gowers).

In view of the pathological findings in some of the allied disorders about to be described, we are of opinion that the last of these theories affords the most probable explanation.

Symptoms. The disease commences slowly and gradually, with unsteadiness and inco-ordination, resulting in an instability both in standing and walking, which may bring about frequent falls, and oblige the child to abstain from playing with other

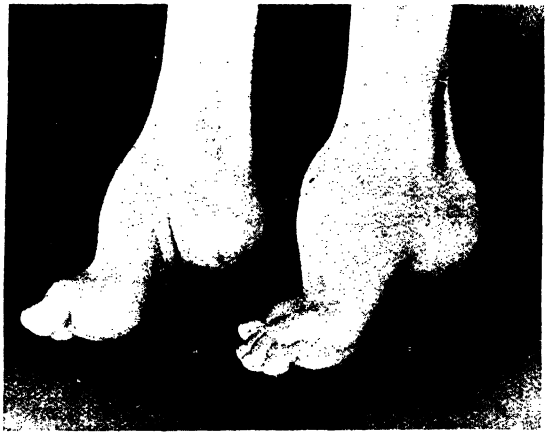


FIG. 161.—Illustrates the characteristic appearance of the feet in Friedreich's disease.

children. The gait is irregular, the feet are set far apart, and the movements are clumsy and inco-ordinate. As time goes on, the patient may require the aid of sticks or crutches, but gradually inco-ordination affects the muscles of the arms and trunk, so that eventually walking becomes impossible. In this stage tremor and unsteadiness of the trunk, arms, and head are observed, and as a rule definite and characteristic deformities of the back and feet.

The deformity first seen is pes cavus, a condition in which the foot is slightly dropped and foreshortened, the heel drawn up, the dorsum arched, the sole hollowed out, the toes hyper-extended at the metatarso-phalangeal, but flexed at the inter-phalangeal joints. Over-extension of the great toe is especially striking. The digital end of the foot is square.

When looked at in profile from the inner aspect, the foot presents a rough Z-shaped appearance. This deformity may be present before any considerable degree of unsteadiness or inco-ordination is noted. (Fig. 161.)

The spinal deformity which is characteristic of the advanced cases develops later, and consists of lateral curvature (scoliosis) with occasionally kypho-scoliosis. (Fig. 162.) In the hands minor degrees of the 'main en griffe' may be observed, but this is relatively rare.

The speech is characteristic, and is of a hesitating, syllabic, explosive order. It results from inco-ordination of the articulatory and respiratory muscular mechanisms. On attempting to speak, the expiratory and facial muscles are thrown into strong contraction, but the glottic opening remains closed. After an effort the air passes through the glottis, giving vent to a loud explosive sound.

FIG. 162.—Photograph of a case of Friedreich's disease showing the spinal curvature (scoliosis).

Mental condition. Some degree of mental dullness is usually observed, not amounting to mental deficiency, but rather to a want of mental acuteness and activity. The disposition is happy and placid, often approaching childishness.

Cranial nerves. Smell, taste, and hearing are not affected. Vision may be impaired. Although optic atrophy was stated never to occur, several cases are now on record in which it has been observed; its occurrence, however, is rare. There is no paralysis of the ocular muscles, but a weakness of sustained conjugate movement is occasionally found. Nystagmus is a common symptom occurring in the form of slow nystagmoid jerkings of the globes in all directions. The pupils are normal in size, shape, and reaction, but in a few cases inequality and impaired light reaction have been observed in association with changes characteristic of congenital syphilis.

The bulbar nerves are not paralysed, but inco-ordination of

the muscular movements gives rise to a peculiar form of articulation, which has been already described (p. 476).

Motor system. At first there is no loss of muscular power, but in the later stages a gradual and obvious impairment develops, which becomes marked in the terminal stages. It is probably owing to the motor weakness that the deformities of the feet, spine, and hands are brought about. Slight general muscular wasting is found, but there is no atrophy, fibrillation, or electrical changes characteristic of muscular degeneration. Hypotonia is usually present, especially in the early stages, but during the later spasticity and contractures may supervene.

Sensory system. In a certain number of cases complaint is made of cramp-like or darting and dull aching pains, but these are never severe. In most cases no objective loss of sensation is detected; loss of the sense of position is quite exceptional, but slight impairment of both the superficial and deep forms of sensibility may sometimes be detected.

Reflexes. The deep reflexes may at first be unaffected, but later become diminished and eventually abolished. The plantar reflex is extensor in type, and the superficial epigastric and abdominal reflexes are usually lost.

The *sphincters* are rarely affected. Trophic changes, though not unknown, are uncommon. Visceral crises are never present.

An abnormal type of the disease is seen in cases which commence with symptoms of ataxic paraplegia, nystagmus, articulatory defects, increase of the deep reflexes, extensor responses, and sphincter trouble. When seen in the early stage, these cases present a typical picture of disseminated sclerosis, but occurring at an age when disseminated sclerosis is rare or unknown. In two such cases, the subsequent loss of the deep reflexes and the development of characteristic deformities changed the picture of disseminated sclerosis into one of Friedreich's disease.

The **differential diagnosis** has to be made from juvenile tabes (p. 399), disseminated sclerosis (p. 418), Pott's disease (p. 357), and cerebellar tumour (p. 262).

The **course** of the disease is chronic, the symptoms lasting for thirty or more years. Death results from intercurrent

affections, not infrequently from cardiac failure, independent of valvular disease.

The treatment is limited to the alleviation of symptoms as they arise, in association with massage, passive movements, and Frænkel's exercises (p. 399).

CHRONIC DISORDERS GIVING RISE TO SYMPTOMS OF CEREBELLAR DISEASE

Under the title of Hereditary Cerebellar Ataxia, Marie described a series of cases, collected from the literature, in which signs of cerebellar disease occurred, sometimes in families and usually starting in early adult life. As Holmes[1] has shown, the anatomical investigation and the clinical study of the cases do not justify the application of this term, and he states that no form of disease exists to which this designation may be fitly given. The following classification, suggested by Holmes, would appear to be at once the most natural and the simplest wherewith to study these disorders.

The cases fall into the following four subdivisions:—

1. Primary parenchymatous degeneration of the cerebellum.
2. Olivo-ponto-cerebellar atrophy.
3. Degeneration of the spino-cerebellar tracts.
4. Congenital smallness of the central nervous system, associated with cerebellar symptoms.

These disorders are all characterised by a symptomatology, which does not differ materially in its main features in any of the particular varieties.

The general symptoms may be stated to be—

(*a*) A reeling, staggering, or drunken gait characteristic of cerebellar disorders; (*b*) irregularity, uncertainty, and sometimes tremor in the movements of the arms; and (*c*) a hesitating, scanning, and usually explosive form of articulation. Certain individual peculiarities have also been observed, to which brief attention will presently be directed.

The underlying pathological changes are in some cases degeneration or sclerosis of the cortex of the cerebellum. In other cases the sclerotic change is confined to some portion of the afferent or efferent cerebellar systems.

[1] Holmes, *Brain*, 1907.

1. Primary progressive cerebellar degeneration (Holmes)

This is a familial disorder, occurring in adults between the ages of thirty and forty, and progressing slowly to a fatal termination after a number of years. Cases of a somewhat similar type have also been described by Fraser [1] as occurring in children. Its primary and outstanding features are: (1) a reeling or staggering gait; (2) inco-ordination and uncertainty in the movements of the arms; (3) a hesitating, scanning, and explosive articulation; and (4) tremors of the head and limbs and nystagmus. No motor or sensory paralysis has been observed, nor is there any organic change in the reflexes. The mental condition is unaffected.

Microscopical examination reveals a primary and progressive degeneration of the cortex of the cerebellum, shown mainly in a disappearance of the cells of Purkinjé and their efferent fibres. A secondary neuroglial proliferation and sclerosis are found in the subcortical cerebellar white matter. The spino-cerebellar and efferent cerebellar tracts are intact.

2. Olivo-ponto-cerebellar atrophy (Thomas)

This condition is neither hereditary, familial, nor congenital. It commences at a late period of life and progresses to a fatal termination. It is characterised clinically by defective equilibration in standing and walking, inco-ordination and tremor of the arms may or may not be present, articulation is slow and scanning, and nystagmus is usually observed.

The pathological lesions are atrophy of the cerebellar cortex, degeneration of the inferior olivary bodies, and atrophy of the grey substance or nuclei of the pons. The middle cerebellar peduncles are completely, and the restiform bodies partly, degenerated.

In other cases, which seem to form a connecting link between Friedreich's disease and that next to be described, Thomas and Menzel found, in addition to the changes just recorded, an atrophy of the spino-cerebellar tracts, with which was associated some degree of degeneration of the posterior

[1] Fraser, *Glasgow Medical Journal*, 1880.

and lateral columns of the spinal cord. This type was seen in younger patients, was a familial disorder and presented, in addition to the cerebellar symptoms, spasticity of the legs and increase of the deep reflexes.

3. Degeneration of the spino-cerebellar tracts (Klippel and Durante; Sanger Brown)

This is a familial condition, coming on between the ages of sixteen and thirty-five. It is characterised by a reeling cerebellar gait, without any tendency to Rombergism, ataxia of the legs, arms, facial and ocular muscles, nystagmus, and indistinct and scanning articulation. Sometimes failure of vision from optic atrophy is observed. There may or may not be interference with the pupillary light-reflex. Rigidity, weakness of voluntary movements of the limbs, and contractures of the legs are seen in the later stages; but, except for the contractures, no deformities are present. The tendon reflexes are normal or exaggerated.

The outstanding pathological feature in these cases, of which quite a number have been examined, was observed to be a complete degeneration of the dorso-cerebellar tract of Flechsig, with degeneration and atrophy of the cells of Clarke's column. The ventro-cerebellar, or tract of Gowers, was also affected, but to a less extent. The posterior columns of the cord were in part degenerated. The nervous system generally was small, but very little alteration was observed in the cortex of the cerebellum.

4. Congenital smallness of the central nervous system with cerebellar symptoms (Nonne)

This is probably a developmental condition, and the symptoms arise during puberty or adolescence. These are uncertain gait, explosive articulation, nystagmoid jerkings of the eyes, and optic atrophy. The mental condition may also show deterioration. The whole central nervous system, especially the cerebellum, is unusually small. No definite degeneration of the nervous system has been detected.

HEREDITARY [FAMILIAL] SPASTIC PARAPLEGIA

This disorder is characterised by the gradual onset of spastic paralysis, which commences in early life from seven or eight years onwards. In some cases it is hereditary, but more commonly familial, while not a few instances of sporadic occurrence have been recorded.

FIG. 163.

FIG. 164.

FIGS. 163 AND 164.—Illustrate spastic contractures in a case of family spastic paralysis.

Spastic paralysis affecting several members of one family may be due to an individual cause affecting all the children of certain parents, without being in any degree truly hereditary.

The morbid changes consist of degeneration in the crossed pyramidal tracts, and sometimes also of the postero-internal column.

Symptoms. In its pure form the disease is characterised by the development of a spastic paraplegia, chiefly affecting the lower limbs and sometimes the upper, without involvement of

the face. The deep reflexes are increased, extensor plantar response is present, and in some cases slight sphincter trouble. The mental condition is good. Ocular symptoms are not present. There is neither inco-ordination nor loss of sensation.

The malady is progressive, increasing to complete paralysis with spasticity, adductor spasm, and drawing up of the heels.

Its distinction from cerebral diplegia is based upon the later onset of the paralysis, the absence of mental enfeeblement, and the slowly progressive character of the symptoms.

In a second type, in addition to the spastic symptoms, deformities of the feet, instability, and slight degrees of inco-ordination have been described. Cases have also been recorded in which wasting of the small muscles of the hand has been observed.

There are on record cases of family disease in which the clinical picture has shown every grade of variation, from a purely spastic type to the well-marked 'complex' of Friedreich's disease and 'hereditary cerebellar ataxy.' The class into which many of the cases may be placed varies with the progress of the malady.

Although certain definite types may therefore be recognised, it is only the post-mortem examination which can decide the final classification.

AMAUROTIC FAMILY IDIOCY

This is a rare disease, characterised by the gradual onset of blindness, with mental and physical impairment, and ending invariably in death.

It is practically confined to children of Jewish parentage, and may occur in more than one member of the same family. It is characterised by disappearance of the cortical cells, and by some degree of degeneration of the myeline sheaths of the cortico-spinal efferent system.

The child is apparently healthy at birth, but at or about four months of age paralytic weakness appears in the muscles of the neck, body, and limbs. At first the palsy is of the flaccid type, but soon some degree of spasticity makes its appearance with wasting, more especially of the muscles of the hands. Concurrently with the motor symptoms, a change

in the mental state of the child is noted by the mother. It ceases to take interest in its surroundings, becomes apathetic, and shows signs that vision is also impaired. This progresses to complete blindness; and with the increase in the loss of vision, the muscular paralysis becomes more definite and spastic. The reflexes remain unaffected.

The ocular phenomena are characteristic of the malady. At the macula a cherry-red spot is seen, surrounded by a white halo. There is atrophy of the optic discs, and vision is impaired or altogether abolished. The pupillary light reaction is defective, and nystagmus may appear later.

The malady runs a course of about two years, and invariably terminates fatally.

No treatment has proved of any avail in arresting the disease. It does not appear to have any relation to congenital syphilis.

Its nature is still a subject of discussion. Four theories have been advanced to account for it: (1) cortical agenesis (Sachs); (2) primary cortical degeneration (Russell and Kingdom); (3) a toxic degeneration of the motor neurones (Hirsch); and (4) degeneration resulting from an inherent bio-chemical property of the protoplasm of the nerve cells (Holmes).

HEREDITARY CHOREA. HUNTINGTON'S CHOREA

This is a rare disease, superficially resembling chorea minor, but occurring in adults in whose ancestry it has also been found. It is characterised by the presence of involuntary, purposeless movements in conjunction with psychical symptoms, which progress towards dementia.

According to Huntington, three features characterise the disease: its hereditary nature, its manifestation in adult life, and a tendency towards mental impairment and suicide.

Etiology. The existence of the disease in one or other parent is constant. Should it, however, miss a generation, it is unlikely that it will reappear in succeeding generations. Several members of the same family and generation may be subject to it. The most common age for its appearance is between thirty and forty. It never commences before thirty, but may develop after forty.

Symptoms. The motor symptoms resemble those seen in chorea minor. They are of an involuntary, purposeless character, affecting different groups of muscles, but rarely presenting the quick, jerking character of the movements of true chorea, and often distinctly inco-ordinate rather than choreiform (Osler). They cease during sleep and are accentuated by excitement.

In the face grimaces and gesticulations are common. The speech is slow and hesitating, and eventually becomes indistinct. The gait is erratic, the patient for instance suddenly stopping and resuming after a short interval. On the other hand, it may be swaying and unsteady, the feet being set far apart and the arms in constant motion.

The psychical features are, in the early stages, irritability and depression, often leading to suicide; and in the later, definite mental enfeeblement progressing to dementia.

The **duration** of the disease is prolonged, sometimes to thirty years. It is incurable, the tendency being towards loss of motor power, suicide, or dementia.

PART XII

DISEASES CHARACTERISED BY DISORDERS OF MUSCULAR FUNCTION

MYASTHENIA GRAVIS

This is a subacute or chronic disease, characterised clinically by the rapid development of muscular fatigue and weakness on exertion. It affects most commonly the bulbar, facial, and external ocular muscles, but may be general in distribution.

Etiology. It occurs about equally in the sexes, with, if anything, a preponderance in the female sex, attacking women at a somewhat earlier age than men. It may come on at any time during adolescent or adult life. The malady is neither hereditary nor familial.

Pathology. Changes in the nervous system are neither constant nor striking. In one case, described by Buzzard,[1] small collections of lymphocytes were observed scattered between the nerve cells of the bulbar nuclei—these were similar to the 'lymphorrhages' seen in other tissues—and in another case a similar condition was observed.

The most prominent change is the presence of widely distributed cellular and sometimes serous exudations (lymphorrhages) in the tissues and organs of the body, especially of the muscles, liver, and kidneys. In the muscles, slight fibre alterations, suggestive of toxic degeneration, are frequently present. Pronounced muscular atrophy is rare.

Proliferative and degenerative changes, and new growths in the thymus gland, although not constant, have been observed in a number of cases.

[1] Farquhar Buzzard, *Brain*, 1905.

The most likely explanation is that the disease is due to the presence of a toxic or autotoxic agent, which has a special effect upon the protoplasmic constituents of the voluntary muscles. It is possible that the 'lymphorrhages,' which have been frequently observed, are an expression of the action of this toxic agent. The rôle played by the thymus gland in the production of the disease is not clear.

Symptoms. The symptoms presented by this disease vary considerably, not only in different cases, but in each individual case. There are, however, certain features which remain common to all cases, namely, weakness of the facial and external ocular muscles, with or without weakness of the palatal and articulatory muscles. This weakness is increased or brought on by fatigue, and, as a rule, is worse at the end of the day. It may give rise to slurred speech, impairment of mastication and swallowing, and in some cases to diplopia.

These symptoms, at first transient, may be ascribed to hysteria in the absence of definite weakness, but every case should be examined, and if this is done and an electrical examination of the muscles made, the true nature of the condition will be at once revealed. There is often superadded a general weakness and feeling of fatigue and tiredness, with heaviness of the limbs, and sometimes dull aching pains.

In any particular case the local incidence of weakness may be determined by the occupation, which may necessitate the employment of one group of muscles more than another. For example, a painter whose work necessitated his looking up and using his right arm, developed weakness of the ocular and retrocollic muscles, and of the right deltoid muscle. A teacher who had to talk during the day found that towards evening she could hardly make herself understood, her tongue felt too big for her mouth, speech was slurred, and mastication was impaired. As a third example, the case of a postman might be cited, who first developed the weakness in his legs.

It is only in the early and slight cases that there is any difficulty in recognizing the condition, and even in these cases the history and the facial expression may suffice to point to the nature of the disease, and permit the confirmation of the diagnosis by electrical examination and the discovery of the 'myasthenic reaction.'

Three types of the disease are found: —

1. Cases in which the symptoms are transitory and the characteristic facies absent.

2. Cases in which the facial, ocular, or bulbar symptoms are well marked, but do not tend to progress, and in which general weakness is not a prominent symptom.

3. Cases in which, in addition to the facial, ocular, and bulbar symptoms, marked general weakness, impaired action

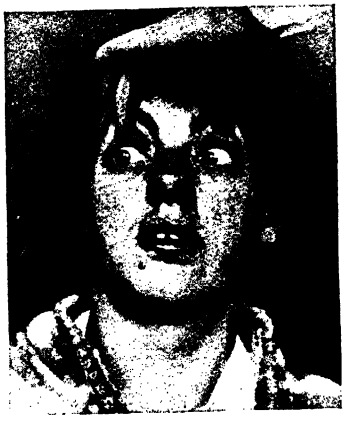


Fig. 165.

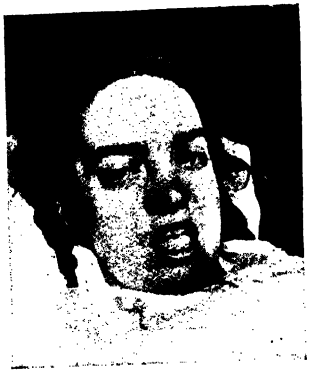


Fig. 166.

Two figures illustrating the facial appearance in a case of myasthenia gravis. Fig. 165 shows external ophthalmoplegia on attempting to look to the right. Fig. 166 shows the myasthenic smile.

of the respiratory muscles, and sometimes dilatation of the heart are present.

The prognosis as regards life is good in the first two types, but bad in the third, death usually occurring suddenly from respiratory failure within two years of the onset of the disease. In all cases there is a tendency to sweating and vaso-motor disturbances, and in women the symptoms are always aggravated by the catamenia.

The **mental** condition is unimpaired, except that myasthenics are often highly emotional and easily upset by trifling circumstances.

Motor system. *Cranial nerves.* Ptosis, usually bilateral and more marked on one side, is a common symptom. It is more pronounced towards the evening. The drooping of the

lids is to some extent counteracted by a tilting of the head backwards, but the over-action of the frontalis, so common in tabetics, is not observed in myasthenic patients.

Diplopia and strabismus from weakness of the external rectus muscle are not uncommon, and in some cases complete ophthalmoplegia externa has been noted. There may, or may not, be nystagmoid movements on extreme deviation of the globes, and the ocular movement is often ill-sustained and easily fatigued. The pupils retain their reaction to light, and the accommodative action of the ciliary muscle is unimpaired. (Fig. 165.)

Weakness of the muscles of mastication is very common, and the mouth is sometimes kept slightly open from a falling down of the lower jaw.

On looking at one of these patients, the facial expression at once attracts attention. At first sight it suggests the myopathic face. The forehead is smooth, slight ptosis is present, and the head gently tilted backwards. The mouth and lower part of the face, when at rest, give the impression of a face characteristic of disgust, the upper lip being slightly retracted, the corner of the mouth drooped, and the lips parted. On smiling, this appearance is exaggerated, the upper lip is curled, the corners of the mouth remain drooped, and the smile has the appearance of a sneer. (Fig. 166.)

On testing voluntary power, the forehead cannot be wrinkled, nor the eyelids firmly closed. Neither whistling nor blowing out the cheeks can be accomplished.

Bulbar symptoms consist of difficulty in swallowing, regurgitation of fluids through the nose, and a nasal tone of speech.

Paralysis of the vocal cords is rare, but some paresis or weakness of the abductors, giving rise to stertor, has been observed.

The tongue may or may not be affected, being less commonly implicated than the other neighbouring muscles. Its appearance, however, is characteristic. In rare cases it has been found atrophied. As a rule it is soft, flabby, tremulous, indented by the teeth, and has three well-marked furrows in the longitudinal direction, one median and two lateral.

Speech may be normal or slightly nasal in tone. Where any defect exists, it may be accentuated and made more

obvious by inducing exhaustion from repetition of words or phrases.

Trunk muscles. The muscles of the neck are often implicated at an early stage. Owing to this, the head tends to fall forwards or backwards. Both the back and abdominal muscles are affected, and more especially the intercostals and other respiratory muscles, so that dyspnœa on exertion and difficulty in breathing are frequent.

The extremities. The most marked weakness is found in the shoulder muscles. This is readily brought out by making the patient maintain the arms abducted horizontally from the side, if necessary against resistance. This leads to rapid fatigue, and finally inability to raise the arms at all. The grasps are feeble and may be easily exhausted. As in the arms, so also in the lower limbs, the proximal muscles, especially the flexors of the hip joint, are readily affected. This, in conjunction with general weakness of the lower limbs, gives rise to a peculiar combination of waddling and steppage gait. In all severe cases, where such deficiencies exist, dyspnœa on exertion effectually hinders progression. This weakness is most marked in the muscles chiefly employed, local effort producing not only local exhaustion, but also an abnormal degree of general muscular fatigue. Muscular weakness gives rise to subjective sensations of aching, pain, and dragging.

The myotatic irritability is increased in duration, but reduced in briskness. Thus a tap on a muscle may induce a slow but well-sustained muscular reaction.

The electrical reactions. A tetanising faradic current induces at first a brisk muscular response, which, however, tends to fade and finally disappears on the continuance of the current. On reapplying the electrodes after a period of rest, a good contraction is again obtained, which also eventually disappears. This reaction is not obtained by the galvanic current.

Similar results may be obtained by isolated faradic shocks, but the application of rapid, interrupted galvanic shocks failed to produce any exhaustion, at least in two cases in which it was tried. Exhaustion of the muscles by faradic currents, in the majority of cases, induces a weakness of voluntary effort; and fatigue induced by voluntary effort certainly causes a more rapid faradic exhaustion.

The above-described myasthenic reactions vary to a great extent, both from day to day and in different muscular groups. It is most characteristic and constant in the facial muscles.

The absence of a myasthenic reaction does not negative the existence of this disease.

Sensory system. There is no loss of sensation, but subjective feelings of aching and pain in association with muscular exhaustion are not uncommon. Occasionally slight muscular hyperæsthesia to deep pressure has been met with (Buzzard).

Reflexes. The *deep* reflexes are brisk, normal or subnormal, and in severe cases may be exhausted by repeated tapping. The *superficial* reflexes are usually increased. The plantars are of the flexor type.

The **sphincters** are never affected.

Vaso-motor functions. Flushing and sweating are common.

In severe cases dilatation of the heart occurs with the development of an apical systolic murmur, but this condition may vary from time to time.

Respiratory complications are invariably fatal.

Pregnancy may occur and labour be accomplished normally, but their influence upon the disease is unfavourable.

Diagnosis. Myasthenia has to be distinguished from several conditions, of which the more important are hysteria, the myopathies, bulbar paralysis and tumour of the pons.

1. *Hysteria.* The slighter forms of myasthenia may simulate hysteria, but the distribution of the weakness, its onset after fatigue and towards evening, the absence of sensory loss or of hysterical seizures, and the presence of the myasthenic reaction are usually sufficient guides.

2. *Muscular dystrophy.* Myasthenia is only liable to be confused with the facio-scapular type, and is to be distinguished from it by the absence of muscular atrophy and hypertrophy, by the relapsing character of the weakness, and by the presence of the myasthenic reaction. The characteristic degenerative electrical changes of the muscular dystrophies are absent.

3. From *bulbar palsy* and from *focal lesions of the pons and medulla* it is distinguished by the absence of atrophy, the age of the patient, and the remission of the symptoms after fatigue.

4. From *chronic external ophthalmoplegia*, by the history of

the onset, by the affection of other parts of the body, by the influence of fatigue, by the remittent character of the symptoms, and by the presence of the myasthenic reaction.

Prognosis. The outlook is bad as regards recovery from the disease.

In the milder forms the duration of life is quite uncertain, but patients have been known to live for fifteen years after the onset of the symptoms.

In acute cases, especially those in which attacks of respiratory difficulty are common, the duration of life rarely exceeds two years.

Cases presenting the symptoms of the disease in a well-marked form may, with care and the avoidance of exertion, live for an indefinite number of years.

Treatment. Rest should be advocated, and in the severe cases rest in bed is essential. In addition, general tonics, especially strychnine hypodermically, are recommended. Warm clothing and the avoidance of chills and respiratory complications are important. All forms of electrical applications and massage should be avoided. Sodium sulphite deserves a wider trial.

PARAMYOCLONUS MULTIPLEX

This is a rare condition, characterised by the repeated occurrence of short, sharp, shock-like clonic contractions of single muscles or portions of muscles, more rarely of groups of muscles. The contractions resemble those produced by the application of a faradic current. They may occur in any of the muscles of the body, but predominate in the supinator longus, biceps, trapezius, pectorals, recti abdominales, quadriceps extensor cruris, and semi-tendinosus.

The mental state is not affected. The contractions do not give rise to pain, but occasion great distress from their inconvenience.

The condition is bilateral, and a muscle on one side is often observed to contract immediately after its fellow of the opposite side. The shock-like movements are increased on emotion and disappear during sleep. They do not interfere with active movements. They may occur at different times in different parts of the body. The rate of contraction varies widely, but

as many as a hundred contractions may be observed in a minute.

On examination, apart from the clonic tremor, the motor functions are normal. The sensory system is unaffected. Handling the limbs may increase the contractions. The deep and superficial reflexes are increased. There is no sphincter trouble. The electrical irritability of the muscles is unaltered.

The condition has arisen after trauma, acute infective diseases, and fright.

The prognosis is grave as regards recovery.

An *hysterical* type has been described, in which the movements are of larger range, affecting groups rather than single muscles, and not involving bundles of fibres. This condition is probably quite distinct from the true form, and the prognosis is good. There may be other associated hysterical stigmata.

A combination of *myoclonus* and *epilepsy* occurring in families has been described by Unverricht. In it the spasm affects the muscles of the body, limbs, tongue, pharynx, palate, and diaphragm. This type has also been observed unassociated with epilepsy, but with mental enfeeblement and backwardness.

Part XIII

DISEASES OF OBSCURE ORIGIN CHARACTERISED CHIEFLY BY DISORDERS OF MOTION

Chapter I

PARALYSIS AGITANS

[SYN.: PARKINSON'S DISEASE]

This is a progressive disease, usually characterised by muscular rigidity, which gives rise to a mask-like expression of face and a typical attitude and gait, but frequently attended by the presence of a rhythmic tremor.

Etiology. The disease commences most commonly between the ages of forty and seventy, the majority of cases arising in the middle period between fifty and sixty. It is rare under forty years, but cases are from time to time seen in which the symptoms have arisen under thirty.

Heredity plays little or no part in its causation. The hereditary feature, which we have found most constant, is a tendency towards longevity in one or both parents. In a number of cases in which this factor was ascertained, the age at death of one or other parent ranged from seventy to ninety years.

There is no doubt that severe mental strain, shock, and worry have an important influence in its causation, many cases commencing definitely after the strain of nursing relatives and the anxiety of the death-bed scene.

Traumatism may also play a part, and sometimes determines the limb in which the tremor starts.

There is an occasional relation between vascular degeneration and the onset of the disease.

Pathology. No special or constant pathology of paralysis agitans is known, although various morbid changes have been found in those dying of this disorder, such as degenerative changes in the walls of the smaller arterioles, pigmentation and vacuolation of the ganglion cells, and increase of the neuroglial tissue. But these alterations are found in other conditions, and are usually the result of senile changes.

The absence of definitely degenerative alterations in the muscles, and the onset of the tremor occasionally as a result of nervous shock, and sometimes of injury, would seem to suggest a psychical basis of the symptoms in some cases. On the other hand, evidence has been advanced by some writers to show that the disease is primarily a muscular condition.

Symptoms. Although the term 'paralysis agitans' suggests tremor as the outstanding feature of the disease, this symptom is in reality only one of its several manifestations, and is by no means an early sign. Two chief types are found:—

(*a*) A type characterised by the early appearance of rigidity, which shows itself primarily in the expressionless face and the well-known attitude and gait. This is usually accompanied by paræsthesia, subjective sensations of heat and cold, and aching and stiffness of the muscles, chiefly of the neck and shoulders.

(*b*) The other and less common type commences with either slight or well-marked tremor, unattended by rigidity or sensory symptoms.

In most cases the first symptom is rigidity, to which tremor is added in the later stages.

The *mental condition* is not usually impaired, but attacks of despondency and depression are not uncommon. This mental attitude is frequently accentuated by the lack of expression on the patient's face, which is characterised by a mask-like appearance and a failure of emotional display.

Cranial nerves. The most striking feature on examining the cranial nerves is the fixed, intense, and anxious facial expression, arising from the rigidity of the facial muscles and the wiping out of the emotional movements. In some cases tremor of the lower jaw and tongue are observed. The articulation tends towards a monotone, and although each

word is clearly and distinctly pronounced, there is no inflexion. In the later stages the voice is high pitched, giving rise to 'the piping voice.' The ocular movements are good, but in some cases weakness of the convergent action of the eyeballs has been noted.

Motor system. Rigidity, not amounting to spasticity, is the chief feature. The attitude is quite characteristic of the malady. Weakness and difficulty of movement are associated

FIG. 167.—The face in a case of paralysis agitans.

with rigidity and directly due to it. The muscular actions are slow and the grasps feeble. Motor weakness is slight and rarely advances to paralysis, and even in the late stages some movement is retained. The muscles retain their normal volume and their electrical irritability.

Rigidity. This is the characteristic feature of the disease. It is usually the earliest symptom, and affects the thumb or forefinger before any tremor makes its appearance. Cases of this early type usually present considerable difficulty in the diagnosis. (Fig. 168.)

To the rigidity are ascribed many of the most typical features of the malady, such as the mask-like facial appearance, the forward position of the head, the rigid and curved

back, and the posture of the hands with the fingers flexed at the metacarpo-phalangeal and extended at the inter-phalangeal joints. The appearance of the hands closely resembles that seen in rheumatoid arthritis, a condition which is not rarely associated with it. The legs are slightly flexed at the knees and the thighs adducted, and a tendency to talipes may develop.

In consequence of this attitude, the gait is slow and hesitating; having started, however, the patient may progress with increasing rapidity, as if he were running after his centre of gravity (festination). In some instances the converse condition, in which there is a tendency to run backwards, has been observed (retropulsion).

FIG. 168.—Characteristic attitude of paralysis agitans.

Tremor. This is coarse and rhythmical, characterised by alternating opposition and extension of the thumb, or flexion and extension of the fore or other fingers, producing the well-known 'cigarette rolling' or 'bread crumbling' movements. Other movements are flexion and extension at the metacarpo-phalangeal joints, or at the wrist, or pronation and supination movements of the forearm, or all these movements may be combined at one time. Such movements may be seen, from a slight but characteristic tremor up to marked shaking of the whole arm. In the lower limb the tremor may be flexion and extension at the ankle joint, so as to produce a tapping sound when the foot is at rest upon the floor. Movements of the lower jaw are less frequently observed, and are usually due to definite tremors in the mylohyoid and digastric muscles.

To-and-fro movements of the head are sometimes seen, but rotation movements are rare. Tremor may also implicate the lips and tongue.

The special characters of this tremor are, in addition to its rhythmic and usually coarse features, its continuance during repose, and its temporary suspension during the

FIG. 169.

FIG. 170.

FIGS. 169 AND 170.—Two figures illustrative of the characteristic attitude of the head, limbs, and body in paralysis agitans.

execution of voluntary movements. It may also be temporarily arrested by an effort of will. Arrest of the movements in one limb may induce an exaggerated tremor in another limb. In the later stages, however, the tremor cannot be voluntarily arrested, and is sometimes found to show exaggeration on volitional and emotional movements.

The onset of the tremor is usually insidious, and most

commonly in the thumb or fingers. If it commences in the upper limb, the lower limb upon the same side is involved before the limbs upon the opposite side. It affects the proximal rather than the distal portions of the lower limb.

In those cases in which tremor is an early and prominent feature, rigidity is rare, and the characteristic facial expression and attitude are late in appearing, or even absent. In this type the range or excursion of the tremor is larger and more irregular.

Complaint is made of various paræsthesia. Amongst these

FIG. 171.—The appearance and position of the hands in paralysis agitans.

may be mentioned abnormal sensations of heat and cold unassociated with any rise of temperature, and aching sensations about the limbs and back of the neck.

The deep *reflexes* are brisk, but ankle clonus is never present. The superficial reflexes are unchanged and the plantar is of the flexor type.

Sphincter control is retained to the end.

Restlessness at night, sometimes accompanied by insomnia, may be present.

The **differential diagnosis** has to be made from senile tremor, double hemiplegia, bilateral cortical degeneration, and focal lesions of the mid-brain.

Senile tremor is characterised by its occurrence in extreme old age, its irregularity, its bilateral distribution, by an

absence of rigidity, and by the early involvement of the head.

Double hemiplegia is distinguished by the presence of reflex changes characteristic of organic disease, notably the extensor plantar response, by the excess of weakness over rigidity, and by the absence of tremor and the presence of exaggerated emotional display.

Bilateral cortical degeneration most closely resembles paralysis agitans, inasmuch as a slowly progressive rigidity with motor weakness is present. Although the facial expression has a superficial resemblance to paralysis agitans, on closer examination the mask-like expression is seen to be due to fixation of the normal lines. The reflexes are at first normal, as in paralysis agitans, but eventually become characteristic of organic disease. The mental condition shows a steady deterioration, with spasmodic laughing and crying.

In *Focal lesions of the mid-brain* the onset is sudden, the symptoms are unilateral, impairment of the upward movement of the eyes and of the pupillary light reaction are present, and a tendency towards subsidence of the symptoms develops in course of time.

Prognosis. It is a chronic progressive disorder, of long duration, and having little tendency to cause death. It has been known to last from fifteen to thirty years, but the average duration is from ten to twelve years. Death is usually due to an intercurrent malady, such as malignant disease, pulmonary complications, or exhaustion.

Treatment. Paralysis agitans is a disease for which little can be done, even of a temporary character. In cases characterised by rigidity, massage, passive movements, and warm baths are of some use. In the tremulous forms, mild faradism applied to the limbs which are the seat of tremor is often beneficial.

Of drug treatment we have found most benefit from daily hypodermic injections of hydrobromate of hyoscine (gr. $\frac{1}{100}$ to $\frac{1}{50}$), especially in the early stages and in patients under fifty years of age. Few other drugs are of much service. Arsenic and strychnine are probably of most use in improving the general condition and relieving pain, and despondency. Temporary benefit may be derived from the bromides, chloral, and cannabis indica.

In those cases in which the gait tends to become festinant, the patient should be trained to take long steps by means of carefully regulated walking exercises. This will often materially improve the gait and enable the patient to get about with considerable freedom and comfort.

In view of the age at which the disease commences, it would seem advisable to try the effect of the organic extracts —thyroid, pituitary, ovarian, and testicular.

Chapter II

CHOREA

(SYN.: ST. VITUS'S DANCE. SYDENHAM'S CHOREA)

Chorea is a disease chiefly affecting children and young adults, intimately associated with rheumatic manifestations, and characterised by psychical changes and motor disturbances, the latter consisting of irregular, jerking, purposeless, semi-purposive, involuntary movements.

Etiology. Females are more frequently affected than males, in the proportion of about three to one. Its occurrence is most common between the ages of five and fifteen. It is rare after twenty, except when associated with pregnancy. A form occurring in old age, and known as senile chorea, has been described. It is much more common in hospital than in private practice. Its seasonal incidence is in striking correspondence with that of rheumatism, the period of greatest frequency commencing in December, reaching a maximum in March, falling in April, and showing a further rise in May, from which month a steady decline takes place to November.

A family disposition towards rheumatism and nervous affections are striking features in many cases. The patients are of the sanguine temperament.

Although emotion and fright have been extensively quoted as exciting causes, careful inquiry will bring to light the fact

that some signs of the disease had been previously observed. Amongst other ascribed causes, imitation, trauma, reflex irritation, and eye-strain are mentioned, but while these may have some influence in aggravating the symptoms, they are purely accidental and in no sense causal influences.

The relation between *rheumatism and chorea* was pointed out in France and in our own country many years ago; but attention has been lately redirected to it, and during the past few years observations have been made establishing an etiological relation between the two disorders. Chorea may precede, coincide with, or follow acute rheumatism. In many cases the association is indisputable, but in others vague pains and sore throat are the only previous rheumatic symptoms.

An analysis of our cases shows that out of 58 cases of chorea minor only 6, or 10·3 per cent., were without any personal or hereditary history or sign of rheumatism. Of the remainder 17, or 29 per cent., gave a history of rheumatic fever preceding the first attack of chorea, 15, or 25·8 per cent., gave an account of rheumatic pains, while 12, or 20·7 per cent., although presenting no personal sign or history of rheumatism, gave a history of rheumatism or chorea in their family. In two cases, in which there was no personal or family history of chorea or rheumatism, valvular disease of the heart was detected on auscultation.

A diplococcus, identical with that found by Poynton and Paine[1] in the blood and valvular vegetations in acute rheumatism, has been observed in the cerebral cortex, blood, and valves of the heart in cases of fatal chorea. The organism has also been obtained during life from the cerebro-spinal fluid in cases of chorea. This association is striking, and is made more so by the finding of the same organism in the brains of fatal cases of chorea occurring during pregnancy (Paine, Poynton, and Holmes).

Infectious disorders do not appear to play any active part in the production of chorea, except perhaps scarlet fever. On the other hand, the occurrence of an acute disorder during the course of chorea may induce a lessening of the disease.

Chorea and epilepsy. An intimate relation seems to exist

[1] Poynton and Paine, *Lancet*, 1901; *Brain*, 1905.

between chorea and epilepsy, but it is more common to find epilepsy mentioned in the family history of choreics than the reverse. Both chorea and epilepsy may predispose towards each other; and in neuropathic families epilepsy and chorea may be present in different members. Epilepsy and chorea may also be present, either simultaneously or at different times, in the same person. A history of infantile convulsions is not uncommon in those suffering from chorea.

Symptomatology. The symptoms of the disease usually set in more or less gradually, and are at first of a psychical nature. A change is noticed in the child's behaviour. He shows irritability of temper and peevishness, while his manner is nervous or apathetic. At this stage the malady is not recognised, and consequently the child is scolded or punished. He then becomes emotional and has fits of crying or of temper, with occasionally night-terrors and restlessness. He is fidgety and clumsy, especially at table or when under observation. Complaint may be made of headache or of pains in the limbs, especially the legs, followed by the typical movements of chorea, which explain the previous behaviour and symptoms. The hands are usually earliest affected, then the face, and later the legs. Very often one side is affected earlier and more extensively than the other, but as a rule the movements are bilateral, although in some cases they remain one-sided (hemichorea).

The facial appearance is characteristic. In nervous and high-spirited children the expression is one of overflowing nervousness, the eyes are bright and glistening, the face is continually being twisted into grimaces and is never still. The head is jerked about, and articulation may be impaired, especially when the right side is involved. If the child is standing, the arms are kept out of sight behind the back. The shoulders are wriggled, and the hands are seen to be jerked about with irregular, rapid, and in co-ordinated movements. These movements are not clonic or confined to one muscle, but are the result of contractions of groups of muscles, and have a purposive character, though their aim is purposeless. Mental excitement or attempts to perform fine movements result in their exaggeration.

In severer cases the movements are continuous, the patient being unable to walk or to lie still, and presenting a constant

whirlpool of restless motion. He has a wild, restless look, and herpes may be seen on the lips.

The maniacal cases are rare in children, but are more common in young adults and in pregnancy. As a rule they develop gradually out of a simple case, the patient either first becoming dull or heavy, with delusions, mental excitement, and inarticulate incoherent speech, or the primary restless state passes directly into one of delirium and mania. The patient is sleepless, and tosses about wildly in bed, with continuous movements of all parts of the body. In these cases the temperature may rise to hyperpyrexia.

The *mental condition* frequently shows some sign of impairment during the course of the malady. The facial expression is vacant, there is a failure of the power of attention, and often a want of emotional stability. In the early stages, irritability, obstinacy, and waywardness may reveal the fact that the child is not in its usual health. A form of chorea—chorea insaniens—is characterised by psychical features of an acute type, incoherence of speech, delusional and hallucinatory symptoms, and the usual phenomena of an acutely maniacal state.

Motor system. The most obvious affection is the occurrence of the characteristic involuntary movements already described. It may be stated that in the majority of cases the movements, which are rapid and jerky in character, are not truly clonic, as described by some writers. They are the consequence of the inco-ordinated action of groups of muscles, and as such are purposive in appearance, though purposeless in effect. It is true that in the face a twitching movement of one or more muscles may be observed, not amounting to a grimace, and it is not uncommon for tic-like movements to develop out of an attack of chorea and to persist for some time. These are not genuine choreic movements, but exhibit all the features of a tic (p. 584).

Choreic movements are not limited to any particular muscular group, but are observed now in one place, now in another. Voluntary action may be well carried out, provided that the performance of the act is not interfered with by the occurrence of involuntary movement. Realising this, the patient carries out the action hurriedly, in order to effect his purpose during a quiescent period.

The movements of chorea are increased by emotion and excitement, and in the slighter cases subside during sleep.

Motor power is impaired, and fatigue is easily induced. In some cases, especially in the unilateral types, this weakness is well marked, and is associated with the symptoms of an organic affection of the motor path. In one type of the malady—chorea mollis—an almost complete loss of power exists; in these cases voluntary movement is slight or absent, hypotonia is present, and the deep reflexes are abolished. This type of the malady is usually found in children of lymphatic temperament.

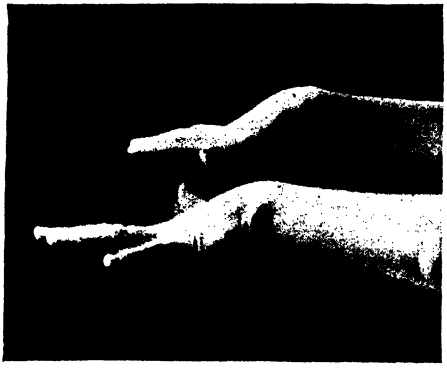


FIG. 172.—Illustrates a not uncommon appearance of the outstretched hands in a case of chorea.

The muscles are well developed, there is no atrophy, but a tendency to hypotonia is constant. The hand in chorea frequently shows a tendency towards hyper-extension of the proximal phalanges. (Fig. 172.)

The reflexes. The deep reflexes may be normal, exaggerated, or absent. A slight increase is the most common deviation from the normal. A more or less characteristic phenomenon is an almost tonic prolongation of the jerk, the response to the tap being slightly delayed, and the effect prolonged. They may be abolished in the paralytic form.

The superficial reflexes are brisk, but in the hemiplegic cases they may be lost or diminished on the affected side. In these cases an extensor plantar response is sometimes found.

Sensation. There are no objective sensory changes, but pains in the limbs and joints frequently precede or accompany the choreic movements.

The cardio-vascular system. An organic affection of the valves of the heart, more especially the mitral cusps, is especially frequent in chorea. According to Gowers, 90 per cent. of the fatal cases exhibit valvular disease and endocarditis. We found 50 per cent. of the ordinary cases of chorea

minor, examined in hospital out-patient practice, with valvular murmurs.

Although less common than endocarditis, the association of chorea with pericarditis has long been known.

In the cases of chorea minor which recover satisfactorily systolic murmurs are not uncommon, but in many cases these are of a hæmic character. In all cases, however, the heart should be carefully watched during the progress of the malady. The rarity of aortic disease, as compared with mitral regurgitation—an observation to which attention has been directed by other writers—was fully confirmed in our own series of cases.

The pulse may be irregular or intermittent, and is usually increased in frequency.

Various cutaneous rheumatic affections may also coexist, such as erythema nodosum and purpura rheumatica. Some degree of pyrexia is found in the severe cases, but in the common type of chorea minor the temperature is normal. When the temperature is raised, endocarditis or other cardiac complication is present.

Course and duration. Chorea is essentially a relapsing malady, the number of attacks varying from two up to six or more over a number of years. In some cases the movements never entirely disappear during the intervals. The frequency of periodic attacks may be seen from the following table, which shows the number of relapses in a series of fifty-eight cases of chorea minor:—

34	cases	with	1	attack.
13	,,	,,	2	attacks
4	,,	,,	3	,,
5	,,	,,	4	,,
1	case	,,	5	,,
1	..	..	8	..
58	cases	,,	105	,,

The duration of an average attack is commonly about three months, during which period it runs its course of incidence, full development, and gradual subsidence.

Death may be due to exhaustion arising from the intensity

of the motor disturbance and the difficulty in maintaining the strength by suitable nourishment, or from such complications as endocarditis, pericarditis, embolism, or hyperpyrexia.

Treatment. Rest in bed and ample nourishment are the two main factors guiding the management of all cases of chorea, whether slight or severe. Drugs may also be prescribed with great advantage.

Rest in bed, or, if the movements are severe, upon a mattress on the floor in the corner of the room, should be maintained until the movements have ceased. It may be satisfactorily combined with seclusion behind screens, as carried out in the wards of a hospital, or under the care and supervision of a nurse in private practice.

Abundance of nourishment, mainly milk and eggs, should be given. If the malady is severe, the patient may have to be fed by hand, or even by means of a tube passed through the nose.

Drugs are often of assistance in association with the above. In the definitely rheumatic cases, in which the onset of the disease has been preceded or accompanied by rheumatic pains or arthritis, the salicylates or aspirin are of great value. In our experience aspirin is more efficacious than the salicylates, and is usually well borne. It should be given in doses of 10 to 15 grs. every four hours for two or three days, and then reduced to thrice daily. In those cases without obvious rheumatic manifestations, the administration of arsenic (Fowler's solution in doses increasing from three to five, ten or more drops thrice daily) is recommended. The long-continued use of arsenic, even in small doses, should be carefully watched, as it is known to have induced gastro-intestinal symptoms, pigmentation of the skin, and arsenical paralysis, sometimes of an intractable character.

Chloral hydrate, in doses sufficiently large to induce sleep, has been found of use in chronic cases.

In the severe types of the disease, hydrobromate of hyoscine (gr. $\frac{1}{100}$ to $\frac{1}{75}$) given hypodermically is of much service. In these cases also alcohol may act as a most efficient sedative.

In cases with much psychical disturbance the bromides are of especial value.

During convalescence tonic treatment by means of cod-

liver oil and iron are essential. A tendency to tonsillitis or enlargement of the tonsils should also be attended to, as the presence of hypertrophied tonsils favours a relapse of the symptoms.

Chapter III

TETANY

This disease is characterised by the occurrence of paroxysmal or continued tonic spasms, bilateral in distribution, affecting chiefly the muscles of the extremities, but sometimes also those of the trunk and face, associated with a characteristic excitability of the nerves and muscles to mechanical and electrical stimulation.

Etiology. The disease is more especially one of childhood, and boys suffer more than girls. The underlying causes appear to be debility, and toxic poisoning arising from gastric and intestinal disorder. It is often associated with rickets and dilatation of the stomach. Amongst adults it has been observed in women debilitated during pregnancy or after prolonged lactation, and in other cases has followed directly upon the removal of the thyroid gland. An acute epidemic form has been described in Austria in which during the spring shoemakers and tailors were specially affected. Other cases have followed on poisoning by chloroform, lead, and ergot of rye.

Symptoms. The earliest sign of the malady is numbness and tingling in the hands and feet, followed shortly by the sudden onset of tonic spasm in the muscles of the extremities. The attitude of the hands and feet is characteristic. The fingers are flexed at the metacarpo-phalangeal and extended at the interphalangeal joints; the thumb and the points of the fingers are approximated, and the hand assumes a cone-shaped appearance. The wrists are flexed and pronated, the elbows flexed, and the arms adducted. The feet are arched and inverted, with the toes pointed and the legs extended and adducted in tonic contraction. More

rarely the muscles of the face and jaw are affected, and sometimes those of the pharynx, larynx, and tongue. In rare cases the trunk muscles may also be involved.

The spasm varies in intensity and duration. It may last for a few minutes or persist for several days. It may be overcome by force or resist all attempts at relaxation. Its onset is sudden, but its resolution is gradual. It may at times be attended by pain in the muscles.

Several characteristic phenomena are present during an attack:—

(*a*) The spasm may be induced by pressure on a nerve trunk or plexus.

(*b*) Percussion of a motor nerve may cause tetanic contraction of a muscle.

(*c*) Percussion or electrical stimulation of a sensory nerve may induce a spasm.

(*d*) The muscular reaction to galvanism is of a tetanic character, and the anodal closing contraction is greater than the cathodal. No objective sensory disturbance is present. The deep reflexes are brisk and the plantar response is of the flexor type.

During the course of the malady the temperature is raised and the pulse accelerated, and œdema, redness, and sweating of the hands and feet may be observed.

Prognosis. Recovery usually takes place when the exciting cause has been removed. Death from tetany is rare, but may occur as a result of malnutrition from gastric and intestinal disorder.

Treatment. A brisk purge should be administered in all cases, and attention paid to the diet. In cases in which gastric dilatation is present, lavage of the stomach should be carried out. The administration of intestinal antiseptics, such as salol, may be of use. During the attack, complete rest, with bromides and chloral internally, are of value. In severe cases it may be necessary to give inhalations of chloroform or injections of morphia. In those cases directly due to removal of the thyroid gland, extract of thyroid may be administered with advantage.

Chapter IV

OCCUPATION NEUROSES

This condition is a local disturbance of innervation of the muscles required in the performance of special acts or movements necessary for the carrying out of certain occupations.

Although the action of the muscles is impaired mainly in so far as the special movements are concerned, yet in the severe types of the disease other actions of the affected muscles may be interfered with.

Paralytic conditions associated with the carrying out of certain trades, and known as 'craft palsies,' have also been described. These differ from the occupation neuroses by the presence of muscular atrophy and sensory disturbances suggestive of a localised peripheral neuritis.

Etiology. The occupation neuroses occur chiefly in those whose profession, or trade, requires the constant use and application of certain muscles of the hand and arm, more rarely of the lower limb. The finer the movement and the more persistently it is applied over long periods of time, the more readily will the neuroses develop in those predisposed towards them.

They are found in persons in a debilitated state of health, and principally in those of a nervous disposition, who are often at the same time suffering from neurasthenia.

They are more often observed in males of adult age than in females, and they are not unknown in children. The occupations in which they most commonly occur are writing, sewing, painting, composing, piano- and violin-playing, and typewriting.

Symptoms. The symptoms may be described according as they are of a neuralgic or spasmodic and cramp-like character. In mild cases a sense of discomfort, of aching, or of a readiness to fatigue, rather than actual pain may be the only symptom. In the more acute types definite pain, rarely however accompanied by tenderness along the nerves, may be present. A prolonged and persistent effort over many hours at writing may lead to a sense of fatigue in the hand and arm, which should not be regarded as a definite neurosis, for a

short rest will relieve the symptom. Should, however, the strain thus induced be continued daily over many months or years in predisposed persons, a definite type of occupation cramp or neuralgia will eventually be developed.

A survey of the symptoms found in the several forms of occupation cramp reveals the fact that the muscles primarily affected are those subjected to severe and prolonged strain, as in holding an object such as a pen, or in forcible contraction, as occurs in the piano-player or the violinist. Prolonged and often-repeated muscular effort of any nature is a frequent cause of the neurosis.

The *spasmodic* or cramp-like symptoms may be described in conjunction with the muscular effort required to carry out the action.

(*a*) Occupations requiring the use of the prehensile muscles of the hand, such as writing, sewing, painting, composing.

The commonest neurosis of this class is writer's cramp, which may be taken as a type of the affection. The spasm affects the flexors of the thumb and index finger. The pen is forcibly gripped for a time, but eventually slips from the hand; or the spasm may lead to an irregular and misdirected use of the pen, the point of which catches on the paper. The spasm may gradually overflow from the hand, so as to affect the extensors, supinators, and pronators of the wrist. The writing is jerky and irregular, and the paper shows splutters of ink where the point of the pen has caught. It is accompanied by pain in the ball of the thumb, in the index finger, and about the wrist.

In tailors and seamstresses the thumb and index finger are those mainly affected. In dairymaids the cramp is in the flexors and extensors of the wrist and fingers.

(*b*) Occupations requiring forcible, but not necessarily continuous muscular efforts, as in piano- and violin-playing, typewriting, and flute-playing.

In pianists the cramp takes the form of a spasmodic raising of the fingers from off the keys, or of their forcible retention upon them. The strain is mainly upon the extensor muscles of the forearm, and the pain is occasionally severe, and affects the upper arm and shoulder as well as the forearm and wrist.

In violinists and 'cellists the strain is chiefly upon the

left or string hand, the fingers of which press upon the strings, and in 'cellists particularly upon the abductor and opponens pollicis (Poore).[1] The cramp is usually accompanied by pain in the arm and shoulder. The right or bow hand may also be the seat of cramp, owing to the strain upon the prehensors of the hand, but this is less common.

(*c*) Occupations requiring frequently repeated muscular efforts accompanied by strain. Examples of this are seen in blacksmiths, in whom the upper arm and shoulder muscles, especially the triceps and the deltoid, are implicated, and hammermen, where a difficulty occurs in directing the arm which holds the hammer. In a case of treadler's cramp described by Rivers, the glutei and the hamstring muscles of the right leg were affected. Tiptoe dancers are notoriously prone to spasm of the muscles of the calves of the legs.

Many other occupations from time to time occasionally present a case of occupation neurosis. We have seen spasm of the tongue in a cornet-player; spasm of the orbicularis palpebrarum has been recorded in watchmakers; and miners' nystagmus is a well-recognised condition in those who work in a recumbent posture with the eyes turned upwards to one side.

The *general symptoms* of the occupation neuroses are those of neurasthenia, or of any associated disorder which may be present. Some degree of tremor of the hands is an invariable accompaniment, and is usually more marked in the affected limb. The deep reflexes are brisk.

Differential diagnosis. Most cases present no difficulty, but symptoms resembling those of an occupation neurosis may arise, either in the early stages of organic nervous disease or from a local condition. It is therefore necessary to exclude all such conditions. The following are the disorders in which difficulty in diagnosis may arise:—

Paralysis agitans. Tremor, weakness, and spasticity of the thumb and finger muscles give rise to difficulty and cramp in holding the pen.

Brachial neuralgia resembles in many respects the neuralgic form of the occupation neuroses. Although often associated with symptoms of neurasthenia, this variety of the

[1] Poore, *Allbutt's System of Medicine*, vol. viii.

malady is not accompanied by the spasmodic contraction of the muscles found in the genuine cramp, but presents the neuralgic symptoms in a marked degree.

Partial paralyses of the peripheral nerves of the arm have been known, also, to give rise to symptoms having a resemblance to the occupation palsies.

Articular affections such as rheumatoid arthritis, tenosynovitis of the fingers or wrist, are other conditions which may interfere with movements of the hand.

There is an hysterical or psychical occupation neurosis, in which a fear of inducing the disorder may interfere with writing, perhaps owing to the patient having seen it in others, or of having heard or read of its effects. The apprehension may be so vivid as to become almost a fixed idea, and produce a state of mind in which writing becomes impossible. This is a rare condition.

Prognosis. The spasmodic forms of occupation neuroses are prolonged and intractable conditions. By aid of complete rest considerable alleviation may be obtained, but a return to work, even where the malady seems to have been cured, is frequently followed by a relapse. In the neuralgic cases, especially those complicated with neurasthenia, recovery is rapid, but relapses are liable to occur.

Treatment. The first essential lies in desisting entirely from the occupation which has given rise to the neurosis. In some cases, more especially in writers, the condition arises from faulty methods. Such should, on resuming work, be corrected. During the period immediately following the cessation of work, local treatment to the affected limb or limbs, especially massage of the muscles and carefully selected Swedish exercises, should be prescribed. Electrical treatment, particularly galvanism, may be adopted with advantage. In the obviously neurasthenic cases a complete or modified rest cure has been found of great benefit. In cases complicated with brachial neuralgia, the arm requires to be rested in a sling, and the other methods of treatment for this condition adopted.

Drugs may be prescribed as required. Tonics are of use in the later stages, but sedatives may be needed in the earlier.

Treatment should be continued for a prolonged period.

Even after a year's rest a return to work may induce a relapse of the symptoms. Some cases never entirely recover: in which event it is necessary for the patient to take up another occupation.

Craft Palsies

Atrophy of the small muscles of the hand occurs in certain occupations requiring long-continued over-exertion of certain muscles (Oppenheim). This atrophy is partly due to over-exertion and partly to pressure upon the nerves.

Craft palsies occur most commonly in platers, filers, locksmiths, rowers, glass-workers, and cigar-rollers. Similar affections have been found in the lower limbs in potato-pickers, seed-sowers, and others.

In addition to muscular atrophy, paræsthesia, numbness, and pains are present, indicating pressure upon the peripheral nerves. Alcoholism appears to favour their occurrence, and other toxic conditions—such as lead-poisoning, influenza, and general states of debility may also act as predisposing factors.

The prognosis is on the whole favourable, and the treatment consists in complete rest and galvano-faradic stimulation of the atrophied muscles, with tonics internally.

TETANOID CHOREA

This disease has derived its name from the appearances which were observed by Gowers in two cases described in 1888.[1]

The disease is probably due to morbid changes induced by inherited syphilis, as Homén suggested in 1890. The changes found after death have been symmetrical softening of both lenticular nuclei, sometimes with thickening of the membranes and of the skull. These changes in the nervous system are associated with cirrhosis of the liver.

The leading features are: its onset during apparent health, the progressive character of the symptoms, its occurrence in families, and its association with cirrhosis of the liver. It affects both males and females, and the symptoms appear in late childhood or early adult life.

[1] Gowers, *Diseases of the Nervous System*, vol. ii, 1888.

The symptoms consist of slow, continued tetanoid spasms of the limbs, sometimes involving the masseters and facial muscles. Difficulty in protruding the tongue, or in closing the jaws, owing to pain has often been observed, and gives rise to dysarthria and difficulty in swallowing. These movements tend to become less marked or less constant, while rigidity develops along with progressive mental impairment and general emaciation.

Death may occur within a few months, or life may be prolonged over several years.

Part XIV

MIGRAINE AND PERIODIC HEADACHE

Migraine and periodic headache are described together, as they appear to be essentially of the same nature, although the phenomena presented in any given case may vary from the typical migraine seizures with their peculiar visual symptoms and gastric disturbances, to a localised headache accompanied or succeeded by nausea.

The characteristic features are the periodicity of the seizures, their persistence throughout the whole of early adult life, their tendency to disappear or lessen towards the end of sexual life, their essentially hereditary character, and the absence of any obvious or well ascertained cause for their occurrence.

Etiology. Periodic headaches may commence at any age, but more commonly during childhood and puberty. Cases on the other hand are observed in which the attacks of headache have not appeared until adult life. Although it is usual for these headaches to diminish both in severity and frequency at or after the climacteric, it is found that some increase of the symptoms may date from this epoch in a number of cases.

Women are probably more liable to periodic headache than men.

Heredity plays an important part in the causation of migraine. It is a disease which runs in families. Many members of the same family are subject to one or other form of paroxysmal headache. The type of headache is not always the same. A mother, who suffers from typical migraine, may have one or more children who are subject to periodic headache without the visual symptoms; or paroxysmal headache may occur as the type of neurosis in one member of a family,

whose other members suffer from functional disorders of which, hysteria, hay-fever and 'nervousness' may be manifestations.

There exists, moreover, a relation between paroxysmal headache and epilepsy. Cases are constantly met with in which periodic headache in one of the parents may be associated with epilepsy in the offspring. It has also been observed that the periodic headaches of early life may be replaced by epileptic fits in the later years, and the coexistence of periodic headache and epilepsy in the same person is a feature occasionally observed.

No constant exciting cause has been determined. Of those which favour the onset, overwork, worry, anxiety, fatigue and mental excitement are the most common. Digestive disturbances, anæmia, and debility are also frequent. The menstrual periods are especially liable to determine headaches in those subject to them. Errors of refraction are a not uncommon association. Stress has been laid upon the influence of toxic conditions—more especially of gout and uric acid.

Symptoms. The symptoms of this disease consist of attacks of headache, which may or may not be preceded by prodromal symptoms, and may or may not be associated with visual phenomena. They are usually unilateral, and are accompanied or succeeded by nausea, retching, or vomiting.

Although some persons have no warning, others are usually made aware of their approach by premonitory symptoms: of which a feeling of coldness in the extremities, excessive fatigue, a 'heady' sensation, restlessness, and even insomnia the night before, may be mentioned.

The headache may come on during the early morning, waking the patient from sleep; or during the day, when its onset is preceded by a definite aura. In some cases the aura consists of loss or perversion of the visual functions. In other cases the aura is in the form of numbness, paræsthesia, or perversions of common sensibility in the limbs, face, or tongue. If on the right side, a paraphasic affection of speech may accompany it. With the cessation of the aura the headache comes on.

The *headache* varies much in character. Sometimes it is of a boring, penetrating nature; at other times it is described

as pulsating or throbbing, or it is said that the head feels as if gripped in a vice.

The position of the headache also varies. It is usually frontal, or referred to the orbit, forehead, or fronto-temporal region. In other cases it is referred to the root of the nose, or supra-orbital margin. It may remain limited to these regions; or, as already described, it may radiate over the vertex to the occipital pole. In rarer cases it commences at the back of the head, and tends to diffuse itself towards the vertex and temples. Its presence gives rise to great intolerance of light and sound. The victims of these attacks lie down in a darkened room and shut themselves off from all noise or communication with others. There is also a profound aversion to food during the persistence of the attack.

It has been urged as a point of differential diagnosis between epilepsy and migraine, that, in the latter condition, *mental symptoms* following the paroxysm do not occur. In many cases this is so; but in others—owing mainly to the intensity of the pain—hysterical manifestations, stupor, and delirium have been described. On the other hand, notwithstanding the frequency of the attacks of migrainous headache or the persistence of the malady over many years, mental impairment of a permanent character is never observed.

Visual symptoms are found in about half the cases of periodic headache, and when present always form the aura, or warning of the impending seizure. They vary greatly in different cases, but those most commonly described, fall into one or other of the following types:—

(*a*) Central scotoma, or defect in or abolition of the central vision. This spot enlarges from the centre to the periphery, and as it expands laterally clears up centrally. In the central dim area spectral phenomena may appear—such as flickering lights, or a central bright spot, or the edge of the spot takes on a zigzag shape, or 'fortification spectrum.'

(*b*) Hemianopsia, or blindness of the corresponding half-fields in both eyes. This usually begins as a small area, and expands until the half-field is completely obliterated.

(*c*) Indefinite symptoms—such as coloured lights, objects in constant vibration comparable to what is seen in cinematographic pictures, sparks, or flashes of light.

Sensory symptoms when present are referred to the upper

limbs, the face, tongue, and lips, and only rarely to the legs. They are generally unilateral occurring on the hemianopic side. They consist of tingling and paræsthesia which pass up the limb into the face and mouth; or the arm only, or the mouth only, may be affected. Following the subjective sensations come numbness and sometimes impairment or loss of sensation. They may be succeeded by slight and temporary loss of power in the arm.

If *aphasia* is present it is usually of the nature of paraphasia, or inability to find the word, or to use the wrong word; only rarely has complete motor aphasia been observed.

Types of migraine. Attacks of migraine vary in different persons, but in all a somewhat similar sequence occurs, which may be best described by referring to some illustrative cases.

(*a*) The attack may be preceded by a feeling of faintness and a craving for food. About an hour later the attack itself commences with a feeling of numbness and deadness in the tip of the tongue and in the fingers of the right hand, sometimes also of the left. This is followed by a feeling of coldness and general pallor. In from five to ten minutes the numbness spreads to the lips, mouth, and face, and speech becomes paraphasic. About this time vision becomes affected, with loss first of the central field, rapidly followed by right-sided hemianopsia, occasionally, but rarely, accompanied by zigzag lights in the right temporal field. About twenty minutes from the onset, the subjective visual and sensory symptoms begin to fade and are replaced by a dull, persistent, boring pain strictly limited above the left eyebrow; perspiration, nausea, and vomiting. The headache increases in intensity, and the general sensation of cold assumes a more intense and throbbing character; the pallor then passes off, and the coldness gives place to a sensation of warmth. The headache gradually lessens and disappears in from twelve to twenty-four hours, leaving a feeling of great prostration behind it.

(*b*) The first sign of the impending attack is a sudden temporary defect of central vision, in which the figures, for example, of a watch are obliterated or rendered indistinct. This is followed after a few minutes by the appearance of a flickering patch of pure white light, as if many thin intersecting lines were dancing irregularly to and fro. This sensation is always one-sided, and the headache which follows

is usually, if not invariably, on the opposite side to that on which the light appears. After some fifteen or twenty minutes the flickering light disappears, and is followed by a localised headache—always frontal and unilateral. This usually lasts for some hours, and is accompanied by flatulence, eructations, nausea, and sometimes by fits of violent retching and vomiting. After the headache has passed away, there is commonly a free flow of pale or colourless urine.

(*c*) The attack begins with a warning sensation of flushing of the face and a feeling of coldness in the extremities. Severe pain of a throbbing or pulsating character then starts in the region of the temples, sometimes on the right, sometimes on the left side. The pain always remains for a time on the side upon which it starts. If the headache is very severe it may spread, later on, over the head. Nausea, severe retching or vomiting, persist throughout the attack.

During the attack the patient passes into a hysterical state from the intensity of the pain. After this is over she is prostrated and exhausted for one or two days.

(*d*) A definite pain in the head begins to come on in the early hours of the morning, but finally settles down in the region of the right eye, forehead, and temple. It does not radiate to the back of the head, but occasionally passes through to the right side of the neck or shoulder. After the headache has persisted for about six hours, retching and vomiting commence and continue along with the headache, which rarely ceases until the evening of the following day. After the attack is over there is sometimes difficulty in raising the right upper lid.

(*e*) The attack begins with pain usually in the left eye, which extends into and down the nose. It then passes along the affected side of the head to the occipital region. During the persistence of the headache, retching and vomiting come on and last for about twenty-four hours.

Course and prognosis. The attacks of headache vary much in frequency and duration. They may be as frequent as two or three per week, or as infrequent as two or three yearly. In severe cases they may be counted by the week, fortnight, or month. They may come on at any time, but usually in the early morning or during the forenoon. The experience of patients varies as regards the duration of the seizure. The

common duration of an average seizure is from six to eight or ten hours; in many cases the headache persists for twenty-four hours or more, and some attacks of headache do not entirely abate for two or three days or longer.

The tendency is towards an amelioration of the severity, and a diminution of the frequency of the attacks as age advances. This is not invariably the case. Some of the most severe and prolonged attacks of paroxysmal headache have been seen in women, who have passed the climacteric. A considerable number of female patients find an increase in the severity and frequency of the headaches some years before the climacteric is reached.

Treatment. All likely excitants of the attack, of which the more important are fatigue, mental and bodily stress and strain, overwork, excitement, and late hours, should be avoided. Indiscretions in diet, especially towards the end of a spell of freedom from attacks, should be avoided. Errors of refraction should be searched for in all cases in young people, and, if necessary, corrected. The debilitating influences of anæmia, influenza, and other toxic conditions should be treated along general lines. It seems, however, impossible to prevent an occasional attack, even on the part of those persons who have carefully and intelligently studied their malady.

Acting on the theory that in many cases the attacks are attributable to an excess of uric acid, xanthin, or hypoxanthin, mainly ingested with certain articles of food, the purin free diet, suggested by Haig, has been recommended and occasionally found beneficial in reducing the number and severity of the attacks, and in some instances even arresting them altogether. The cases successfully treated in this way have been associated with an increase of the arterial tension before the attack.

On account of the persistent character of the malady and tendency for the attacks of headache to recur frequently, many drugs have been recommended, and tried—some with a view to diminish or arrest the attacks, and others to abort, modify, or subdue the seizures as they arise.

Amongst the former, the bromides, administered as described under epilepsy, alone or in combination with phenacetin and caffeine, have been of service in many cases. The tinctures of gelsemium or of belladonna have been found

useful auxiliaries to the bromides. The prolonged administration of the valerianates of zinc and iron, with or without the addition of small doses of cannabis indica, has also been found of advantage in cases complicated with anæmia, or in which the bromides are badly taken or inconvenient. Nitro-glycerine has been strongly recommended in cases characterised by high arterial tension.

As remedies for the temporary alleviation of the attacks, all the synthetical analgesics may be tried—antipyrin, phenacetin, exalgin, phenalgin, aspirin, &c. One or other of them, alone or in combination, prescribed as a draught during the premonitory stage and repeated in an hour or two, will probably be useful in the majority of cases, although now and again a case will occur in which they prove to be of little service.

A useful remedy is the administration of phenacetin (5 grs.) and caffein (2 grs.) as soon as the attack commences, followed by liq. strychnine (5 ms.) when the headache begins to lessen.

Part XV

HYSTERIA

Hysteria is a psychical disorder, arising from a functional disturbance of the cerebral cortex. Its fundamental characteristic is a dissociation or severance of some mental processes from the main personal consciousness (Janet).[1] This dissociation is the basis of both the temporary and the persistent phenomena of the disease, and shows the common origin of such diverse conditions as the hysterical fit, dual personality, anæsthesia, and paralysis. In the hysterical paroxysm, the patient passes through a phase of movements and actions of which there is little or no subsequent recollection; and in the paralyses, there is loss of consciousness of the existence of the affected limb or side of the body.

Etiology

Hysteria is commonest during puberty and early adolescence, but is not unknown in childhood. It may disappear during adult life, but is liable to recur in persons of neuropathic disposition. The female sex is more often affected than the male, especially in later life. It would seem to have a special proclivity to affect the Latin, Slavonic, and Jewish races.

It occurs in all social grades, being as frequently found in hospital as in private practice.

Heredity plays an important part in the production of the disease. The neuropathic disposition is shown by the occurrence of hysteria, epilepsy, insanity, the tics, alcoholism, and 'nervousness' in other members of the family.

[1] Janet, *The Major Symptoms of Hysteria*, 1907.

Hysteria may be excited by all forms of emotional disturbance – shock, fright, worry, or grief; by prolonged ill-health – anæmia, pelvic disorders, and other physical factors of a like kind. Traumatism is a common cause of hysteria, which is attributable rather to the psychical conditions attendant upon an accident than to any bodily injury.

Symptomatology

Hysteria is characterised by a variety of symptoms, differing materially in type, persistence, and distribution, but all attributable to the same fundamental psychical state.

As regards type, some of the symptoms – such as paralysis – denote a loss of function; while others – such as spasms, agitations, and tremors – indicate an excess of function. When considered from the standpoint of persistence, some are transitory – such as the convulsions and the automatisms – and to them the term 'accidents' of hysteria has been applied. Others are more enduring, and on account of their persistence have been named the 'stigmata' of hysteria; these are the anæsthesias, paralyses, and contractures.

Hysterical temperament. The principal feature of the hysterical temperament is an undue nervousness and susceptibility to both physical and psychical impressions. On this account mainly, normal or everyday phenomena are not only misinterpreted, but are sometimes so exaggerated as to assume an altogether disproportionate significance.

Minor illustrations of this state are seen in the starts or jumps which these patients exhibit when a door is slammed, or when any sudden action or movement occurs; or, in the psychical sphere, in the taking to heart a word or phrase spoken in jest or uttered without any unkind intent. Attacks of so-called 'hysterics' – such as screaming – which may accompany an unusual sight, such as a serious accident, may be regarded as a more exaggerated form of the same condition.

Another feature of the hysterical temperament is susceptibility to hypnotism and auto-suggestion.

Hysterical persons also may possess many of the temperamental disabilities common to neuropathic subjects – such as abnormalities of emotion and of will power. Feebleness of will,

indecision, obstinacy, and doubt are often common accompaniments of the hysterical state. Although the intelligence is good and the memory normal, judgment is often weak and easily influenced by temporary emotions.

Mental state. The basis of the mental state in hysteria consists of a dissociation, or disparity of consciousness, whereby certain phases of consciousness may be lost or perverted. The most characteristic instance of dissociation is seen in the condition known as double consciousness, or dual personality. In this manifestation of hysteria two entirely separate states of activity are found—one, however, being the dominant state. In one of these phases the patient is her natural self in thought, word, and deed; but in the other she exhibits a different and sometimes antagonistic personality. There is no conscious recollection of what has occurred in one state when in the alternate condition, although the memory of the somnambulistic phase may be revived by hypnotic suggestion.

Lesser degrees of dissociation are illustrated in those amnesic phases in which portions of the previous existence of the patient may be entirely blotted out and forgotten.

In cases of disparity or inequality of consciousness, the phenomena of dissociation are less extensive or less complete. For example, there may be a failure of memory confined to certain events or certain persons; or there may exist a want of conscious appreciation of a portion of the body. Thus, one of our patients, suffering from hysterical paralysis of the right arm had no conscious perception of her arm, and stated that she felt as if the arm had been 'lopped off' at the shoulder joint.

Hysterical Paroxysms

These are of several kinds and vary both in intensity, character, and frequency. They are: –

1. Minor Hysteria ('Hysterics').
2. Major Hysteria.
3. 'Hystero-epilepsy.'
4. Automatic phenomena and dual personality.
5. Hysterical stupor.

1. The *minor hysterical attack* may follow an emotional

shock, or occur without any obvious cause. It commences with a sensation of palpitation, or of a rising from the stomach, which is followed by a sensation of a lump in the throat (globus hystericus). Repeated acts of swallowing are made with the object of removing the sensation of the lump. A feeling of suffocation ensues, the patient tears at the neck-band of her dress, or throws herself into a chair, with starting eyes, in a state of great agitation and distress. She pants for breath and calls for help. She may tear her hair, or wring her hands, or she may swoon or break out into uncontrolled laughter or weeping. Recovery gradually ensues with eructations of wind, especially when much globus has been present. A large quantity of pale limpid urine is usually passed after the attack, which is followed by considerable exhaustion. Any one of the main constituents of the attack may form the sole feature of an individual seizure.

2. *The major hysterical attack.* The seizure may be preceded by an aura of pain in the region of the ovaries or stomach; or by the globus hystericus, palpitation, general excitement, dizziness, or sensations – usually bilateral – referred to the limbs. The patient may utter a cry, and then more or less suddenly fall or sink down apparently unconscious. The fall is never quite sudden, as in epilepsy, nor does she injure herself. What usually happens is that the patient throws herself on to a chair or couch, and then slips gradually on to the floor. Then the seizure commences, and consists of convulsive movements – invariably bilateral – affecting the arms, legs, and head. The patient drums on the floor with her heels, throws her arms about, and tosses her head from side to side. There is a marked tendency towards the development of opisthotonos. The eyes are deviated upwards and inwards, in some cases the eyelids are firmly closed; the corneal reflex may be abolished, but the pupillary light reflex is not lost. Tongue-biting does not occur, but the patient may bite her lips or the finger of an onlooker. The character of the convulsions varies from opisthotonic rigidity to wild, semi-purposive, convulsive-like movements. There is no deviation of the head and eyes to one side, as in epilepsy, and the rapid alternation between the opisthotonic and convulsive phases is quite characteristic. A further common feature is the increase of the movements and the struggling of the

patient, when an effort is made to restrain her. If a pin be placed in the direction in which the movements are taking place, a diversion of the action not uncommonly occurs.

The duration of the seizure varies; it is rarely less than ten minutes, but may be prolonged from a half to three or more hours.

Urine is rarely passed during the attack.

Recovery is abrupt, the patient suddenly sits up, rubs her eyes, and asks where she is. There is no post-paroxysmal mental confusion or automatism. Memory of the events of the fit may be present, but more commonly there is only a vague idea, or no recollection of it at all.

3. '*Hystero-epileptic*' *seizures.* These seizures are rare in this country, but are not infrequent in France, and it is to the French writers that we owe the classical descriptions of 'la grande hystérie.' In many of the attacks loss of consciousness is complete, but throughout the fit it may be obvious from the motor phenomena that the patient is acting a subconscious idea. The attack may commence with opisthotonos, the body resting on the feet and occipital region of the skull, the back highly arched, and the belly prominent. This attidude may suddenly give place to one of adoration, in which the patient kneels with the hands clasped, and the head and eyes thrown upwards; or there may be the assumption of a crucifixion position, in which she lies on her back with the arms abducted and outstretched at right angles to the body. The attack is usually followed by delirium, hallucinations, cramps, and contractions of the limb. The whole attack may last from a quarter up to half an hour, or longer.

4. *Automatic phenomena.* These are phases in which the patient appears to be in a dream, and may carry out acts and movements, which are almost impossible under normal conditions. These attacks are usually started by a severe emotional shock. They are sometimes of short duration, a few hours, or may be prolonged for days or weeks. They are preceded by a prodromal stage of irritability and headache, and may be succeeded by an after-stage of exhaustion, confusion, and headache. During the attacks patients may travel long distances, and awake to find themselves in an unknown

locality. The condition more closely resembles the somnambulistic state than any other; for, during the phase, complicated acts and movements are effected, semi-purposive in intent, but in reality automatic.

A condition very similar to the above is found in epilepsy, as well as in alcoholism and states of mental deficiency. According to Janet, the hysterical character of the attacks is established when it can be artificially induced and a recollection of it revived by hypnotic suggestion.

5. *Trance or stupor.* This condition may persist for many days, the patient lying quiet and often presenting a death-like appearance. The skin is wax-like, the eyelids semi-closed, the respirations superficial, the temperature slightly subnormal, and the heart's action feeble. There is, however, often a suggestive twitching or tremulousness about the eyelids, and the pupils respond to light. In the slighter varieties of hysterical sleep the patient may move her limbs, or change her position, or even mutter. In others, the appearance is more suggestive of the cataleptic state, in which the limbs are in a condition of plastic rigidity, and remain in any position in which they are placed. On recovery, there is loss of memory of the events of the sleep period.

These sleep periods are caused by psychical circumstances, and may be either of sudden or gradual onset; but some can be induced by pressure upon the hypnogenic points such as the breasts.

Hypnotism has come to be used as an important agent in the differential diagnosis of the automatic states, because in hysterical cases automatism, trance, and stupor can be induced artificially by hypnotism; whereas in epilepsy, psychasthenia, and insanity, hypnotism fails to reproduce the symptoms.

The Cranial Nerves

Smell and taste. These may be diminished, or lost, on one side, in association with hysterical hemianæsthesia, or abolished on both sides.

Vision. The following are the visual symptoms of hysteria:

(*a*) Concentric narrowing, or contraction of the visual field. This may be unilateral or bilateral. When the former, it is commonly upon the same side as the cutaneous anæsthesia,

the field being reduced to a circular area as small as ten, or even five, degrees; if bilateral, it is more marked on one side than the other. The amblyopic area either embraces all forms of vision, or is limited to colour perception. It has been stated that in hysteria the area for red remains large, and is the last to be lost; while in colour amblyopia from organic disease, the red and the green fields are impaired early.

(*b*) Amaurosis, or hysterical blindness, is more often unilateral and upon the same side as the hemianæsthesia. It is unaccompanied by any change in the optic nerves. It is usually of traumatic origin, of sudden onset and disappearance, and requires great care in the diagnosis.

Hemianopsia and central scotoma are so rare in hysteria (if, indeed, they ever occur), that the existence of these symptoms usually points to organic disease.

Photophobia is a not uncommon hysterical symptom.

Hearing. Hysterical deafness usually incomplete in character is not infrequent, and, if unilateral, is found upon the side of the motor or sensory paralysis. Being a cortical condition, it responds to the tests for nerve and labyrinthine deafness—i.e., there is a positive Rinné reaction, and the perosseous and aerial conduction are synchronously impaired. It is sometimes combined with mutism. There may be an associated anæsthesia of the external auditory meatus. In organic disease of the labyrinth there is tinnitus aurium and frequently vertigo.

Hysterical hyperaceusis is an affection in which noises and sounds are so keenly perceived as to cause mental pain.

Oculo-motor nerves. Occasionally there is an apparent paralysis of the conjugate movements of the globes. In organic paralysis of conjugate movement, a patient, who is unable voluntarily to move the eyes to one or other side, can only follow the observer's finger by turning the head in the direction required. In hysteria, on the other hand, she is able to keep her eyes fixed upon an object, when the head is passively moved in the opposite direction.

An appearance of false ptosis may be produced by spasm of the orbicularis palpebrarum. Paralysis of single ocular muscles probably never occurs in hysteria. Nystagmus is never an hysterical symptom.

Fifth nerve. Anæsthesia over the face and head may be

part of a general or unilateral affection of sensation. If it exists apart from general anæsthesia, it does not coincide with the sensory distribution of the nerve on the skin, but reaches to the margin of the jaw, taking in that portion of the skin of the face, which is supplied by the cervical nerves.

Seventh and twelfth nerves. The face and tongue are

Fig. 173.—Illustrates hysterical spasm of the right side of the face.

rarely paralysed. Both may be affected by spasm. Spasm of the tongue on one side, when present, is a characteristic hysterical symptom.

Hysterical affections of speech. These may occur alone, or in association with other hysterical stigmata. Hysterical mutism is rare, and is closely related to hysterical aphonia. Both may be induced or disappear suddenly, under the influence of strong emotional excitement. In mutism, the patient is unable to speak at all, even in a whisper; in aphonia, whispering is preserved. In both the disability is in the vocal mechanism, but is more advanced in mutism than

in aphonia (Wyllie). Stammering may also be found in hysteria. Sometimes it follows an attack of mutism or aphonia. It may be accompanied by spasmodic movements or contortions of the face, jaw, and eyelids.

Motor Phenomena

The motor symptoms of hysteria are :—

1. Paralyses and contractures.
2. Tremors.

1. Paralyses and contractures

Paralyses may be of two types; the one flaccid, and the other associated with muscular stiffness and contracture.

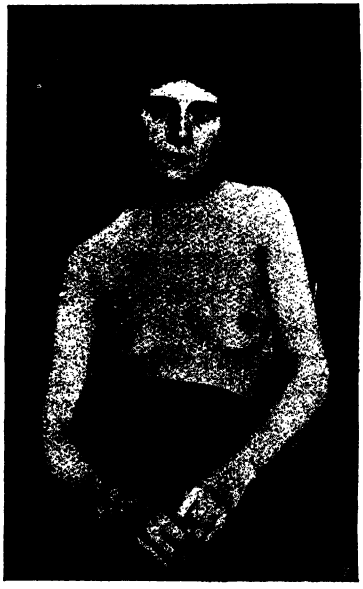


Fig. 174.—A case of hysterical flaccid paralysis affecting the right arm.

The flaccid paralysis is the less common variety. It is characterised by a limp or flaccid condition of the paralysed limb or limbs. As a rule it is of sudden onset, and almost invariably associated with complete loss of sensation. The muscles are not wasted, and the electrical reactions are normal. The limb may be passively moved in all directions, hypotonia is not present, and, if tested by the state of the tendon reflexes, the muscular tone may appear to be increased. Voluntary movement is completely abolished, and when the patient is asked to move the limb, no effort is made to do so. The paralysis is entirely one of voluntary motion. In the upper limb, although adduction to the side is impossible when the arm is held out horizontally from the shoulder, yet on coughing the latissimus dorsi is seen to contract, illustrating paralysis of voluntary movement, as far as that muscle is

concerned. If a patient with hysterical hemiplegia be asked to raise herself from the supine position, without using her arms, the paralysed leg may remain stationary on the bed, while the non-paralysed limb rises. This necessitates the active contraction of the muscles of the paralysed limb: as, normally, on attempting to sit up, the legs are actively extended at the hips, in order to provide a fulcrum for the flexor action of the abdominal muscles. If, in organic hemiplegia, a similar attempt is made to sit up,

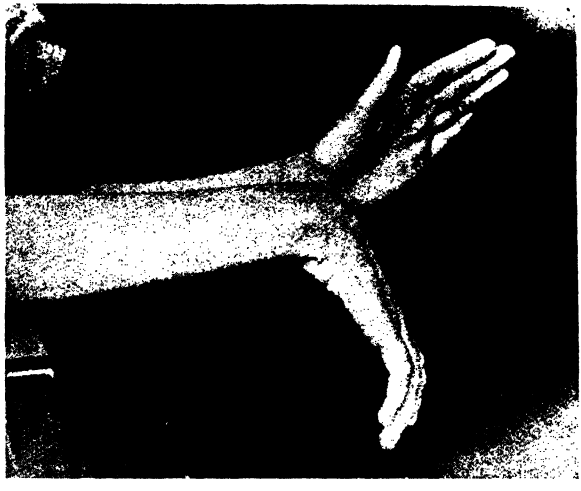


FIG. 175.—Hysterical drop wrist; the paralysis is of the flaccid type.

the paralysed leg tends to rise from the bed to a greater extent than that of the opposite side.

The second type of paralysis is associated with stiffness of the muscles. This is the more common form of hysterical palsy. The muscles are not wasted, and may be slightly hypertonic. The voluntary paralysis is less complete than in the flaccid form, slight movement being usually preserved. There is no true spasticity, although the limb may be held in a more or less stiff attitude from active muscular contraction. In this type of paralysis, when the patient is asked to perform a movement, all the outward evidence of great effort is demonstrated in associated movements of the face, trunk, and limbs.

If, for example, the patient is asked to strongly flex the

forearm, and the observer, supporting the forearm in one hand, places the other over the biceps and triceps muscles, he will feel, first, a well-marked contraction of the triceps, followed by a feeble contraction of the flexors of the forearm. If the arm is then semi-flexed at the elbow, and the patient asked to extend the arm, the converse will be observed: first a tightening of the biceps, and then a feeble contraction of the triceps. Similar phenomena may be seen at the knee or ankle joints. This prior contraction of the antagonists before the prime movers is a highly characteristic phenomenon of the functional palsies, and, as Beevor[1] has shown, does not occur in organic affections. In consequence of this, the voluntary movements present a jerky, irregular action. In some cases the synergic and associated actions of the paralysed limb are not affected; in this it simulates organic disease.

FIG. 176.—A case of hysterical paraplegia with contractures of the lower limbs.

In distribution the paralysis is hemiplegic, paraplegic, or monoplegic. In both the hemi- and paraplegic varieties the gait is usually of a characteristic type; and the paralysed limbs are dragged along the ground, the feet are shuffled—no attempt being made to raise them from off the floor; or the feet are placed in such a position that the dorsum rests upon the ground, while the sole points upwards and backwards.

The paralyses are often of long duration, lasting months or years, but are as a rule curable.

Paralysis is of the cortical type in that movements and not muscles are affected.

Contractures. Certain cases of hysterical palsy present well-marked contractures. These are due to active contractions of various muscles, and may simulate the positions observed in organic spastic contractures, or they may fix the

[1] Beevor, *Croonian Lectures*, 1903.

limb in more or less characteristic hysterical attitudes. Contractures may affect the whole limb, or portions only of a limb. In the upper extremity, the tendency is towards flexion of the elbow, wrist, and fingers, with adduction of the arm to the side of the body; in the lower extremity, towards extension at the hip and knee joints, with the foot in a position of talipes equino-varus. In such cases passive movement is impossible, but under an anæsthetic the contracture passes away, a

FIG. 177.—Illustrates a case of hysterical contracture of the hands and fingers.

FIG. 178.—A case of hysterical contracture of the right ankle and foot.

phenomenon which does not occur in organic contractures where structural alterations have taken place. Contractures are not confined to the limbs. One half of the tongue may be affected, and on protrusion is deviated to the same side. An appearance of ptosis may be produced by contracture of the orbicularis palpebrarum, and torticollis may result from contracture of the sterno-mastoid muscle.

A form of motor paralysis occurring in hysteria is known as *astasia abasia*. In this condition there is no loss of power of movement as long as the patient—usually a young person—

lies in bed, but as soon as an attempt is made to stand or walk, the legs give way and a fall occurs. Corresponding phenomena are seen in the hands and arms. Here the patient appears to have full power, but is quite unable to use a needle, play the piano, or hold and use a pen. It has been shown also that such persons may move their lips in the ordinary way, but are unable to blow or to whistle.

FIG. 179.—A case of hysterical contracture of the right foot.

2. Tremors

Three types of hysterical tremor are found: –

(*a*) *Vibratory tremor.* This is a fine vibratory tremor, like that seen in Graves's disease, and alcoholism. It is not under the control of the will, it may be present during rest, and is increased under observation and when the hands are outstretched.

A variety of the above is a coarse vibratory tremor. It may be present or absent during rest, and is increased on voluntary movement. It has this peculiarity—that if the tremor is arrested by holding the affected limb it starts, or is increased, in another.

(*b*) *Intention tremor.* A tremor of medium rhythm, absent during rest, but occurring on voluntary effort—similar to that seen in disseminated sclerosis.

(*c*) *Rhythmic tremor.* A slow tremor resembling that seen in paralysis agitans, persisting during rest, and only slightly, if at all, under voluntary control. A more or less persistent tremor, which is confined to the legs and has the features of the ankle clonus, may also be observed.

Sensory Phenomena

Subjective sensations are less common in hysteria than in organic nervous disease. When present they are chiefly in the form of pains. The common seats of spontaneous pain are the head, back, side of the chest, under the left breast, the epigastrium, and the inguinal regions. Not only may pain be located in these regions, but it may be the most prominent symptom in association with hysterical affections of the joints, viscera, breasts, and the vertebral column. In many cases the area to which the pain is referred is peculiarly hyperæsthetic; so that light pressure causes severe pain, while deep pressure, when gradually applied, does not give rise to discomfort. In the cases with paræsthesia or numbness, deadness, and coldness, neither the objective sensory loss nor the motor palsy is complete.

Tenderness on pressure is a characteristic feature in hysteria, and is to be distinguished from the hyperæsthesia to contact already mentioned. The tender areas are usually limited in extent; the commonest situations are the left inguinal, the left inframammary and epigastric regions, and various points along the spine, and sometimes on the head.

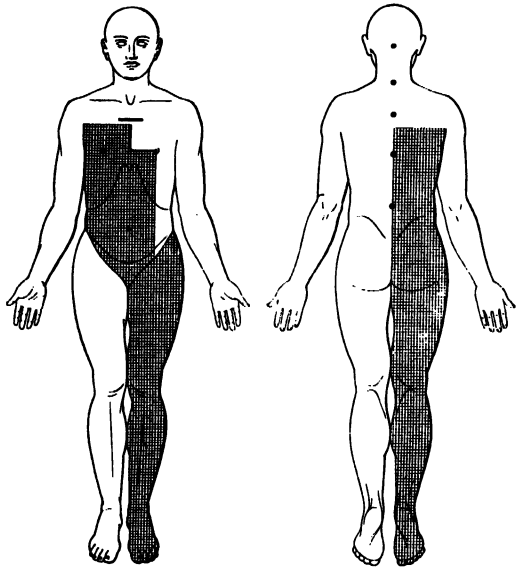
In some cases pressure exerted upon one of these spots produces an hysterical seizure, in consequence of which the term 'hystero-genetic zones' has been applied to them.

Objective sensory disturbance is by no means universal in hysteria, but is one of its most constant signs. In contrast to the objective sensory loss of organic disease, which is usually recognised by the patient in hysteria, the patient may be unaware of its existence; so much so, indeed, that the opinion has been expressed that the loss of sensation may have been suggested by the examination. This is probable in some cases, and under no circumstances is it warrantable to ask patients, if they 'feel so and so,' when each test is applied, but rather to encourage them to state what the sensation conveyed to their mind is, and to get them to locate it.

The onset of anæsthesia is often sudden, being induced by an emotional shock, or injury, or following upon an hysterical seizure. It is a mobile form of anæsthesia, shifting from one

to another part of the body. It may disappear under the influence of morphia, alcohol, and chloroform anæsthesia. It may persist for years, and sometimes disappears quite suddenly.

The following method, suggested by Janet, is useful in demonstrating hysterical anæsthesia. If the patient is told to say 'Yes,' when touched, and 'No,' when not touched, it will frequently happen that the answer 'No' is given as often



FIGS. 180 AND 181.—Illustrate the front and back views of a case of hysterical anæsthesia. Observe that the sensory loss is incomplete in distribution, and that in front it passes across the middle line.

from the anæsthetic side, as 'Yes' from the æsthetic side of the body.

Another test is the following, and may be applied when one limb is paralysed. In many hysterical cases the patient is obsessed with the idea of paralysis, and, although when tested in the ordinary way, is found to be anæsthetic over the paralysed arm, yet when investigated through the motor system, the hysterical nature of the condition may be fully demonstrated. She is first asked to place a finger of the paralysed hand upon any place where she is touched. She is

then touched upon some part of the æsthetic side, when it may happen that the paralysed arm is slightly moved, but, recognising the paralysis, she states that she cannot move the arm. The examiner at once tells her to use the sound arm instead, and proceeds immediately to touch her on some part of the anæsthetic area. In most cases the patient, without any hesitation, correctly localises the spot touched.

It should be pointed out that these apparent inconsistencies are not due to malingering, but result from the definite and well-recognised psychical condition, which is the basis of hysteria.

Loss of sensation may be described, first as regards its character, secondly its degree, and thirdly its distribution.

(*a*) *Character.* In severe cases, usually associated with a flaccid paralysis, all forms of sensation may be lost. In others, the sense of position and muscle pain sensibility may be preserved, with loss of the cutaneous sensibilities. Most commonly all forms of cutaneous sensation are affected, although in rare cases a dissociation of painful and thermal from tactile sensibility has been described.

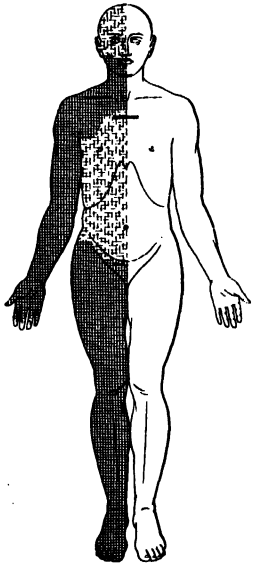


FIG. 182.—Chart showing a common type of hysterical hemi-anæsthesia. The dotted areas indicate where the sensory loss was incomplete.

(*b*) *Degree.* The loss may be complete. In the majority of cases, however, impairment only is found. All forms may be recognised correctly, but less distinctly than normal. In some cases the impairment of cutaneous pain sensibility is disproportionate to the degree of impairment of the other forms, and almost amounts to analgesia. In cases with incomplete loss, localisation is usually fairly correct.

(*c*) *Distribution.* Hysterical anæsthesia may be generalised over the whole body. It may extend up to a circular limit round the neck. It may be unilateral, involving mucous membranes and the special senses on the same side. It may

be limited to an upper or lower limb, or to both upper and both lower extremities. When limited to the upper limb it may involve the fingers only, or extend as far as the wrist, elbow, or shoulder, or to the middle line of the body. The upper limits are definite and usually circular in the transverse plane of the limb or body. In some cases a complete loss may extend to a definite line round the wrist, and a less complete loss to a definite line at the elbow. These are the 'glove' anæsthesias. Of a like distribution are the anæsthesias of the lower limb ('shoe,' 'sock,' or 'stocking' anæsthesia).

It is obvious that these areas do not correspond either to nerves, or to segmental regions. They most resemble the anæsthesia of organic cerebral conditions; but are distinguished by their definite upper limit. If affecting all four limbs, they have some resemblance to the anæsthesias of peripheral neuritis and early stages of sub-acute combined degeneration of the cord, in which conditions the upper limit is ill defined and fades into normal sensation. Hysterical anæsthesia may also be patchy, or may affect opposite quadrants of the body, such as the arm and upper part of the body on one side, and the leg and lower part of the body on the opposite side. Or three-fourths of the trunk may be involved to a line drawn through the nipple. (Fig. 180.)

The Reflexes

The tendon reflexes are never abolished; as a rule they are increased. A true ankle clonus, as in organic disease, is never found. A spurious ankle clonus, which is really a form of rhythmic tremor, may be elicited, but when present it is not associated with spasticity. It may commence when the muscular tone is relaxed, and often ends suddenly.

The plantar reflexes are never extensor even after an hysterical seizure. In a large number of cases the reflexes are absent, usually in association with anæsthesia. In many cases they are present, and although sometimes diminished, are flexor in type.

The abdominal reflexes are present, but in cases with anæsthesia over the abdomen they may not be elicited.

The corneal reflex may be diminished, but if tested by blowing upon the eye of the anæsthetic side, both eyes blink.

The pharyngeal irritability is diminished or lost.

Retention of urine is a not infrequent symptom of hysteria, but incontinence of a persistent type is unknown. Similarly, although constipation is common, incontinence of fæces never occurs.

Local Manifestations of Hysteria simulating Organic Disease

Anorexia nervosa may be induced in some hysterical girls by a hypersensitiveness to criticism and ridicule as to their getting stout. In other cases it arises from no obvious cause. It consists of a persistent refusal of food, and a gradual loss of appetite, impaired digestion, occasionally associated with vomiting, and finally a state of profound inanition. The patient becomes a 'living skeleton,' with extreme reduction of bodily and mental power. Its onset is most common in girls during the period of puberty, and may continue for several years, or even throughout life.

Hysterical vomiting is more frequent than anorexia, and may eventually lead to a severe form of inanition, although it may persist for a long time without giving rise to marked impairment of nutrition. Its commencement is usually dependent upon an organic condition such as gastric ulcer, gastritis, or flatulent distension of the stomach. The vomiting occurs immediately after food, and is unassociated with effort, pain, or nausea. It eventually becomes a species of tic, in so far as it may persist after the original exciting cause has been removed; and if it is prevented or arrested, a state of mental anguish, or even a hysterical seizure is induced.

Hysterical flatulence is a very common phenomenon. It arises in two ways: either as a result of globus, or of a tic of swallowing ensuing upon the globus hystericus. By the repetition of this act the stomach is filled with saliva and air, and rapidly becomes distended. Eructations follow, producing in the patient a pleasurable sensation. The nature of this affection may be demonstrated by asking the patient, when the stomach has been emptied, to continue to eructate. She at once then commences to swallow air, and then to bring it up again. In other cases 'wind-sucking,' analogous to

what is seen in horses, may be observed. The air is sucked into the stomach, and the patient may rapidly distend both the stomach and the intestines. This may occasion a pronounced flatulent distension, amounting to a phantom tumour. In one such case voluntary distension of either the stomach or the intestines was possible, and the patient was able to pass the ingested air from the intestines back into the stomach. Perforation has been known to occur in such cases.

Dysphagia is due to spasm of the œsophagus, and is a rarer condition than the hysterical vomiting or flatulence.

Respiratory symptoms. Cough, barking, intermittent respiration, hiccough, yawning are all of the nature of hysterical tics. Polypnœa, or phenomenal frequency of the respirations, has been known to follow severe emotional shock.

'Hysterical spine.' A frequent manifestation of hysteria is the condition known as 'hysterical spine.' It is characterised by subjective pain, which may be referred to the whole length of the back, or to certain more localised portions of it. It is common over the lower sacral and coccygeal regions (coccygodynia), over the lower and mid-dorsal regions, and the cervical region. The pain may be of a more or less continuous aching character, which is generally relieved by rest upon the back. On examination of the spine no curvature is present, but in many cases, owing to general weakness, a postural lateral distortion of the spinal column may be observed. Angular curvature never arises from hysteria. An apparent loss of mobility may be seen. The inorganic (functional) character of this distortion may be demonstrated by asking the patient to stand upright with the feet together, and then to bend forward with both arms equally extended as if to touch the feet, when the postural curvature immediately disappears. Tests by aid of lateral rotation movements may also be applied.

On percussion of the spine, tenderness may be general or localised. It may be more pronounced on pressure over the muscles on one or other side of the vertebral spines. It is also characteristic that the tenderness is as acute on light as on deep pressure.

The two conditions which simulate hysterical spine are tuberculous caries and malignant disease of the vertebræ. The

former may be recognised by the local position of the pain and by the immobility of the spine, by the localised tenderness to deep pressure and on movement, by a local prominence of one or more vertebræ, and by evidence of pressure upon the nerve roots or spinal cord. An evening rise of temperature would be in favour of tuberculous disease. In doubtful cases an X-ray photograph ought to be taken.

More treacherous is the simulation of hysteria by malignant disease of the spine Such cases usually occur after forty years of age, and are often associated with some degree of cachexia and loss of weight without obvious cause. In the early stages pain may be the only symptom, and may be referred to parts of the body other than the spine. The history of previous operative interference for the removal of a growth in any part of the body —especially the breast, the orbit, and the pelvic organs—should warn the observer against the hasty diagnosis of functional disorder. Lumps or growths on any of the other bones — especially the ribs – visceral tumours, or enlarged glands, should in all cases be especially sought for.

FIG. 183.—Illustrates a case of hysterical contracture of the left hip, simulating disease of the hip joint.

Hysterical joints. The commonest form is seen in simulation of hip-joint disease, which may arise suddenly from some emotional cause or trauma. It is characterised by pain intensified by any form of movment, tenderness on palpation with protective contraction of the surrounding muscles. Although

apparent shortening of the limb occurs in these cases, from the position in which the limb is maintained, no real shortening is found on measurement. Tenderness may be associated with hyperæsthesia over wide areas around the joint. The X-ray examination reveals no evidence of organic joint affection, and under anæsthesia the joint is normally movable. In these cases there is an absence of constitutional symptoms, and other manifestations of hysteria are usually present.

A word of warning may be said about the diagnosis of 'hysterical knee,' as pain in the knee without organic affection of that joint, is frequently the first and only sign of hip-joint disease.

'Hysterical breast' is not common; pain is usually confined to one organ, and no sign of organic disease is apparent.

Cardio-vascular system. Palpitation, and increased action of the heart are common, and may be associated with globus as the aura of an hysterical seizure. Cardiac pain, sometimes associated with faintness, may simulate angina pectoris, but the absence of vascular degeneration and the exciting cause of the attack, are usually sufficient grounds on which to form a diagnosis.

Vaso-motor and trophic symptoms. The most prominent of the vasomotor phenomena is an abnormal or unusual pulsation of the arteries. This is especially observed in the abdominal aorta, and may persist without change for months or years.

A curious phenomenon, recognised for many years as a feature of hysteria, is that no bleeding occurs when the skin of the paralysed limbs is pricked or incised.

In hysteria the paralysed limbs are frequently blue and cold, or of a mottled appearance. A form of œdema which does not pit on pressure, and which may be most marked in the morning, has also been described.

Two entirely opposite phenomena in the secretion of urine are also observed in hysteria. One is the copious flow of pale limpid urine which follows an hysterical seizure, and the other the suppression of the urinary secretion, which has been known to persist for a number of consecutive days.

TABLE GIVING POINTS IN DIFFERENTIAL DIAGNOSIS BETWEEN DISSEMINATED SCLEROSIS, HYSTERIA, EXTRA-CEREBELLAR TUMOURS, COMPRESSION PARAPLEGIAS, SUBACUTE COMBINED SCLEROSIS, AND CEREBRO-SPINAL SYPHILIS

	DISSEMINATED SCLEROSIS.	HYSTERIA.	EXTRA-CEREBELLAR TUMOURS.	COMPRESSION PARAPLEGIAS.	SUBACUTE COMBINED SCLEROSIS.	CEREBRO-SPINAL SYPHILIS.
Age at onset	Early adult life	Variable	Adult life	At any age	From 45 to 65	Adult life, history of syphilis
Course of disease	Remitting and relapsing tends to progress	Remitting and relapsing	Slow and progressive	Slow and progressive May be sudden	Progressive	Remitting or progressive
Motor symptoms and gait	Paraplegic, monoplegic, and hemiplegic Temporary ocular palsies Gait frequently unsteady Spastic or ataxic	Paraplegic, monoplegic, and hemiplegic Gait not that of organic disease	Paralysis of limbs rare; and if present, late Ocular palsies rare Gait unsteady and of 'drunken' reeling type	Paralysis below level of compression	Ocular palsies rare Gait spastic and sometimes unsteady Paralysis bilateral (legs more than arms)	Paraplegia, hemiplegia, monoplegia
Sensory symptoms	Subjective common Objective rare and usually slight	Subjective rare Objective well marked, and characteristic, hemianæsthesias, or stocking and glove type	Sensation unaffected, except slight over face on one side; deafness present on side of lesion	Sensory loss below level of compression	Subjective well marked Objective at first in hands and feet, later of segmental type	Brown-Séquard symptoms Segmental below lesion
Vision and optic discs	Amblyopia, contracted fields, central scotoma Pallor of optic discs	Amblyopia, blindness, marked contraction of visual fields Discs never affected	If vision affected due to optic neuritis or secondary optic atrophy	Vision not affected Discs normal	Vision not affected Discs normal	Not usually affected Pupils may be inactive to light
Tremors	Intention tremors in head, body, and limbs	Intention, vibratory, and rhythmic tremors	On side of lesion ataxy without spasticity; on side opposite lesion spasticity	Tremor not present	May be some instability of arm movements, but no intention tremor	Tremor may be present
Nystagmus	Nystagmus equal on both sides	Never nystagmus	Nystagmus coarse to side of lesion, fine to the other	No nystagmus	Nystagmus not present	Nystagmus not present
Reflexes	Deep reflexes exaggerated Plantars extensor Abdominals absent True ankle clonus	Deep reflexes exaggerated Plantars absent or flexor Abdominals may be absent Pseudo-clonus of foot	Deep usually exaggerated Plantars may be extensor Abdominals may be absent Clonus may be present	Deep exaggerated Plantars extensor Abdominals absent below level of lesion Clonus usually present	Deep exaggerated in early, lost in late stages Plantars extensor Abdominals absent Clonus present in spastic stage	Deep exaggerated Plantars extensor Abdominals absent Clonus present
Sphincters	Affected early	Never affected, but retention of urine may occur	Never affected until late stages	Affected with onset of paraplegia	Affected in middle and late stages	Affected early or late

Diagnosis

Hysteria has to be diagnosed from a number of maladies of an organic nature. The diseases with which it may be confounded, and the main points in differential diagnosis, are detailed in the Table on p. 548.

Prognosis and course

The phenomena of hysteria are essentially curable. They have been known to persist for many years,and yet under suitable treatment to have completely and permanently disappeared.

The disease shows a remarkable tendency to remission and relapse of symptoms – a feature, however, by no means peculiar to it, as it is also an outstanding characteristic of disseminated sclerosis, a malady with the early stages of which hysteria is most apt to be confused.

In the common hysterical symptoms – such as fits and paralysis—occurring in young persons from emotional shock – the outlook as regards recovery is most satisfactory; also in traumatic cases, when the question of compensation has been settled. It is notorious, on the other hand, that traumatic hysteria may persist for an indefinite time pending the settlement of a claim.

It is rare for hysteria, or any of its manifestations, to cause death, but this has been known to occur in severe cases of anorexia from exhaustion and inanition.

Treatment

In dealing with the hysterical person of either sex, certain principles ought to be always kept in mind by the physician. In the first place, it is important for successful treatment that he should obtain both the confidence and the co-operation of his patient. He should neither make light of symptoms which appear important to the sufferer, nor magnify others which are of only slight import; and, above all, he should enter into the details of the treatment with care, thoroughness, and knowledge.

In the second place, as the carrying out of the treatment,

sometimes a long and tedious process, devolves upon nurses or attendants, the physician should satisfy himself that these are persons sufficiently competent and trustworthy to execute his orders, and are at the same time sufficiently tactful to manage those whose emotional state, for the time being at all events, requires to be handled with skill, patience, and authority.

There is only one method universally applicable for the treatment of hysteria which is likely to lead to a successful result, viz. *isolation*. This is the basis of the well-known method of Weir Mitchell, in which the patient is removed from all intercourse, personally or by correspondence, with relatives and friends. For the proper carrying out of this system, it is essential that the patient be removed to a nursing home, institution, or hospital, and placed under the care and supervision of a tactful and thoroughly responsible nurse or attendant. Such complete isolation is essential in the majority of cases of hysteria, whatever may be the cause or the particular symptoms of individual cases. Various modifications are occasionally employed, such as sending the patient away from home under care of a nurse, or isolating her with a nurse in one of the rooms of the house, but these are rarely so satisfactory.

Having effected the isolation of the patient, it is necessary to apply certain therapeutic methods and agents. In the first place it is important that the patient should be treated by complete *rest in bed* over a period of four to six weeks or longer.

With a view to the improvement of general nutrition, it is important that abundant nourishment be given, more especially in the form of milk—three to eight pints daily. Massage may be applied, either once daily or more often, over a period of six weeks, two months, or more, according to the necessities of particular patients.

Medicinal remedies alone are of little value in the treatment of hysteria, but may be of use for the relief of symptoms, or as temporary expedients. Of these the most efficacious are the preparations of valerian, assafœtida, the bromides in small doses, iron, and arsenic. Tonic remedies, such as cod-liver oil, Easton's syrup, and quinine, may be given from time to time or during convalescence.

In hysterical paralysis and contracture, the application of the faradic current to the limbs and back is often of valuable assistance in assisting to restore motion and sensation. During hysterical convulsions the use of a strong faradic current conveyed through a wire brush and applied generally may modify or cut short a seizure.

In conjunction with the above methods spinal douches, or the alternate application of hot and cold water to the back, for a few minutes each morning are often of service.

It has already been stated that hysterical patients are susceptible to the influence of suggestion. Acting upon this element in the mental state of hysteria, many physicians have regarded hypnotic suggestion as a satisfactory therapeutic remedy. It has been urged, however, that the employment of this agent is likely to intensify a feature of the disease which it is intended to cure. Our own experience of the remedy has been limited, but those who have made a study of the subject speak favourably, not only of the immediate effects, but of the lasting results which are induced.

AKINESIA ALGERA

This term is given to an affection in which the patient is unable to move on account of pain, or from the onset of pain on movement, for which no appreciable cause can be found. Three varieties are described: (*a*) complete akinesia algera; (*b*) dyskinesia algera, in which gentle movements are still possible; and (*c*) apraxia algera, in which speaking, reading, writing or thinking are impossible, owing to the development of pains in the head. It may persist for months or years, and in one case which we have studied the patient was bed-ridden for seven years, but ultimately recovered completely.

The clinical condition is quite definite, although the nature of the malady is obscure. Möbius, who first described it, regarded the pains as being of hysterical nature, but they are probably of a paranoic or psychical character.

Part XVI

NEURASTHENIA

Neurasthenia is a condition generally associated with a lowered state of the general health and characterised by symptoms of mental and physical fatigue, usually accompanied by subjective sensory symptoms, but without any evidence of organic disease of the nervous system.

Etiology. Neurasthenia is a disease of adult life, being most frequently met with between the ages of twenty-five and fifty. Both sexes are affected, but the disease is more common among men than women.

Heredity. Heredity undoubtedly plays a part in the causation of the disease, and although it is unusual to obtain a history of grave nervous disorders, such as epilepsy or insanity in the parents, yet, in the majority of cases, there is a history of 'nervousness' or 'headaches' in one or other parent. Perhaps more important is the inheritance of a weak constitution, the patient being liable to various ailments and easily 'run down.' Alcoholism and consanguinity in the parents is noted in a small proportion of the cases.

Predisposing causes. Anything which tends to lower the general vitality is liable to bring on the disease. Thus it is that in most cases some definite physical derangement is present, either of a temporary or permanent nature, such as dyspepsia, constipation, anæmia, vaso-motor disturbance, or phosphaturia. It ought not to be forgotten that symptoms of neurasthenia may be early signs of grave organic disease, such as dementia paralytica, melancholia, malignant disease, tuberculosis, Addison's disease, myasthenia gravis, or renal disease. Of the acute diseases, influenza and typhoid fever are specially liable to be followed by neurasthenia.

In young women the strenuous combination of study with

social indulgences frequently tends to the production of the malady. Excessive child-bearing, prolonged lactation, and the climacteric period favour its development in some women, but, on the other hand, the menopause in many cases rings down the curtain upon neurasthenia of many years' standing.

Immediate causes. The most common is overwork attended with worry, and this perhaps accounts in part for the greater prevalence of the disease in urban than in rural districts. Prolonged stress and strain are other fruitful sources of neurasthenia, as was seen in many of those who served in the late South African war, and as is commonly noted in those who break down after nursing some friend or relative during a long illness.

Excesses of various kinds, more particularly of the sexual functions, emotional shocks and physical injuries may all contribute to the development of neurasthenia.

Traumatic neurasthenia is a term applied loosely to cases in which after injuries, sometimes of a trivial character, symptoms of neurasthenia or functional paralysis develop. These symptoms are due to the fright or shock attendant upon the injury rather than to any lesion of the nervous tissues or their coverings. If these cases are examined closely they may be divided into two classes: (1) those in which the symptoms are neurasthenic, and (2) those in which the symptoms are hysterical or functional. Traumatic neurasthenia is of special importance from the medico-legal aspect, as it not infrequently forms the basis of claims for compensation.

Symptoms. Neurasthenia is characterised by a number of symptoms, psychical and physical, mainly of a subjective character. They are all, however, dependent upon an exhausted, debilitated, or irritable nervous system.

The preponderance of the physical or psychical symptoms depends largely upon the social and mental status of the patient. In brain workers mental symptoms are most obtrusive, but in labourers and those whose work is physical rather than mental, bodily symptoms are the more troublesome.

Psychical symptoms. The patient gradually becomes conscious that his brain fatigues more easily, that he is unable to concentrate his attention upon his work, that his

memory has become impaired, and that he tires readily over simple exercises, which formerly gave satisfaction and enjoyment. In this state he usually resorts to 'pick-me-ups,' or seeks relief in physical or social distractions, but without success. Neither work nor pleasure interests him, he is irritable over trifles, easily worried, and cannot make a decision on even the most trivial matters. He starts to work full of energy and good intentions, which however dissolve whenever he is confronted with his task. He attempts to do something else, and again finds that his energy and will power fail him. He becomes depressed, and various fears and apprehensions begin to oppress him. He cannot sleep properly; if he falls asleep on going to bed, he wakes up in the early hours of the morning, or if after lying awake all night he gets some sleep in the early morning, he wakes up unrefreshed and miserable. As a general rule he feels better and brighter in the evening, but dreads the approach of night time. In this condition he falls an easy prey to quack literature. He reads about his symptoms, and his mind becomes filled with and obsessed by apprehensive imaginings. His conversation is entirely devoted to detailing his symptoms, and he seeks to get sympathy from all he comes in touch with. He cannot take food, and eventually becomes a misery alike to himself and his friends.

The examination of the nervous system reveals the characteristic mental state—loss of brain power and mental energy, nervousness, depression, and irritability. In other cases there is a placid, self-satisfied air of resignation, but in both types there is an overwhelming desire on the part of the patient to describe in the most minute detail every symptom, ache and pain, requiring on the part of the physician great forbearance and patience, lest he give the idea that he thinks there is nothing wrong.

The *cranial nerves* are unaffected.

The *motor system* shows general loss of power and proneness to fatigue. There may be varying degrees of fine or coarse vibratory tremor, but there is neither spasticity nor flaccidity. Observation usually intensifies the symptoms. The electrical reactions are normal.

Sensory system. Subjective sensations are referred chiefly to the head, back and legs, and are of every imaginable

character. The most common have been already mentioned. Objective sensory changes are much less common, and usually consist of a diminution rather than a loss of sensation. The chief exceptions to this rule are seen in cases of traumatic paralysis, which are described under hysteria (p. 530).

The reflexes. The deep reflexes are almost invariably increased, and a knee jerk may sometimes be obtained by tapping the shin. In these cases the eliciting of the jerk may cause the patient a 'jar at the spine' and cause him to start. With the increase in the deep reflexes, however, there is no corresponding spasticity, and clonus is not present. The superficial reflexes are usually exaggerated. The plantar reflexes are flexor in type. The sphincters are not affected.

Clinical types. Some symptoms of neurasthenia form the outstanding feature of particular cases, so that certain types have been described.

1. Cases in which headache of a persistent character is the outstanding symptom.

2. Cases in which gastric symptoms preponderate, dyspepsia of a specially troublesome and protracted form, or increase in the secretion of hydrochloric acid, or falling down of the stomach (gastroptosis).

3. Cases in which the circulatory system is especially prone to disturbances of a functional character. In these, palpitation, rapid action of the heart, tremors of the hands, suggesting Graves's disease, and breathlessness on exertion are observed.

4. Another type is seen especially in young men, in which the symptoms are referred to the generative organs and the sexual functions. In these cases nocturnal emissions are frequent and are often accompanied by a prostatic discharge. This type is associated with marked depression, hypochondriacal feelings and imaginings of sexual impotence. Sexual desire may be diminished and the sexual act imperfect from feeble erection and premature emission. The persistence of these symptoms eventually leads to distress of mind, fatigue and exhaustion, want of decision and distaste of work, and not infrequently considerable insomnia.

The physical condition in neurasthenia is sometimes not materially affected. There are some cases in which loss of

body weight is well marked. Other patients are anæmic and badly nourished. Constipation is frequent, and in most cases some flatulence and dyspepsia exist apart from definite dilatation and gastroptosis. The extremities are sometimes blue and cold.

The heart's action is frequently accelerated, but there is rarely any irregularity, and the pulse is regular and of fair volume.

The temperature is, as a rule, subnormal.

The urine is usually small in quantity, phosphates are frequently present in considerable quantities, and abnormal quantities of oxalate of lime, uric acid, and of indican have been noted.

Differential diagnosis of neurasthenia from disorders presenting neurasthenic-like symptoms. The diseases from which neurasthenia has to be distinguished are:—

1. The psychoses of puberty and adolescence—dementia precox.
2. Mild or modified types of mania and melancholia—(manic-depressive insanity).
3. The early (neurasthenic) stage of some types of paralytic dementia.
4. Psychasthenia.
5. Organic diseases of the nervous or other systems.

1. *The psychoses of puberty and adolescence.* Neurasthenia in its true form is unknown during puberty and is rarely seen in the early years of adolescence. Symptoms of a neurasthenic-like type, therefore, arising during these epochs should be viewed with suspicion and the prognosis guardedly given.

The symptoms of these psychoses consist of excitement or depression, occasionally attended by delusions or fancies of a fleeting nature. Suicidal tendencies are not uncommon. After the continuance of such symptoms for an indefinite time, spontaneous recovery or varying degrees of dementia may supervene. In other cases the symptoms are more obviously hysterical, or of a psychasthenic character accompanied by insomnia. Their prominent characteristic is a tendency towards relapse and remission.

2. *Mild or modified types of mania-melancholia.* This type of psychosis is common during adult life, and in its

milder forms may readily be mistaken for, and is frequently treated as, neurasthenia. Although this psychosis presents the exhaustion, feebleness of will power, and want of energy so well shown in neurasthenia, the chief feature by which it may be distinguished from the latter is the marked tendency towards melancholy or mental depression. It is often accompanied by considerable insomnia. The majority of patients suffering from this type of psychosis will give a history of one or more previous attacks.

It is rare in young people, but not uncommon throughout adult life and at the climacteric epoch in women.

3. *The neurasthenic type of early paralytic dementia.* This occurs chiefly in adult males who have had syphilis. Such cases may, or may not, present physical signs of organic disease, such as loss of the tendon jerks, or reflex pupillary immobility. If these signs are found in association with neurasthenic symptoms, the onset of paralytic dementia may almost with certainty be diagnosed. The failure to obtain these signs of organic disease does not necessarily exclude the onset of paralysis; therefore in those cases a lumbar puncture ought to be done and a cytological examination made of the cerebro-spinal fluid. If, on examination of the centrifuged deposit, the lymphocyte count shows 150 to 200 or more lymphocytes, the diagnosis of general paralysis may be made with complete assurance. Although an increase of the lymphocytes is present in most cases of cerebro-spinal syphilis, yet the count is rarely so high as in the parasyphilitic diseases.

4. *Psychasthenia.* This type of psycho-neurosis as found in young adults is largely of a remitting character, while in the later years of life it may be more persistent and chronic. It is primarily characterised by the obtrusiveness of certain morbid fancies and fears, apprehensions, fixed ideas and obsessions. It is less likely to be confounded with neurasthenia than those conditions just described.

5. *Organic diseases of the nervous or other systems.* In every case presenting neurasthenic symptoms a most complete examination is necessary, and this should be repeated at intervals.

Course and prognosis. Neurasthenia runs a prolonged course, sometimes lasting many years. Its duration, however, depends to a large extent upon the cause. If due to a

temporary and removable stress or strain, recovery under suitable treatment may occur within a few months. But the temperament which favours the development of the disease usually favours the prolongation of the symptoms. If the patient is in a position to abandon work, to undergo a thorough 'rest cure,' or to take a sufficiently extended change, recovery usually results.

In cases attributed to toxic causes, such as influenza, enteric fever, or other post-febrile states, the prognosis is good; in those associated with pyorrhœa and rheumatoid arthritis, the prognosis is that of the causal condition.

In traumatic cases the 'rest cure' finds one of its most useful applications. Should an action for compensation be impending, there is little chance of recovery until this is settled; when settled the recovery is in most cases rapid and complete.

Cases of 'sexual neurasthenia' are persistent and of an inveterate character, owing to the tendency towards hypochondriasis and mental depression.

Treatment. The treatment of neurasthenia is based upon the efficient carrying out of rest, both mental and physical. For this purpose the 'rest cure,' as described by Weir Mitchell, is of great use. The rest cure consists of isolation, rest in bed, massage and abundance of nourishment, preferably in the form of milk. It is usually carried out in homes situated preferably in the country, so as to permit of the patient being carried out into the open air. All cases of neurasthenia, however, do not respond satisfactorily to this method. Men are less amenable to the treatment than women, and in them a modified course may be found sufficiently efficacious. In the modified rest cure, isolation is not essential, while rest may be judiciously combined with mild forms of outdoor recreation. Cases of neurasthenia complicated with pronounced insomnia, or with much mental depression, are not favourable for the rest cure.

An important means of treatment, particularly in the early stages of the disease, or in mild cases, consists in sending the patient away from work and enjoining a change of scene. All forms of climate are not, however, suitable to neurasthenics, and the choice of a satisfactory locality is often a matter of difficulty. Just as in health some individuals

favour the seaside more than the higher altitudes, so in neurasthenia the selection of the locality should be determined by what the patient has found most beneficial when in health. A point of importance in this matter is the avoidance of long railway journeys, and of frequent changes from one place to another. For this reason many neurasthenics who are sent abroad, or upon a voyage round the world, return not only with no material benefit, but often with a decided aggravation of their symptoms.

All forms of outdoor recreation, if not carried to excess and to the production of physical fatigue, are to be encouraged. To the worker in the town, two or three afternoons a week given to golf, or riding exercise, may keep off the onset of neurasthenic symptoms.

Drugs in the treatment of neurasthenia are of little use, except to relieve temporary symptoms. In young women perhaps most advantage is to be obtained from the glycerophosphates of lime and soda, in other cases Easton's syrup acts as a powerful and satisfactory tonic. Of others which may be mentioned, nux vomica, quinine, the mineral acids, the hypophosphites, valerian, iron and arsenic are of most service. In cases presenting much restlessness, with palpitation and a tendency towards insomnia, the bromides find one of their most useful applications. They ought not, however, to be used for more than a short time. In the type of case characterised by headache, analgesics such as antipyrin, phenacetin, and aspirin may be advantageously given from time to time.

In cases presenting symptoms of either gastroptosis or dilatation, a carefully regulated diet with abdominal massage will afford relief, attention being paid to the mouth and teeth. In the event of hyperchlorhydria being present, the excess of acid may be neutralised by hot water, or by a natural alkaline water such as that of Vichy.

Hydrotherapy and electricity may both be useful in otherwise intractable cases of neurasthenia. Neither of them should be relied upon solely, but may be prescribed as auxiliary methods.

The only certain and satisfactory treatment of genuine neurasthenia is complete mental and physical rest, combined with attention to the complicating features already described.

Part XVII

PSYCHASTHENIA

This is a psycho-neurosis characterised by the presence of a variety of symptoms of which nervous fears, apprehensions, and obsessions are the most prominent. It is often accompanied by periodic seizures of a vertiginous character, which are combined with head sensations, aches, and pains. Another feature of the malady is a peculiar dazed reverie or dream state, in which objects and persons do not seem to be real, but which is not associated with any loss of consciousness, such as occurs in epilepsy.

Etiology. The disease is commonest between the ages of twenty-one and forty, but may be found both during puberty and after fifty. It is about equally distributed between the sexes, and seems to stand in some way to the development of the sexual functions, being found in its most typical forms during adolescence. It is as frequent amongst hospital as private patients.

A hereditary neuropathic history is not always obtainable, but when present will be found to be chiefly in the direction of parental alcoholism and 'nervousness.'

The psychopathic nature of this malady is seen in the nervous symptoms presented by the relatives of many of these patients, such as epilepsy, insanity, and alcoholism, and in such nervous associations as periodic headaches, hysterical manifestations, somnambulism, occasionally chorea, tic-like movements, nervous dyspepsia, and the traumatic neuroses.

Symptoms. These are in part temperamental or congenital and in part acquired. The former constitute the psychasthenic or ideo-obsessive constitution, which is common to most psycho-neurotic persons; the latter are seen in the

development of morbid fears, obsessions, and states of reverie or dreaminess.

Of the *obsessions*, an enormous assortment is described by patients. Only a few common instances need be given. Sometimes they are merely general fears and apprehensions, as of 'something going to happen'; fears of being alone, of being in the dark, of being in a crowd, or in a confined space, or they may have reference to some special phenomenon such as becoming insane, or paralysed, or of dying. A curious apprehension is found in a fear of falling through the bed, or the floor, or of sinking through the ground. Or again, sufferers from this affliction describe a feeling of apprehensiveness that they may inflict injuries upon themselves or others, when they handle knives or razors. Sometimes these sensations become so engrossing that a fear of homicide or of suicide is present, although self-destruction rarely occurs.

Allied to the above are states of 'panic,' such as sudden desires amounting to impulsive actions to get out of a train in motion, or to escape from a crowded church or theatre.

In other instances the obsession takes the form of a paralysis of action, the patient being afraid to go about, but yet dreading being alone; or an utter inability to decide upon a particular course of action.

Others are tormented by conscientious scruples, or by ideas that a word lightly spoken may have led to harmful consequences.

Probably of the same fundamental nature as the above, and of greater significance, are the drug habits—dipsomania, morphinomania, and cocainomania. Various moral delinquencies may also be grouped under this heading, amongst which the sexual perversions may be mentioned.

The *reverie or dream states* of psychasthenia have to be distinguished from somewhat similar conditions occurring as the aura of epileptic seizures. In psychasthenia they consist of spells of dazedness, in which the patient feels as if he is in a dream. During this phase there is a feeling of unreality, sometimes referred to objects and people around him; at other times to his own organs and body. These patients state that they have no real existence, that they do not seem to have any body, or that their organs have gone. Others suffer the feeling that 'they are not what they are,'

that nothing makes any impression upon them; they even doubt whether what they see and hear are real.

The psychasthenic condition is also accompanied in many instances by periodic attacks, which seem to occupy a position somewhere on the 'borderland' of epilepsy. Although these may occur during sleep and wake the patient, they are never associated with loss of consciousness or convulsion. To some of these seizures the term 'psycholepsy' has been applied by Janet. They consist of attacks of giddiness, throbbing, and palpitation of the heart, a feeling of choking, a sense of fear and sometimes of impending death. They may be accompanied by excessive perspiration and followed by great exhaustion. Their duration may be from a few to several minutes. In our opinion these attacks are not primarily psychical, but are the consequences of functional disturbances of the lower centres in the medulla oblongata.

The *head sensations* and other paræsthesia are numerous and varied, such as giddiness, feelings of a burning character, sensations of emptiness in the head, or as if the head was opening and shutting, or of tightness round the head. Sometimes there are feelings as of something trickling down the back of the head, or moving about within the brain, or of confusion in the head. Others describe a sensation of bursting, or 'popping,' inside the head.

These sensations may or may not be accompanied by mental depression.

When the symptoms are intense, or at the commencement of a seizure, a feeling of sinking in the epigastrium is often present, and gives rise to the impression that the disease originates in this locality. Dyspepsia, however, is not common, although flatulence and constipation are occasional accompaniments.

Pain in the head is rare, so also is insomnia, although the nights may be disturbed by bad dreams and some tendency to restlessness.

Although fatigue is readily induced on exertion, and an inability to concentrate the attention for any length of time may be present, neither of these symptoms occupies the outstanding position which is characteristic of neurasthenia. The memory may be impaired temporarily during the persistence of the symptoms. One of the features characteristic of

the malady is the inability to stand the effects of alcohol. Although the symptoms may be for a time relieved by it, most cases present a marked idiosyncrasy towards its continued use.

In the psychasthenic condition there is an absence of physical signs of nervous disorder. The reflexes are unaltered, although the knee jerks may be slightly exaggerated. There is no tenderness on pressure over the head or along the spine. Occasional subjective circulatory symptoms such as palpitation, blushing, and morbid flushings are observed, and a proclivity towards flatulence, discomfort after food, and constipation have been already noted. The tongue is usually clean, but has been found to be furred and coated in some cases in which the onset of the symptoms is sudden and their progress acute.

Prognosis. The course and prognosis of the malady is variable. When it occurs in adolescents and young adults —where it is seen in its most characteristic forms—it presents remissions and relapses as its main peculiarities. Under favourable conditions it is a curable malady, but running a prolonged course over many years. When it arises 'de novo' in middle life, the prognosis is less good, as it is more apt to become confirmed, and to be subservient to the varying conditions of overwork, anxiety, shock, worry, or ill-health.

There is no tendency towards dementia. Temporary depression may occasionally supervene, more especially at certain epochs, such as puberty, adolescence, and the climacteric in women, but the underlying obsessive phenomena are not altered. The persistence of fears and apprehensions may give rise to a condition of hypochondriasis and depression.

Treatment. The treatment of psychasthenia and the ideo-obsessive state is fraught with numerous difficulties. In the first place, the symptoms do not lend themselves to amelioration by means of drugs, although valerian and its preparations are more likely to be of assistance than any other remedies, especially if combined with small doses of the bromides.

The rest cure, which is so valuable a therapeutic agent in the treatment of neurasthenia, is more likely to aggravate the symptoms in the psychasthenic. There are, however,

cases in which nervous and physical fatigue form prominent symptoms, and in which a modified rest cure may be of distinct service.

For the treatment of this condition, congenial surroundings are very important. These patients should give up work for a time and lead an open-air life with suitable exercise and amusement. It is important to advise a complete change, as the malady is not infrequently induced by uncongenial and sedentary employment.

Psychotherapy, or treatment by means of suggestion, has been employed advantageously in a number of cases. In many the results are encouraging, and the method should be used in those persons in whom other methods of treatment have been of little or no avail.

Part XVIII

EPILEPSY

Epilepsy is a chronic progressive disease, characterised by the periodic occurrence of seizures, in which loss of consciousness is an essential feature, commonly, but not invariably, associated with convulsion, and frequently accompanied by psychical phenomena.

It generally occurs in persons with a hereditary neuropathic history, which may show itself in signs, or stigmata, of degeneration; it runs its course uninterruptedly, or with remissions, over a number of years, and it terminates either in a cure, in the establishment of the confirmed disease, in delusional insanity, or in dementia.

Etiology

Epilepsy is a prevalent disorder, and although its frequency varies, according to the obtainable statistics from different countries, it may be stated that, on an average, two persons in every thousand of the population are epileptic. Males are more commonly afflicted than females, the greater frequency of the male sex not necessarily depending upon the greater stress and strain to which this sex is exposed, but being a feature of the disease from infancy to old age.

Heredity. The great predisposing cause of epilepsy is an inherited neuropathic predisposition. A family history, more especially of epilepsy, alcoholism and insanity, is obtainable in about 50 per cent. of the cases.

The hereditary transmission of epilepsy may be direct from parent to offspring, or the disease may reveal itself in collateral relatives, such as uncles, aunts, and cousins. Two or more members of the same family may be afflicted, and

periodic headache, 'bilious' attacks, migraine, and chorea may be found in other members.

Age at onset. The commencement of epilepsy is dated from the occurrence of the first fit—a fact which is sometimes difficult to ascertain, as it may occur during sleep, or assume a form which is not recognised as of epileptic character. The greatest number of cases of epilepsy, commencing in any single year, is found during the first twelve months of life. There is then a rapid and extensive fall in the number of cases to the fourth year, from which there is a slight increase up to the seventh year. A small decline occurs about the eighth or ninth year, preliminary to a steady increase in the disease, which reaches a maximum between the ages of twelve and fifteen. From this period onwards, there is a decrease in numbers, with a slight temporary increase between the twenty-fifth and twenty-eighth years.

Although there is no age which may be regarded as exempt from epilepsy, the onset of fits is more common during certain epochs, which may thus be defined: (*a*) from birth to eight or nine years—corresponding to the periods of rapid brain growth and development; (*b*) from the tenth to the twenty-third year—the period of development and maturation of the reproductive organs. The explanation of the onset of epileptic fits is to be found in the normal instability of the nervous system during these two periods, acting upon a constitution predisposed by heredity to convulsions.

In women the onset of epilepsy is almost always accompanied by irregularity in the catamenia, and the relation between 'the period' and the fit incidence is well known. In close association with this function is the influence which pregnancy, the puerperium, and lactation have upon the onset or course of epilepsy. These incidents in the life of a woman may be a cause of epileptic seizures, or they may induce a relapse after a long remission from fits; on the other hand pregnancy and child-bearing have been known to favourably influence the course of epilepsy.

The *causes* of epilepsy are:—

1. A neuropathic heredity as already mentioned.

2. The normal instability of the nervous system during certain epochs in those predisposed by heredity.

3. Miscellaneous exciting causes acting in conjunction

with a neuropathic heredity and the normal instability of the developmental epochs. Amongst such causes are: (1) *Reflex* influences—arising from disease of the nose, eyes, ears, teeth, stomach, and intestines (including the influence of intestinal worms), and from the genital organs. (2) *Psychical* causes—such as fright, mental emotion, prolonged anxiety, grief, and overwork. (3) *Infective, pyrexial,* and *toxic* causes. The most important of these are scarlet fever, alcohol, and tobacco. (4) Seizures of an epileptic character may be associated with *organic disease of the brain.* This does not constitute epilepsy as here defined.

The following are the *organic conditions* in which generalised epileptic fits may occur: (*a*) defective development of the nervous system in infancy and childhood (p. 212); (*b*) cerebral lesions in infancy and childhood (p. 216); (*c*) syphilitic affections of the brain (p. 370); (*d*) vascular diseases of the brain (p. 186); (*e*) tumours (p. 226); (*f*) hydrocephalus and general increase of intracranial pressure (p. 288); (*g*) injury to the head.

Pathology

The *macroscopical* appearances of the brains of epileptics vary. The brain is usually large, the convolutionary development simple, and the membranes not adherent to the cortex. A foam-like or frothy exudate may be seen lying between the membranes and the surface of the cortex. More or less sclerosis of the cortex is present, especially of the outer layers and of the subcortical white matter in cases of old-standing epilepsy complicated with dementia. This sclerosis is best seen in the region of the cornu ammonis, which is atrophied in considerably more than half the cases of this disease. Atrophy of the optic thalamus, more commonly upon the left side, has been described. Other portions of the brain—such as the cerebellum and the medulla oblongata—may also be atrophied. Small petechial hemorrhages, angeiomata or blood tumours, some thickening of the arterial walls, and engorgement of the blood-vessels have also been occasionally noted.

The *microscopical* appearances vary to a large extent, but the following changes have been observed:—

(a) *Changes which are accompaniments, or stigmata of a defectively developed nervous system.*

Diminution in the number and size of the nerve cells of the outer layers of the cerebral cortex has been described The nucleus of the large pyramidal cells is displaced and the cell body presents a granular appearance. The persistence of nerve cells in the white matter and outer layers of the cortex —a feature common to the lower vertebrates, new-born infants, and imbeciles - has also been found.

(b) *Changes which are probably the direct consequences of the seizures.*

Increase of the neuroglia, especially of the first cortical layer and the subcortical white matter, has been observed in old-standing cases of epilepsy. A patchy sclerosis is also found in other parts—such as the optic thalamus, occipital lobes, cerebellum, medulla oblongata, and cornu ammonis. It is associated with a great increase in the neuroglia cells.

The blood-vessels. The veins are engorged and the arteries collapsed and tortuous. The lymph spaces are widely dilated. Hyaline spheres or masses and fibrin threads are seen lying freely in the lumen of the blood-vessels, not only of the cortex, but also in the cerebellum, medulla oblongata, and spinal cord.

Chemical pathology. *Urine.* This presents normal features, except that after a severe seizure, or a series of fits, albumen may be temporarily present. The uric acid excretion is stated to be considerably lessened before a fit and increased afterwards (Haig, Krainsky). Observers are at variance as to whether the toxicity of the urine in epileptics is greater than in normal persons.

Blood. The general characters are those of an average type of chlorosis. A slight leucocytosis is constant, and is markedly increased after a fit. A sudden fall in alkalinity occurs prior to and after a fit (Pugh). There would appear to be an increased tendency to coagulation of the blood.

Cerebro-spinal fluid. Cholin has been found in this fluid in epileptics, probably as a result of the fits. Lymphocytosis has been occasionally observed.

Thymus gland. This gland has been found enlarged in a number of cases.

The examination of the fluids and secretions of the body do not afford an entirely satisfactory basis on which to build

a theory of *auto-intoxication.* Evidence of a toxic condition of the blood exists in hyper-leucocytosis and in a tendency to intravascular clotting, conditions which are present in some of the clinical types of epilepsy. Other views of the auto-toxic causation of epileptic fits are Haig's uric acid theory, Krainsky's carbamate of ammonium, Donath's cholin, and Ceni's cytotoxins. Of none of these is there certain proof: the evidence, such as it is, pointing to these substances as being the effects rather than the cause of epileptic seizures.

Symptomatology

The symptoms may best be described under the two subheadings into which epilepsy may be clinically divided: the convulsive and the psychical elements.

The Convulsive Element

This constitutes the fit in its numerous and varied manifestations. In order to understand and appreciate the manifold features of the epileptic seizure, the doctrine propounded by Herpin may be stated, that the incomplete attacks—cramps, spasms, giddinesses, or partial convulsions—which occur irregularly in the intervals between the major attacks, are the complete seizures reduced to their initial symptoms, and, however diversified they may be, are always or nearly always similar in the same subject.

For descriptive purposes the fit may be subdivided into:

1. The aura.
2. The incomplete fit.
3. The complete fit.

The aura, or warning, is the initial symptom of the attack, or may constitute the whole seizure.

The incomplete attack, or minor fit, consists of the aura, with a further development towards the phenomena of the complete seizure—a type of attack which may or may not be associated with loss or impairment of consciousness.

The complete attack, or major fit, forms the classical epileptic seizure, in which, with or without warning, the victim falls from sudden loss of consciousness and is convulsed.

The warning spreads almost always, though not invariably,

from the periphery of the body towards the head. The sensation ascends from the hands or feet, from the thoracic, epigastric or abdominal organs, or from the organs of special sense. In many cases it is noted that when the sensation reaches the head consciousness is abolished. Sometimes, however, consciousness is abolished at an early stage of the fit: at other times general convulsion commences before consciousness is lost, but in the majority the loss of consciousness occurs synchronously with the onset of muscular spasm or convulsion.

1. Auras and aura fits

Any part of the body may theoretically be the starting-point of a seizure, but there are certain localities in which the onset is more common. The warning may commence in one or other of the following ways, or the entire seizure may be confined to the warning sensation.

(*a*) Sensations referred to the stomach, abdomen, or chest. These consist of various abnormal sensations – such as cramps, spasms, and sometimes pain, occasionally feelings of suffocation or of cardiac distress.

(*b*) Head sensations – such as pains, 'jumping' or 'horrid feelings' in the head; or of something 'striking the brain,' &c.

(*c*) Sensations of a psychical character – such as dreads, sense of fear, &c. which are occasionally associated with a dreamy state or a feeling of unreality or of non-existence.

(*d*) Subjective or perverted sensations of special sensibility, olfactory, gustatory, auditory, and visual warnings. It is especially in association with the olfactory sensations that the dreamy state is present – a symptom which has been found to arise from disease of the uncinate gyrus (p. 255).

(*e*) Sensations referred to the limbs and body – such as the hand, arm, face, leg or foot, head and eyes and the back. The warning may be unilateral or bilateral, and consist of numbness, 'pins-and-needles,' subjective sensations of coldness.

In the event of the warning constituting the whole seizure, consciousness may or may not be temporarily impaired. On the other hand, the warning may proceed to a further development and give rise to the incomplete or complete seizure, as the case may be.

2. Incomplete attacks. Minor fits

As just explained the incomplete attack may consist of the warning sensation with a further development towards the complete seizure along with some interference with consciousness. Hence many incomplete attacks begin with peripheral, epigastric, cephalic, psychical and special sense warnings. Many, on the other hand, have no such aura. These form an important type of minor epileptic seizure. In this category are all those seizures in which consciousness is temporarily lost or impaired, but which are unaccompanied by more than a trifling convulsion. Such are cases of sudden fall, 'loss of memory,' 'spells,' deviation of the eyes and a cry, pallor of the face with deviation of the head and a fall, a sudden cry accompanied by a fall, and many other attacks of a similar character. These attacks are not uncommonly followed by a somnambulistic-like stage, or stage of *automatism.*

3. Complete attacks. Major fits

The major attack may commence with one of the warnings already described, or with sudden and complete loss of consciousness. In a considerable number of cases the fit is ushered in by a cry, resembling a screech, which is produced during inspiration. The loss of consciousness precedes or accompanies the cry. The patient falls down in a state of tonic spasm, which is generalised, but invariably affects one side more than the other. The head and eyes are deviated to one side and the body may be rotated to the same side. The arms are abducted at the shoulder, flexed at the elbow and wrist, the fingers being either extended or flexed. The legs are extended and rotated inwards and the feet inverted. The face becomes livid and cyanosed, and is drawn over towards the more affected side. On the less affected side the leg is extended and the arm may be extended or abducted. During the tonic stage respiration is in abeyance in full inspiration. The superficial veins stand out prominently. This stage may last from a few seconds to a minute or a minute and a half, and when prolonged, the intensity of the spasm may pass from the more to the less affected side, so

that the head and eyes may be deviated to the other side. The spasm may be so great as to cause dislocation of the shoulder or jaw, accidents which may readily occur in succeeding seizures. Death may occur from suffocation in severe cases owing to the face being buried in the pillow.

The first sign of the yielding of the tonic stage is the development of a fine rapid tremor. This rapidly gives place to sudden, quickly repeated, shock-like contractions of greater degree, which become of larger range and greater severity, and occur at gradually lengthening intervals, and finally cease after two or three desultory jerks. The limbs and body are then in a state of flaccidity. Towards the latter part of this stage respiration is resumed—at first by a series of jerky expirations passing into stertorous breathing. The duration of the clonic stage varies, but is longer than the tonic, and clonic movement may continue for three or four minutes after the cessation of the tonic stage.

In slighter seizures the tonic stage may be brief, with fine tremor persisting throughout, the seizure being limited almost entirely to one side, with little or no true clonic stage.

During the tonic stage the pupils are dilated and inactive to light. On return to consciousness they resume their normal size, although if the fit has affected one side more especially the pupil on that side may remain more dilated than its fellow. The pupillary light reflex may return during the clonic stage, even when the pupils are still dilated.

The clonic stage is followed by one of muscular relaxation and stupor, from which the patient passes into deep sleep and finally recovers.

Accidents accompanying fits. During the tonic stage ecchimoses, dislocations, fracture of the teeth and bones, evacuation of urine and fæces, and suffocation may occur. During the clonic stage tongue-biting is very common, and may be a feature of some cases.

Sequelæ of epileptic fits

If the patient be examined immediately after a severe fit, a condition of general muscular relaxation is observed. Temporary loss of the deep reflexes may be demonstrated, but as a rule the deep reflexes are increased and clonus easily

elicited. The superficial abdominal reflexes are temporarily abolished. The behaviour of the plantar reflex is interesting. At first, no response may be obtained on stimulation; later an extensor response is obtained, which after a short period gives place to flexion. Eventually the reflexes become normal, although a permanent increase of the deep reflexes is usual in epileptics.

Transient motor and sensory paralyses—exhaustion paralyses—may also be observed. They are best seen in cases in which one side has been predominantly convulsed. The head and eyes may be deviated towards the less affected side with a slight degree of motor weakness on the opposite side.

Sensory phenomena consist of a slight and general blunting of sensibility over the side which has been most convulsed. This rapidly clears away, the proximal segments recovering before the distal. These phenomena are comparatively rare, but occur in cases in which no evidence of gross cerebral lesion has shown itself in the subsequent history of the case. The transient nature of the symptoms may therefore be put down to a temporary exhaustion rather than to a progressive destruction of the cortical centres.

Amblyopia, restriction of the visual fields, deafness, and defects of smell and taste have also been observed.

The immediate psychical effects are described on p. 569.

The Psychical Element

The mental conditions found in epilepsy are:—

1. The epileptic character and temperament.
2. The paroxysmal psychoses which precede or succeed the convulsive phenomena.
3. The psychoses which replace the convulsions—psychical epileptic equivalents.
4. The permanent interparoxysmal mental state.

1. Temperament

In epilepsy it is usual to find some form of mental obliquity. The memory is usually impaired for recent events, and is often of a perverted kind. Epileptics are self-opinionated and egotistical, and their conversation is prolix and pretentious. Their mental perspective is blurred and

disproportioned. Their judgment is feeble, they are frequently credulous and mystical and given to superstitious ideas and fancies. The majority possess a religious fervour, which forms a marked feature of the disease and contrasts strongly with their actions, their ideas of right and wrong being often vague. Their want of initiative is striking, and mainly on this account they require supervision and direction.

They are subject to moods of hastiness of temper and pugnacity alternating with laziness and lethargy, which make them difficult to live with and often useless as workers. Many show habitual irritability of temper and awkwardness of disposition, while others are imbecile, demented, delusional, or liable to dangerous impulses.

2. Paroxysmal psychoses. Epileptic insanity

These forms of mental aberration or perversion are incidental to the seizures, and occur either as prodromal phenomena or as immediate sequelæ of the attacks. They include all the hallucinatory, delusional, maniacal, melancholic, and psychasthenic states which are observed in the subjects of epileptic fits.

If they are *pre-paroxysmal* in occurrence, they ought to be regarded as a prolonged or modified prodromal stage.

If *post-paroxysmal*, they take the form of definite psychical phenomena lasting for variable periods.

The commoner post-paroxysmal forms are:—

(*a*) Acute epileptic dementia: a condition more commonly observed after serial or status outbursts, consisting of profound, though temporary, mental and physical debility and stupor.

(*b*) Acute epileptic mania: a rarer psychical condition than the preceding, but of much importance on account of the violence of the psycho-motor disturbances. It is usually brief, not lasting more than about twelve hours. Minor degrees are talkativeness, tiresomeness, and exhilaration of spirits.

(*c*) Transitory delusional states. These are a common form of post-paroxysmal psychosis, and may last for several days before the patient is restored to his normal mental condition.

(*d*) Automatism. Most seizures—but especially the minor forms—are succeeded by some kind of automatic action, of which undressing is probably the most common.

3. Psychical epileptic equivalents

It is now generally conceded that psychical phenomena may replace convulsive seizures in some cases of epilepsy. The general tendency of recent writing on this subject has been to enlarge the scope of the epileptic manifestations, and to regard as epileptic various psychical conditions of a periodic nature. In this way some psychical states have been included amongst the epileptic manifestations, which are in reality common to all the psychopathies.

Our own observations confirm the opinion that no mental state occurs as an epileptic equivalent, which is not also seen as a pre- or post-paroxysmal psychosis. We are, therefore, in favour of defining psychical epileptic equivalents as the mental phenomena of the pre- and post-paroxysmal states, occurring without convulsion or spasm.

The following conditions are seen as psychical equivalents of epileptic fits:—

(*a*) *Psychical epilepsy*, or attacks of brief duration, consisting mainly in the performance of simple automatic actions. Attacks of a more prolonged character—ambulatory automatism—are sometimes epileptic, but are more commonly hysterical. 'Wandering' is a not uncommon feature in confirmed epilepsy, but is also seen in alcoholism, dementia, and congenital mental deficiency.

(*b*) *Epileptic mania*, resembling in all respects the acute maniacal outburst of the post-paroxysmal psychosis is rare, but should be recognised. It has received the name of 'grand mal intellectuel.' In contrast to it is the 'petit mal intellectuel,' or *epileptic impulsion*—short attacks of extreme suddenness, in which the patient may effect a homicidal act.

(*c*) *Transitory delusional states.*

(*d*) *Catatonic stupor.*

4. The permanent dementia

The permanent or inter-paroxysmal mental state of epileptics varies from that in which little or no mental impairment can be detected to one in which all the features of dementia are observed—viz. defective memory, confusion of ideas, impaired capacity for work, absence of initiative, and a slow and

dull comprehension. The proportion of epileptics in which this condition is present varies; but it may be stated that in institutions for epileptics, where those are collected who are unable to work owing to their infirmity, only about 18 per cent. of the inmates are without some mental deficiency.

The inter-paroxysmal dementia, although an integral part of the disease, is modified by various factors—such as a strong neuropathic family history, the duration of the seizures, the age at the onset, and the frequency and character of the attacks.

Clinical Types of Epilepsy

Epilepsy is commonly described and its clinical varieties grouped according to the type, the frequency, or the time of occurrence of the seizures. If classified according to the character of the seizures three main divisions may be described—the major, minor, and combined types; if according to the periodic frequency of the seizures—serial epilepsy, and the status epilepticus; if according to the time of occurrence—nocturnal and diurnal epilepsies.

The *major type* of epilepsy is characterised by the recurrence of major convulsive seizures varying in frequency from one or two up to twenty or more *per mensem*. As in all forms of epilepsy there is great constancy in the occurrence of the fits, little change either in character or frequency being observed over long periods of time. The mental condition in this type is not associated with any special degree of dementia, which is indeed largely determined by the frequency of the fits. During many years the mental condition may not show signs of deterioration, but a marked increase in the fits, or an attack of status epilepticus may be the starting-point of progressive dementia or delusional insanity.

The *minor type*, characterised by the presence of minor seizures only, is in its pure form not common. The 'sensations' may be very frequent—up to several daily. The usual psychical state consists of a mild degree of mental impairment—chiefly loss of memory for recent events.

The *combined type* is characterised by the combination of major and minor seizures, sometimes associated with

psychomotor attacks. It is the type of epilepsy which shows the greatest frequency of fits, sometimes up to several hundred in the course of a month or two. As might be expected this type presents the most profound degrees of dementia, and is little influenced by treatment.

Serial epilepsy is a type of the disease in which a series or succession of fits of either the major or minor type, or of a combination of major and minor attacks, forms the picture of the disease. It is a subacute form of epilepsy and differs from the status epilepticus by a more or less complete return to consciousness between the seizures. It may occur as the type of the disease, as a modified form of status, or as an occasional complication of the major, minor, or combined types. The serial outburst is usually heralded by a prodromal increase in the single seizures, which increase in frequency until a climax is reached, from which a numerical decrease in frequency occurs, with eventual cessation of fits for a time. This type gives rise to temporary mental failure which has been already described, and not infrequently terminates in dementia.

Status epilepticus is the most acute manifestation of epilepsy, and in some of its developments may form the type of the disease. It may be an occasional feature of all forms of epilepsy, or it may result from some accidental circumstance, such as sudden stoppage of the bromides, emotional shock, an acute inflammatory disorder, or a fall or blow upon the head.

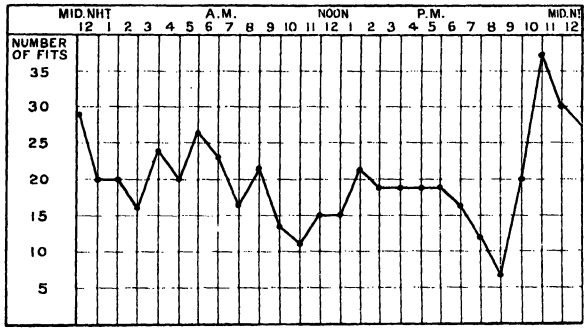
Its onset is foreshadowed by the increase of the ordinary seizures, which increase in frequency, until one attack succeeds another so rapidly that consciousness is not regained. The attack ends in coma and exhaustion, in which the patient may die, or he may recover after a short period of delusional insanity and irritability.

Status epilepticus is the most serious complication of epilepsy, and although the prognosis is grave, it is not necessarily fatal.

Nocturnal and diurnal epilepsy. There is a type of epilepsy, in which the fits occur only during sleep; and it is a well known fact that in the combined type of the disease, the minor fits may occur by day, and the major during the sleeping hours.

The usual incidence of epileptic fits is an irregular periodicity, and observations have frequently been made to ascertain whether any hour of the day or night especially favours the occurrence of seizures. Our own observations are recorded in the annexed chart, which shows (1) that fits are more common during sleeping than waking hours, and (2) that the fit incidence attains its maximum during the early hours of deepest sleep.

CHART SHOWING THE HOURLY FREQUENCY OF FITS IN SIXTY-TWO CASES OF EPILEPSY



Onset, course, and termination

Epilepsy may commence in a variety of ways, of which the following are the most frequent:-

(*a*) As the minor type, which may persist for some years before a major convulsion ensues.

(*b*) As the major type which persists.

(*c*) As the nocturnal variety, which may exist unrecognised for many years.

(*d*) By the occurrence of a single fit, which may precede the full development of the disease by months or years.

(*e*) By an attack of status epilepticus, occurring during confinement or the puerperium.

Commencing in one or other of these ways, epilepsy may persist for many years. The frequency of the seizures varies enormously, but a general idea may be obtained by reference to the following Table, which shows the total number and percentage frequency of epileptic fits in 300 cases of the disease, observed in an institution for epileptics.

Frequency.	*Total.*	*Percentage.*
Daily fits . .	46	15
Weekly fits . .	133	44
Monthly fits . .	94	31
Quarterly fits . .	12	4
Yearly fits . .	12	4
At longer intervals .	3	1
Total . .	300	99

Long remissions, sometimes lasting for many years, are a not uncommon feature of epilepsy. They may be seen:—

(*a*) During childhood, usually from about five to twelve years of age, in those whose fits commence in infancy. A rarer period of remission extends over the ages of puberty and early adolescence.

(*b*) After the complete establishment of the disease, either spontaneously or assisted by treatment. These remissions may last from two up to thirty years, but the commonest period of freedom lasts about three or four years. Freedom from fits for more than four years is frequently regarded as a 'cure,' of epilepsy.

Termination. Epilepsy is a progressive disorder ending either in a cure in a small percentage (approximately ten per cent.) or in mental infirmity, delusional insanity or dementia. The actual cause of death is found in pulmonary complications, the status epilepticus, accidents occurring during a fit, organic heart disease, or as the direct consequence of a fit.

The mean age at death is stated to vary from thirty to forty-eight years of age.

Diagnosis

The recognition of epileptic seizures is based upon certain features, one or more of which should be present before a diagnosis of epilepsy is made. These are:—

(*a*) Sudden loss or obscuration of consciousness. The existence of this feature at some period is essential to the diagnosis of epilepsy, although each and every epileptic manifestation is not necessarily associated with a loss of consciousness.

(*b*) A sudden fall from obscuration of consciousness is an epileptic symptom, but many types of seizure are unaccompanied by a fall.

(*c*) The sequence of tonic spasm followed by clonic convulsion, with their effects—bed-wetting and tongue-biting—is conclusive evidence.

DIFFERENTIAL DIAGNOSIS BETWEEN AN EPILEPTIC AND AN HYSTERICAL FIT

	EPILEPTIC FIT.	HYSTERICAL FIT.
Cause	No exciting cause	Exciting causes frequent
Aura	May be present	Usually no aura
Onset	Sudden; sometimes with a cry	Gradual; sometimes with screaming
Consciousness	Lost	Impaired or perverted
Fall	Sudden; often causing injury	Gradual; does not cause injury
Corneal Reflex	Absent	Impaired, sometimes absent
Pupil Reflex	Reaction to light abolished	Reaction to light retained
Motor symptoms	Tonic spasm passing into clonic. Conjugate deviation of head and eyes to side more affected; movements unaffected by interference	Tonic spasm, opisthotonos or sudden change to prosthotonos. Movements semi-purposive and co-ordinated. Strabismus divergent or convergent. Movement increased by interference.
Micturition	May occur during tonic stage	Occurs after fit is over
Tongue-biting	May occur during clonic stage	Never occurs, but patient may bite her lips or her attendants
Post-convulsive stage	Stupor, drowsiness; sometimes automatism and mental confusion	Sudden recovery, patient starts up and asks what has happened. No automatism or mental confusion
Reflexes	Transient reflex changes as in disturbance of the pyramidal system	No changes of an organic nature

A differential diagnosis has to be made from:—

1. Hysterical seizures.
2. Focal epilepsy.
3. Attacks associated with labyrinthine disease.
4. Cardiac syncopal attacks.
5. 'Vagal' attacks.

1. *Epileptic and hysterical attacks.* The epileptic fit pursues a well-defined course, and is followed by a series of immediate sequelæ. In the hysterical seizure no such course and sequelæ are observed. The rolling or squinting movements of the eyeballs, and the oscillations of the head in hysteria, are quite distinct from the tonic conjugate deviation of the head and eyes in epilepsy. The tonic spasm of the limbs in epilepsy contrasts with the tonic spasm of the neck and trunk in hysteria. The steady evolution and eventual cessation of the clonic convulsions of epilepsy are quite distinct from the vibratory tremors of the hysterical seizure. The sudden relaxation of spasm and the almost instantaneous return to consciousness in the latter are noteworthy features.

The main points in differential diagnosis are given in the Table on p. 575.

2. *Epilepsy and focal epilepsy.* In focal epilepsy consciousness is preserved. The fit commences with clonic convulsions in a particular locality, and spreads from its seat of origin by a definite march. In severe cases the convulsion may become generalised with abolition of consciousness. In doubtful cases an examination of the motor, sensory, and reflex phenomena will usually permit of a diagnosis being made.

3. *Minor epilepsy and aural vertigo.* The chief points of resemblance and difference are seen in the following Table:—

Minor epilepsy.	*Aural vertigo.*
Onset sudden.	Onset sudden.
Duration brief.	Duration brief.
Temporary loss of consciousness.	Temporary loss of consciousness sometimes present.
Fall, more or less sudden.	Fall, usually with a feeling of being forced or dragged down.

Minor epilepsy.	*Aural vertigo.*
Frequently post-paroxysmal automatism.	Never automatism.
No local signs of ear disease.	Tinnitus and labyrinthine deafness.
Inter-paroxysmal mental state as in epilepsy.	No impairment or dementia.

4. *Epilepsy and cardiac syncope.* It is only in the minor type of epilepsy that a difficulty is likely to arise. The points in the differential diagnosis are:—

Minor epilepsy.	*Cardiac syncope.*
Loss of consciousness sudden and complete.	Loss of consciousness gradual and deliberate.
Pallor and flushing of the face rare.	Marked pallor of face, and a general feeling of coldness.
Pulse not affected.	Feeble action of pulse.
Post-paroxysmal automatism and confusion.	Never confusion after the attack.

5. *Minor epilepsy and 'vagal' attacks.* The following are the features of the 'vagal' attack as given by Sir W. Gowers[1]: (*a*) the aura is referred to the abdominal, cardiac, or respiratory organs; (*b*) the sensation ascends to the chest, throat, and head; (*c*) it is accompanied by a feeling of cardiac or respiratory distress, of fear or of impending death; (*d*) there is no true loss of consciousness, but sometimes a feeling of unreality; (*e*) the attack terminates with acceleration of the heart's action; (*f*) it lasts about fifteen or twenty minutes.

Prognosis

Epilepsy is a chronic disease, and in the majority of cases tends towards increasing frequency of the seizures and mental deterioration. A considerable percentage of cases, as a result of carefully considered and well sustained treatment over a number of years, result in improvement or recovery.

The following facts have to be taken into consideration in estimating the prognosis.

[1] Gowers, Sir W., *The Borderland of Epilepsy*, 1907.

1. A neuropathic family history does not necessarily stand in the way of arrest of the disease, or of its improvement under treatment.

2. The age at onset is important. If arising under ten years, the outlook is unfavourable; if arising during puberty, the chances of improvement or recovery are greater. Senile epilepsy, or that arising over fifty or fifty-five years of age, is a tractable disorder.

3. The duration of the disease influences the prognosis to the extent that the earlier a patient comes under treatment the more hopeful is the outlook. Fits may be arrested even after a duration of twenty or thirty years.

4. The frequency and character of the fits. Major seizures occurring at long intervals present the most favourable form of epilepsy. Fits occurring daily or weekly are unfavourable, especially when of the combined type. Minor seizures, especially when associated with slight degrees of mental impairment, are unsatisfactory to treat.

5. Marked mental impairment is an unfavourable feature; epileptics of a degenerative type are also unfavourable.

A cure or permanent arrest of fits takes place in from ten to twelve per cent. of the cases of epilepsy, which have persisted with treatment over prolonged periods.

Treatment

In the treatment of epilepsy two factors have to be considered: (1) the inherent instability of the nervous system, and (2) the various conditions and circumstances which may excite or aggravate the disease. The first factor is invariably present in greater or less degree, and in most cases careful examination reveals one or more exciting causes. Every case ought therefore to be studied systematically; and the importance of maintaining the general health of the patient cannot be too strongly insisted upon. In many instances the instability of the nervous system *per se* is slight, and provided that exciting causes can be removed very little sedative treatment is sufficient to keep the patient free from all manifestations of the disease.

Some of the more common of the exciting or reflex causes of epilepsy are worthy of special mention:—

Alimentary system. Diseased teeth, pyorrhœa, gastric disturbance, constipation, and worms.

Circulatory system. Low or high blood pressure, feeble circulation, an unstable vaso-motor system, and anæmia.

Reproductive system. Sexual excitement and irregularity in the menstrual functions in women.

Attention ought also to be paid to the general hygiene of the patient as regards exercise, fresh air, regular mode of life, and the avoidance of excitement or emotional stress.

Medicinal treatment. The *salts of bromine* are the most useful remedies in the treatment of epilepsy. They may be given in various forms—such as the bromides of potassium, sodium, ammonium, or strontium. Of these salts, potassium bromide is probably the most efficient. Sodium bromide may be substituted in the less severe cases. Ammonium bromide has been recommended in cases where the patients are depressed—more, however, on theoretical than on practical grounds. Strontium bromide has less tendency to produce acne than the other salts of bromine, and may therefore be given in cases where this complication is troublesome. The bromides may (1) arrest the fits permanently, (2) arrest the fits temporarily, (3) lessen the frequency or severity of the fits, or (4) exercise no apparent effect.

Borax (sodium biborate) is of no special value when given alone, but is sometimes serviceable when combined with bromide, especially in cases of minor epilepsy. The dose may vary from ten to twenty grains.

Oxide of zinc in doses of one to two grains once daily is sometimes beneficial in cases of minor epilepsy. An objection to its use is that it is apt to cause vomiting.

Belladonna is often successful in the treatment of minor epilepsy. It is prescribed in the form of the tincture and may be given in doses of three to five minims combined with bromide. In some cases of minor epilepsy, in which the bromides fail, the administration of belladona alone is attended with success.

Digitalis, strophanthus, strychnine, and *nux vomica* are specially useful, when combined with bromide, in those cases of epilepsy in which the fits only occur during sleep or in which there is a low blood pressure.

Nitro-glycerine has been advocated in cases in which arterial

spasm precedes the onset of the fits. In such cases it may be successfully employed, but it should not be prescribed unless the indications for its use are clearly established.

Arsenic is often prescribed, but has no special effect on epilepsy. It may act as a tonic, however, and is useful in combination with the bromides, as it reduces the tendency to acne and bromide rash.

Iron may be given in association with bromide in cases with anæmia; but it must be remembered that in a small number of cases iron tends to increase the severity of the fits.

Method of administration. The choice of remedies, the dose and the time of administration will vary according to the requirements of each case. In many instances the best results can only be obtained by practical observation of the effect of different forms of treatment. The following are the more important points to be considered before commencing treatment: (1) the character of the malady, whether the major, minor, or combined type; (2) the frequency of the seizures; (3) the time at which the seizures occur.

1. *Character of the malady.* In major epilepsy, bromide, in doses of fifteen to twenty-five grains combined with arsenic, is the most successful remedy. In minor epilepsy bromide should be given in small or moderate doses—ten to twenty grains in combination with tincture of belladonna (two to five minims) or with borax (ten to twenty grains); if there is circulatory disturbance digitalis, strychnine, nux vomica, or nitro-glycerine may be combined with the bromides, according to the indications present in each case. Zinc oxide is useful in some cases. In combined epilepsy bromide may be given alone or in association with other drugs.

2. *Frequency of the seizures.* The amount of bromide ought to be varied in proportion to the frequency of the fits. If the fits only occur once in every two or three weeks, a daily dose of ten to fifteen grains will probably be sufficient to control or overcome the fits. If the attacks are more frequent the bromide must be given more often (twice or thrice daily).

3. *Time of occurrence.* If the fits occur only at night, a twenty to thirty grain dose of bromide should be given at bedtime; and if there is any evidence of a low blood pressure, digitalis or nux vomica may be added with great advantage.

If the fits occur immediately after the patient rises in the morning, the medicine should be taken half an hour before rising. If the fits occur at irregular intervals the medicine should be given three times daily in moderate doses. In cases of serial or periodic epilepsy, the dose should be increased to anticipate the attacks, and continued in lessened amount during the intervals.

Bromism. Some patients are specially susceptible to the action of bromide and its injudicious use may give rise to toxic symptoms known as 'bromism.' In this state the mental faculties are blunted, the memory is impaired, apathy and listnessness induced, the speech is slow, the tongue tremulous, and saliva dribbles from the mouth. The gait is often staggering and the limbs weak. An eruption of acne frequently covers the skin.

Such cases are, however, extremely rare. A more common error in the treatment of epilepsy lies in not giving sufficient bromide, or in cutting off the drug too soon or too abruptly. To stop the administration of bromide suddenly in patients who have been taking it for long periods is fraught with grave danger and serious risk. In most cases the fits return with renewed violence, and in not a few instances status epilepticus has been induced. Many cases of epilepsy would be cured if the taking of a small dose of bromide once daily were made imperative for two or three years after the fits had ceased.

General hygienic methods should be prescribed along with medicinal treatment. It is sometimes better to treat even an early case of epilepsy in an institution, or under the care and supervision of a well-trained nurse-attendant. If fits are few in number, the schooling or other mental instruction should not be stopped. If they are frequent, or if the mental condition is defective, instruction should be carried out either in a special school or under private tuition at home. Field exercises and games, ordinary amusements and recreation should not be interdicted. Massage and spinal douches are often of use in the promotion of the circulation, which is notoriously sluggish. Indoor hospital treatment is unsatisfactory, except in cases of great debility, or when the fits are frequent—as in serial epilepsy and the status epilepticus.

Dietetic treatment is often a useful auxiliary to medicinal means. In all cases the diet should be such as can be easily digested, and the meals taken at regular hours. In some cases a special diet may be prescribed with advantage. Vegetable diet, 'salt starvation,' and, above all, a purin-free diet, permit the amount of bromide salt being reduced to a minimum. Alcohol and malt extracts tend to aggravate the disease, and should be avoided in all cases. Tobacco may be permitted to a moderate extent.

Outdoor employment in robust persons is especially recommended; but for the physically frail and feeble some lighter form of work is desirable.

Status epilepticus. If a gradual increase in the number of fits suggests the onset of status epilepticus, the dose of bromide salt should be doubled, and ten or fifteen grains of chloral hydrate added and prescribed every four or six hours. During the height of a status attack nothing will arrest the seizures except the inhalation of chloroform. Hydrobromate of hyoscine ($\frac{1}{75}$ gr. to $\frac{1}{50}$ gr.) may be of temporary use. In the after stage of stupor careful nursing, abundance of light nourishment and tonics are essential.

Acute mania. The patient should be protected from injuring himself or others. Resort may be had to a padded room, or other form of restraint. No drug is more satisfactory than the injection hypodermically of $\frac{1}{75}$ grain or $\frac{1}{50}$ grain of hydrobromate of hyoscine.

It is an old observation that a fit commencing with a peripheral warning in the hand may be arrested by the application of a strap round the wrist or arm. Pressure over the pit of the stomach, a draught of cold water, or of sal volatile, has been known to arrest seizures with epigastric warnings. Some patients by a form of auto-suggestion, or the bringing to bear a strong determination, have succeeded in overcoming the attack. The inhalation of nitrite of amyl is especially valuable in aborting those attacks, which give a warning of their onset.

During a fit, the patient should be laid on the floor. Anything likely to constrict the neck should be removed. Tongue-biting may be prevented by inserting a cork between the jaws. The post-paroxysmal sleep should be encouraged.

Epilepsy in Association with Organic Disease

Epileptic seizures, indistinguishable from those of idiopathic epilepsy, are seen in association with the following conditions:—

1. Cerebral diplegia and infantile hemiplegia.
2. Intracranial tumour.
3. Cerebral syphilis and general paralysis.
4. Traumatic lesions of the brain.
5. Hydrocephalus.
6. Vascular lesions—senile epilepsy.

In certain cases the physical signs of gross organic disease of the brain are present; but in many, such signs are either absent or late in appearing. The association of epilepsy with any one of the above conditions may not depend upon the direct action of the lesion, but upon indirect effects—such as general instability of the nervous system, or an alteration of the normal physiological conditions.

Part XIX

THE TICS

(SYN.: HABIT SPASM; HABIT CHOREA. FRENCH—'MALADIES DES TICS')

A tic may be defined as a 'co-ordinated, systematised, purposive act, reproducing in an involuntary manner the co-ordinated movements of every-day life' (Miege).[1] Such movements may be initiated by a definite peripheral irritation. Voluntary repetition of the movement in the early stages may eventually become automatic and involuntary long after the original exciting cause has yielded to treatment and disappeared. For example, a foreign body upon the cornea leads reflexly to blinking movements of the eyelids, which may be continued as a 'blinking tic' after the removal of the irritation or the subsidence of conjunctivitis.

In normal and mentally stable persons, reflexly induced movements do not lead to an involuntary and automatic repetition; this only occurs when the psychical condition is unstable or deficient.

Although the psychical stigmata may be well marked, the mental qualities associated with them may be highly developed along certain lines, leading in many cases to brilliancy, but frequently also to eccentricity. History records numerous instances of tic movements which were outstanding characteristics of many of the world's great men.

In addition to the involuntary and automatic movements mentioned as characteristic of the tics, there is also a form of

[1] Miege and Feindel, *Tics and their Treatment* (Translated by Dr. S. A. K. Wilson), 1907.

psychical tic, dependent upon idea. These are the obsessive tics, which form a troublesome type of the malady, and may or may not be accompanied by motor equivalents. Thus we find persons who must carry out certain actions in a definite way; others who are impelled to do certain acts for fear of something happening if they do not. Of similar character is the dread of touching certain objects (mysophobia), and an irresistible impulse to count a certain

FIG. 184.

FIG. 185.

FIGS. 184 AND 185.—Two figures illustrating tic movements of the head and neck.

number before doing anything (arithmomania), or the necessity to find certain words, which have to be repeated (onomatomania).

Etiology. Tics may arise at any age after the years of early childhood. They most commonly begin during youth.

They affect the sexes about equally. Heredity plays a very important part in their causation. In the parents or collaterals, neuropathic maladies are noted—such as hysteria, alcoholism, epilepsy, chorea, tics, and organic nervous diseases. On the other hand, mental precocity, brilliancy, and eccentricity may also be observed.

Infective disorders have been known to both increase and

to lessen tics when developed. Mimicry and imitation in the young may be factors in their causation. Brain fag, overwork, worry, and anxiety are likely to favour their development in adults.

VARIETIES OF MOTOR TIC

(*a*) Facial tics are seen in blinking movements of the eyelids, smiling, grimacing, mimicry, winking, tonic closure of the eyelids, and smacking movements of the lips.

(*b*) Tics of the jaws are observed in teeth grinding, abnormal movements of the lower jaw, and trismus; also as part of more complicated actions—such as biting the lips (cheilo-phagia) and biting the nails (onycho-phagia).

(*c*) Tics of the neck are characterised by movements of jerking and tossing of the head, elevation or depression of the chin, rotation and inclination, and movements of affirmation and negation.

Torticollis may be regarded in many cases as a tic of the neck muscles (p. 590).

(*d*) Tics of the upper limb are common, and consist of elevation, depression, and twitchings of the shoulder; abduction and adduction movements of the arm; shrugging of the shoulders, beating and striking the chest, and scratching movements of the hands.

(*e*) In the lower limbs, knocking or rubbing one leg against the other, tics of gait, kicking, genuflexion and various complicated actions of a characteristic kind are seen.

(*f*) Tics of the digestive system are found in clicking movements of the soft palate, continuous swallowing of saliva, constant eructations, air swallowing and its associated phenomena (aero-phagia).

(*g*) Respiratory tics are seen in sniffing, snoring, whistling, blowing, coughing, sobbing, and hiccoughing.

(*h*) Tics of speech are seen in the emission of words or phrases without any relation to what is being said, and during periods of silence. Under this heading the irresistible utterance of oaths, or obscene language (coprolalia), the repetition of words or sounds (echolalia), and the imitation of gestures (echokinesis) may be included. These last conditions are more usually found in a special type of the malady—

convulsive tic, or Gilles de la Tourette's disease—which requires a separate mention.

Convulsive tic. This is a form of tic—rarely seen in this country—characterised by spasmodic jerking movements, with which are associated uncontrollable utterances, impulsions, and obsessions dependent upon imperative ideas.

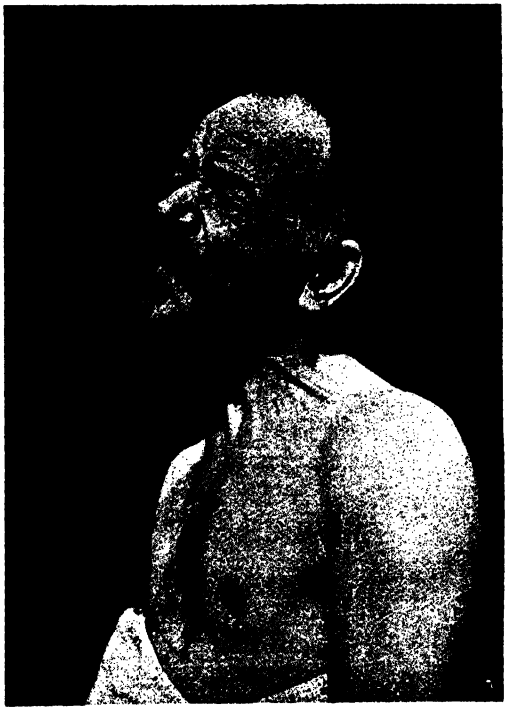


FIG. 186.—Illustrates the appearance of the face in a case of extensive tic-like spasm of the facial muscles.

The spasmodic movements are more generalised and bizarre than in the simple tics previously described. The exclamatory utterances consist in the emission of words, sounds, or phrases entirely irrelevant and sometimes blasphemous; in the repeating of names, sounds, or unfamiliar words, and the imitation of gestures. These patients suffer from imperative ideas and obsessions, and present the typical features of the psychical foundation of the tics to an exaggerated extent.

This variety of the disease is probably the same as that described in other countries under different names—such as 'Jumpers' of the state of Maine, the 'latah' of the Malays, and the 'myriat chit' (play the fool) of Siberia.

Facial spasm. This is a condition more properly described under the tics, in which spasmodic twitchings are limited to the distribution of the seventh nerve upon one side. The spasms are of a clonic character, and come on in attacks lasting from several minutes or some hours, with intervening periods of comparative freedom. They may involve all portions of the facial musculature, or be mainly limited to the orbicularis palpebrarum, the angle of the mouth, and the chin muscles.

The cause may be found in a peripheral irritation in the distribution of the fifth nerve—such as the nose, eyes, mouth, or teeth. In the majority of cases no such cause is detected; or, if at one time such exciting cause was present, it has long since ceased to act.

The disease has a prolonged course, usually persisting throughout the remainder of life. It is not painful. It is more common in women than in men, and treatment is of little avail.

The only condition with which this is likely to be confused is a localised or epileptiform convulsion arising from cortical irritation of the face area.

Diagnosis. As already defined, the diagnosis of tic is based upon a coexistent motor and mental association. There are, however, numerous disturbances of motility—such as spasm, chorea, cramp-like movements, myoclonus, mannerisms, and stereotyped acts, which it is important should be distinguished from tic.

This is often no easy matter, but certain general guiding lines may be laid down.

(*a*) Tic movements are under the influence of the will to the extent that they can be controlled temporarily, but their repression is accompanied by mental distress.

(*b*) Distraction of the mind lessens, fatigue and mental emotion increase the activity of the movements.

(*c*) Tics, as a rule, disappear during sleep.

(*d*) Tics are associated with, and found exclusively in, those who present certain definite mental peculiarities.

The chief points of differential diagnosis may best be reproduced in the form of a table.

TABLE GIVING THE CHIEF POINTS OF DIFFERENTIAL DIAGNOSIS

	TIC.	CLONIC SPASM.	CHOREA.	PARAMYOCLONUS MULTIPLEX.	STEREOTYPED ACT.
Influence of volition	Controlled temporarily with great distress	No effect	Slight increase or no effect	None	Checked or modified, but with mental distress
Type of movement	Co-ordinate, purposive, repeated	Abrupt, in-co-ordinate, limited to definite area	Erratic, irregular, not repeated in similar fashion	Muscular fascicular contractions, instantaneous, involuntary, bilateral	Mannerisms of multiform type and character
Influence of sleep	Usually arrested	Not arrested	Arrested	Continued during sleep as a rule	Arrested
Associated symptoms	Obsessions. Mental features as described in text	Possibly signs of organic disease	Pains, perhaps arthritis and endocarditis	Increase of myotatic irritability Legs usually most affected	None
Course of the disease	Usually persistent, sometimes incurable	Depends on cause	Recovery usual	Incurable	Recovery possible

Prognosis. Taken as a whole, the outlook as regards recovery in cases of tic is unfavourable. In young patients, however, suitable treatment directed more especially to the correction of the movements by hygienic and educational methods, may bring about a satisfactory and sometimes permanent arrest. When they start in adults, on the other hand, or if the movements have been in existence for long periods, the disease is likely to become permanent. In the severer forms with pronounced mental symptoms—such as the convulsive tics—the prognosis is grave; for insanity may develop, and the persistence of the motor disturbances and severe obsessional feeling may lead to suicide.

Treatment. All ordinary methods of treatment are of little use in this disease.

Medicinal remedies—such as the bromides, valerian, cannabis indica, belladonna, gelsemium, and arsenic may, one and all, be of some temporary value. A useful combination

has been found in a mixture containing bromides, cannabis indica, and conium.

Massage, hydrotherapeutics, and the application of electricity are also of little more than transient value. Certainly some forms of torticollis are made worse by the galvanic current.

Suggestion is of little use in cases of true tic. In cases of hysterical spasm, or in those tic-like conditions associated with hysteria, hypnotic suggestion is of real benefit, and is sometimes effectual in promoting a cure.

The only certain and satisfactory treatment of tic movements lies in the persistent, prolonged, and regular execution of special movements of the affected muscular groups. By this means both the mental and physical elements of the malady are corrected. The treatment rests generally upon the principle of providing exercises, which, in the first place, tend to arrest the abnormal movements, and, secondly, counteract the abnormal by normal movements. On the one hand, the patient is encouraged to restrain the movements—at first for short periods, and later for longer periods—in all positions and attitudes: from those of greatest ease and comfort up to those which involve walking and talking. On the other hand, and concurrently, the patient is instructed in the execution of slow, regular, systematic, and accurate movements of the muscles involved in the tic.

These séances should at first last only a few minutes, and later be increased up to half an hour several times daily. They should be carried out before a mirror, so that irregularities and mistakes may be seen and corrected. They require to be continued for long periods, even after the tic has apparently subsided.

Although 'habits' in children are not tics, they should be carefully handled by judicious, moral, and systematic treatment.

TORTICOLLIS TIC

Putting aside the cases of 'rheumatic stiff-neck,' spasmodic torticollis is the most common variety of wry-neck. It is one of the manifestations of tic.

In the early stages of the disease a movement of the head is present—at first temporary and controllable · but later on, and in severer cases, the deviation of the head may become persistent without relaxation, and associated with spasmodic movements of other muscles.

Etiology. It is more common in men than in women. It occurs mainly in adult life. A neurotic heredity is found in most cases, and the disease frequently develops in those who have exhibited nervous or hysterical tendencies. It may follow upon injury, sudden movement of the head, or in consequence of pain.

FIG. 187.—Shows the appearance presented by a case of spasmodic torticollis.

Symptoms. The onset of the spasmodic movements is gradual over several weeks or months. In the early stages there may be a temporary and complete cessation, but once developed the progress is usually steady.

In whatever manner it starts, three fundamental features are demonstrable; first, the movements are such as can be voluntarily imitated; secondly, in the early stages, the movements are to a large extent under voluntary control; and, thirdly, the movements are the consequence of an uncontrollable impulse.

If traced to its origin, it is found that in the initial stages a desire to perform a movement is present. The mental impression of the patient is that if the movement is carried out mental satisfaction will result. The result is, however, the reverse, as dissatisfaction and remorse follow in its wake. If the movement is controlled by an effort of the will, great distress ensues. These are the features common to all tics.

The spasms consist of sudden quick contractions of the affected muscles, causing the head to turn in the direction of action of the muscle. In other cases it is of a slower and more prolonged character, by which the head may be turned steadily to one or other side, and maintained there for an appreciable period before the spasm relaxes.

On the other hand, it may be arrested by some simple process, such as placing the finger on or near the chin.

Fig. 188.—Photograph of a case of antero-collic spasm.

The symptoms vary according to the muscles which are affected. The sterno-mastoid is most often the seat of spasm, but it may be associated with the upper part of the trapezius upon the same side. The splenius, more especially on the opposite side, is less commonly involved. The scaleni, platysma myöides, and the deeper neck muscles are only rarely the seat of spasm.

Various types of spasmodic turning of the head are observed. If the sterno-mastoid is alone affected, the face is turned to the opposite side, the chin is protruded, and the head inclined towards the same side as the affected muscle.

An associated spasm of the upper part of the trapezius on the same side will incline the head to that side and draw it slightly backwards. The simultaneous action of both trapezii and splenii results in a backward movement of the head (retro-collic spasm).

A spread of the spasm to other and adjacent muscles is not uncommon. Thus the face, shoulder, and arm may become affected, and in severe cases the muscles of the back.

The spasms always cease during sleep.

Sensory symptoms are rare. Pain of a neuralgic type may be complained of at the back of the head and neck. The spasm itself is not painful, but creates great discomfort and distress.

The course of the disease varies. In some cases it ceases either spontaneously, or in consequence of treatment after some months or years. Some degree of spasm usually persists in severe cases. It has no influence upon the duration of life.

Treatment. The malady does not lend itself satisfactorily to the influence of drugs; and what has been said upon the treatment of tics in general holds good for that of torticollis spasm. The bromides, valerian, belladonna, trional and the hypnotics, cannabis indica, and conium may all be tried with some benefit. In the severe cases, with spread of the spasm to the shoulders, arms, and back, we have seen much temporary benefit result from hypodermic injections of the hydrobromate of hyoscine.

Galvanism has been of occasional use in some cases when the positive pole is applied over the affected muscles. In other cases it has apparently aggravated the spasms.

Hypnotic suggestion has been of use in cases which are of an hysterical character.

Operative interference such as division of the spinal accessory nerve may temporarily arrest the movements; but the only operation likely to induce permanent benefit consists in dividing the spinal accessory nerve on one side and the posterior primary divisions of the cervical nerves upon the other.

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